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Contents:

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VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1664 reports were processed during this period.

Q fever: Twelve cases of Q fever (10 males, 2 females) were reported during this period. Except for an 11-year-old schoolgirl who visited a farm on a school excursion, ages ranged from 19 to 46 years. Three cases were identified as meatworkers. None of the 5 cases from South Australia were vaccinated.

Influenza: Forty-eight cases of influenza B were reported during this period - all were from Western Australia. This brings the number of cases reported by the State Health Laboratory, Perth, and Princess Margaret Hospital, Perth, to 74, and the total reported to CDI this year to 84.

Cases are predominately in young children:

Age	Per cent of cases
<1 year	31
1-4 years	18
5-14 years	19
15-24 years	4
25-49 years	13
60+ years	15

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- Figures given may be subject to revision.

Both sexes are equally represented.

Most cases (57/84) have been diagnosed by virus isolation with 27 cases serologically confirmed.

Influenza A activity continues to be sporadic with 4 cases reported during this fortnight (cumulative total 19). A third of these cases are in over 60 year-olds.

RSV: Four hundred and forty-nine (449) cases of RSV were reported during this fortnight bringing the year's total to 2110. Seventy per cent of the total cases are in children under 12 months of age, with another 23 per cent in children aged 1-4 years.

Echovirus type 4: Ten reports of echovirus type 4 were received during this period - all from Victoria. Collection dates were distributed over 3 months. A total of 26 reports have been received so far this year; most cases have been associated with meningitis. A cluster of 7 cases was also seen in Western Australia in January this year. Forty-seven cases of echovirus type 4 were reported to *CDI* last year.

Echovirus type 30: The outbreak of echovirus type 30 which commenced in April 1988 continues with 6 cases reported during this fortnight. Already this year 214 cases have been reported; 215 were reported last year making a total of 429 cases since the outbreak began.

SALMONELLA TYPHIMURIUM PHAGE TYPE 201a IN SOUTH AUSTRALIA

(Contributed by C. Murray, P.S. Gasiorowski, C. White, and P. Drake, South Australian Health Commission)

An outbreak of *Salmonella typhimurium* phage type 201a has been evident in Victoria since December 1988. Initially 25 people were infected after attending a restaurant. To date there have been 65 human isolates and two isolations of *S. typhimurium* phage type 201a from Victorian boned mutton.

In South Australia, the first human isolate of *S. typhimurium* phage type 201a was identified on 15 January 1989 and cases continued until 6 March 1989. Forty-one human isolations were made.

The source of these infections was expected to be a common foodstuff, therefore:

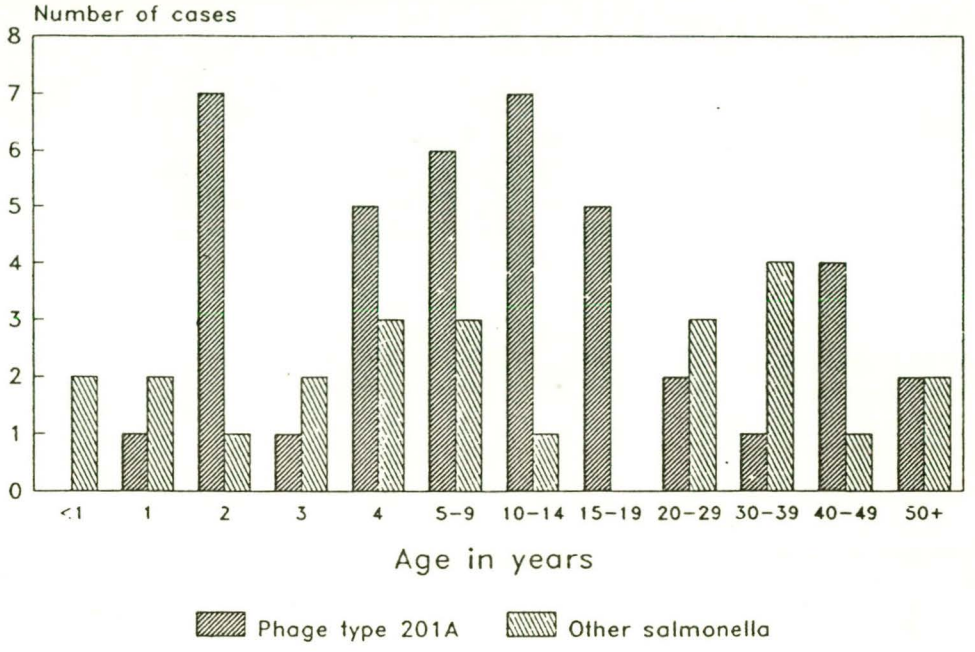
- 32 of the 41 cases of *S. typhimurium* phage type 201a were intensively questioned regarding:
  - travel;
  - food eaten prior to illness; and
  - whether they were exposed to infected people; and
- 32 family contacts were screened for salmonella infections.

Similarly, 24 cases of salmonellosis due to other salmonella species, which had been notified during the period of investigation were intensively questioned.

The distribution of salmonellosis cases by age, both for *S. typhimurium* phage type 201a and for other salmonellas, is shown in Figure 1. The predominance of cases in persons aged

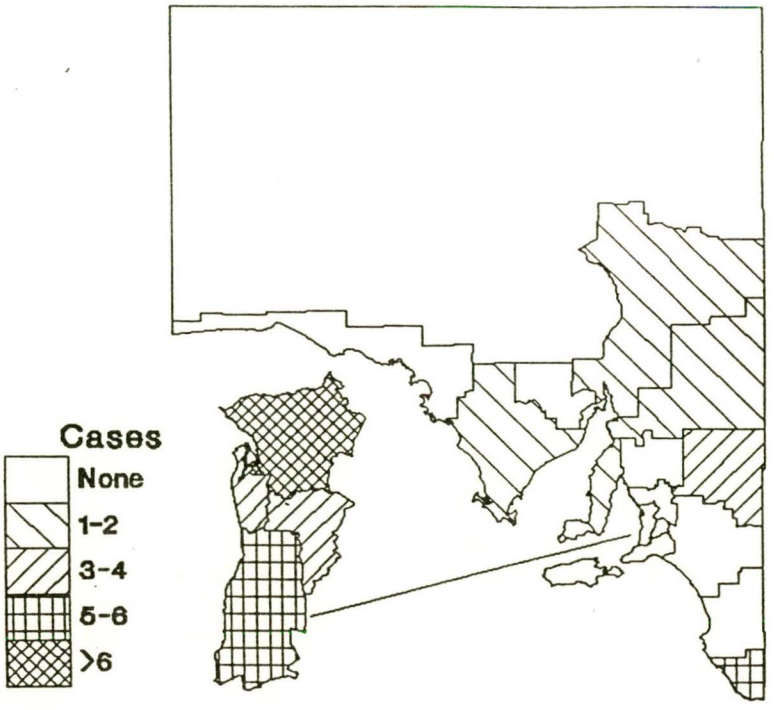
2 years and 4-19 years is marked. The sex ratio was almost equal with males being slightly over represented; 55.0% of the *S. typhimurium* 201a cases and 54.0% of the other salmonella cases.

Figure 1: Age grouping of salmonellosis cases by type.



Cases were distributed statewide, with the highest single concentration located in the northern metropolitan CURB region of Adelaide (Figure 2).

Figure 2: Geographical distribution of cases of salmonellosis notified to the South Australian Health Commission, January to March, 1989.



The age, sex, and geographical location of the cases corresponded with expectations of the source being in the commercial food chain, however intensive follow-up of the thirty two cases failed to give any clue to foods common to all cases.

Phage type 201a has not been a common phage type. It is a variant of phage type 201, and was previously called RDNC type (reacts but does not conform to a standard pattern). The use of 201a to recognise this type has only been used at the Institute of Medical and Veterinary Science from the beginning of 1989, when this outbreak began.

On reviewing previous phage typing records, prior to December 1988 phage type 201a had only been seen from sheep, with all except one isolate coming from Victoria where it has been endemic for a long period of time. The one non-Victorian isolate was recovered from sheep in South Australia in September 1988. A limited survey of sheep faeces during the investigation of this outbreak failed to identify the organism in sheep in South Australia.

This episode is an example of a common situation in Australia where an obvious epidemic has been intensively studied (including the hiring of extra staff dedicated to the task) without the source of the outbreak being identified. Infectious disease epidemiologists are rare in Australia and a pool of experienced personnel are urgently required to help make sense of such situations as described here, with over 100 persons ill enough to warrant at least a medical consultation and expensive laboratory investigations.

ISOLATION OF LEGIONELLA FROM COOLING TOWERS SUPPLIED BY ARTESIAN BORES

(Contributed by Dr Sally Ng and Mr Colin Derbyshire, Health Department Victoria)

A large part of Australia is dependent on groundwater as a source of potable water which, in certain areas, is supplied by artesian bores. Artesian bores can be 500 to 2000 metres deep and, as the water temperature can exceed 60°C, cooling towers are used to cool the water prior to it entering the distribution system.

Cooling towers associated with air-conditioning systems and industrial processes have been implicated in outbreaks of legionellosis. Now, *Legionella* has been isolated from samples of water taken from cooling towers used to cool water obtained from artesian bores.

L. pneumophila serogroup 1 was isolated from water samples taken from two cooling towers servicing two artesian bores in western Victoria. In both systems, water leaves the bore at approximately 41°C and is cooled to 26°C to 30°C. The high soluble iron content is oxidised to insoluble iron by aeration within the cooling towers. The insoluble iron compounds gravitate to the bottom of the tower cold water basin and require regular physical removal.

In one system, water was chlorinated in the tower cold water basin prior to entering the distribution system. In the other

system, water was chlorinated as it left the tower *en route* to covered storage before entering the same distribution system.

L. pneumophila serogroup 1 had never been isolated from samples of water taken from the distribution system, although it was isolated from water in the hot and cold water basins in both towers. The absence of *Legionella* in water leaving the bores suggests that aeration and air scrubbing within the towers had led to *Legionella* contamination of the water.

Although these two cooling towers had not been implicated in cases of legionellosis, a risk existed for the following reasons:

(a) Persons working, living or otherwise close to the towers could inhale *Legionella*-contaminated drift (aerosol) emanating from the towers.

(b) Cooled water leaving the towers is still at a temperature which is conducive to the proliferation of *Legionella*. Persons showering in this tepid water from cold taps could inhale *Legionella*-contaminated aerosols if the water is inadequately disinfected.

Control of *Legionella* within the cooling towers was effected by recycling chlorinated water, via the hot basins, throughout the towers. This procedure, carried out at intervals of not greater than one week, minimised the risk of *Legionella* contamination of tower drift.

Continuous effective chlorination of water entering and within the distribution system also serves as further protection from *Legionella* which may otherwise be disseminated by aerosol generating devices such as shower heads and garden sprinklers.

As the water is used for human consumption and normal domestic purposes, the wide range of chemicals normally used in cooling towers that service air-conditioning systems and industrial processes cannot be used.

To date, there has been no further isolation of *Legionella* from samples of water from the cooling towers or the distribution system.

A CASE OF SUBACUTE SCLEROSING PANENCEPHALITIS (SSPE) IN A PREVIOUSLY IMMUNISED CHILD

(Based on Infectious Diseases Bulletin, No 51, May 1989, Department of Microbiology, Royal Children's Hospital, Melbourne)

In March 1989 a case of subacute sclerosing panencephalitis (SSPE) was diagnosed in a 15-year-old girl. She had been immunised against measles at 15 months of age, and had a febrile illness at 2 years of age which was diagnosed as measles by her local medical officer, although it was apparently atypical. Her case history follows.

In December 1987, the patient (then 13 years old) presented with a 2 week history of 4 subtle episodes of transient loss of postural tone in the right arm and/or right leg and paresis of the right side of the mouth with associated aphasia. These episodes lasted for seconds only (2-5secs) and there was not

clonus. She was fully aware of them and otherwise well with no prodrome, visual disturbance, headache or vomiting and no obvious precipitant. Intercurrently she was well. Physical examination was normal. Differential diagnosis included epilepsy, complicated migraine and an embolic aetiology. She was subsequently referred to neurology outpatients. CT head scan and EEG showed no diagnostic features.

In June 1988, the patient represented with an increasing frequency of the same episodes averaging 5 per day, but on occasions up to 15 per day. They manifested as dropping of objects or sudden loss of lower limb tone and a subsequent fall. They were quite brief and she continued to attend school. Diagnosis was of focal motor epilepsy and she was commenced on carbamazepine which initially showed good control.

Three months later however (September 1988), there was poor control averaging 5-6 episodes per day. Carbamazepine levels were therapeutic. Apart from the occasional head turning episode (to the right) there were not new signs. Clonazepam was added to treatment but no improvement occurred.

Eleven months after onset a video-EEG was performed. This excluded a psychogenic aetiology and confirmed focal motor epilepsy. Clonazepam was ceased and phenytoin added to therapy but there was little improvement.

She was admitted 14 months after onset (Feburary 1989) for head imaging using both CT scan and MRI. They showed a structural abnormality predominantly in the white matter of the right temporo-occipital lobes. Radiologic appearance was consistent with an infiltrating lesion such as low grade glioma. Physical examination was normal apart from a left homonymous hemianopia and a small area of right macular atrophy. CSF was normal with no cells and a negative cytospin. A brain biopsy revealed extensive perivascular lymphocyte infiltration and gliosis. Bone marrow histology, FBE, chest x-ray and abdominal ultrasound were normal.

Further history showed that there had been some personality changes (flattening of affect, peer group withdrawal) and deterioration in schoolwork and handwriting (teacher's report) in the preceding year. At this stage a history of measles was investigated.

Chronic encephalitides were considered and both serology and CSF had high titres of measles IgG antibodies (ELISA technique). Reprocessing of brain tissue for electron microscopy from paraffin block revealed intranuclear inclusions (granules and short tubules) consistent with measles virus substructures.

Diagnosis was then made of subacute sclerosing panencephalitis and she was discharged on carbamazepine and isoprinosine (an anti-viral and immunopotentiating agent). Another EEG (April 1989) had periodic stereotyped slow wave complexes consistent with SSPE.

Infectious Diseases Bulletin Comment

SSPE is a rare progressive neurodegenerative disorder occuring worldwide in about $1/10^6$ of children who have had measles immunisation and in $1/10^5$ after wild measles infection [1].

The evidence that SSPE can be due to measles vaccine however is quite tenuous [2]. The National SSPE Registry in the USA shows that the incidence of both SSPE and measles is declining as a result of an extensive and effective vaccination program [3]. A Queensland study showed an annual incidence of 1.21 cases/million people or about 10 times the incidence in the USA (0.13/million). By comparison European nations had figures from 0.06-0.5/million. In Auckland, NZ, it was 7.7/million. The large variability is related to vaccination programs, host (co-existence with EBV and herpes zoster at onset), genetic and environmental factors, and demography (increased incidence in rural populations suggesting a possible zoonotic cofactor) [1,3].

Children with SSPE often have had measles at an unusually young age (less than 2 years of age). SSPE is diagnosed at a mean age of 10 years, ranging from 1-21 years [1]. On average there is a 6-7 year interval between measles and onset of SSPE which may first manifest as behavioural, personality or school-function changes (months-1 year). The second phase is of more acute but variable neurological problems eg convulsions, myoclonic jerks, and dyskinesias (up to 1 year). There is a typical EEG pattern by this stage, as well as ocular signs (eg macular degeneration, papilloedema, optic atrophy) in 50% of cases [1]. EEG shows spike and slow wave complexes, often becoming periodic, with intervening depression of rhythmic activity ('suppression bursts'). Characteristic EEG changes were not initially present in this case and historically her behavioural changes paralleled the convulsive disorder.

The prognosis is generally poor; most patients die within 2 years of onset (60% at 1 year) after progressing to a third phase or opisthotonus and coma. A minority survive this phase in an almost decorticate state of mutism and hypotonia. Arrest of progression is infrequent but can occur at any stage. Some 10% have a rapidly progressive form and another 10% a chronic relapsing-remitting form [1].

Diagnosis can be made with serology and typical EEG changes. Brain biopsy is rarely required unless other degenerative disorders, malignancies, or atypical presentations are considered. High IgG titres in CSF (as in this case) are typical in SSPE. Both IgG and IgM antibodies are produced initially but IgM is rarely detectable for more than 6 weeks after infection or recrudescence of measles. Insignificant CSF yet positive serum titres of other common IgG antibodies (eg rubella) in this patient established that there was no blood-brain leak and the CSF measles antibody was of CNS origin. Although patients with SSPE have high titres of measles specific antibodies in sera and CSF there is a relative lack of antibody against one of 6 measles structural polypeptides forming the M protein. Detection of measles virus antigen in brain biopsy by immunoflurescence was limited here by sample reprocessing. Only paraffin sections were available. Intranuclear inclusions of measles virus sub-structures in neuronal cells were seen by electron microscopy. Recent studies using cDNA probes to detect measles virus RNA sequences in circulating lymphocytes have found them in 70%-90% of patients with SSPE compared to less than 10%-15% in aged matched seropositive people [5].

Routine examination of CSF is frequently normal but other helpful evidence (if available) is an increased level of

gamma-globulin, a first-zone rise in colloidal gold (Lange) test and oligoclonal IgG reacting with measles antigen.

Both CT scan and MRI abnormalities are variable and are not specific for SSPE. Early in the illness the scan may be normal. Cerebral oedema may follow in the second stage, then white matter changes which may be focal, multifocal or diffuse. MRI with its greater resolution may show more marked and earlier changes [6,7].

Numerous drugs have been used to treat SSPE but their value has been difficult to assess as the disease runs such a variable course. These have included interferon, transfer factor, levamisole, isoprinosine and amantadine in different doses, routes and durations [1,8,9].

Isoprinosine is an inosine complex which reportedly enhances cell mediated immunity during active viral replication by changing polyribosomal structure thereby impairing the acceptance of foreign messenger material. Reports are conflicing but a number of studies have shown that it has been effective in prolonging life and slowing progress of SSPE, particularly the more chronic form [1,9,10].

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AN OUTBREAK OF VARICELLA AMONGST NURSING STAFF

(Based on CDR 89/19, 12 May 1989)

The outbreak: An outbreak of chickenpox occurred amongst nurses working on a 30 bed long-stay geriatric ward. The index case was an 86-year-old lady who had been a patient on the ward for four years following a cerebro-vascular accident. On 22 October she developed lesions of Herpes zoster (shingles) affecting the right iliac area. The patient continued to be nursed in the open ward although acyclovir was given for three days. On 9 November, 18 days after the development of Herpes zoster in the patient, one nurse developed varicella, to be followed by three others in the next two days. Two further cases developed in nursing staff on 16 and 25 November. Thus, six of 21 nursing staff who worked on the ward at that time

were affected, an attack rate of 29%. The only case from whom serology was available showed seroconversion to Varicella zoster virus. All the affected staff recovered completely and returned to work within 20 days. Altogether 88 working days were lost due to this outbreak.

CDR Editorial Comment: The relationship between varicella and Herpes zoster is well known, as is the observation that susceptible persons are at risk of developing varicella following contact with cases of varicella of Herpes zoster.

Herpes zoster is common, particularly in an elderly population. The reported attack rate is 3.39 per 1,000 per year, of which most cases occur in people over 60 years [1]. The level of immunity to Varicella zoster virus in adults is also high. Ninety-five and a half per cent of parturient women born in the United States were immune [2]. These factors, along with the generally mild nature of varicella in children, may give an air of complacency to its management. Nevertheless, primary varicella in adults is frequently unpleasant and can be life threatening, especially in those who are immunocompromised or pregnant [1,3].

The high attack rate amongst nurses in this outbreak is most unusual as less than 5% of the population should have been susceptible [2]. A higher proportion of adults from tropical areas are susceptible to varicella [2,4], but all of the affected nurses were of caucasian extraction. Twelve of the unaffected nurses in the outbreak were contacted. Of these 12, 11 gave a positive past history of having had chickenpox. The remaining nurse, who could not recall ever having had chickenpox, gave a positive micro-immunofluorescence test with a titre of 1 in 16. Therefore all the nurses traced, who did not develop chickenpox whilst working on the ward at the time, either had a past history of chickenpox or positive serology.

This outbreak illustrates the importance of taking an aggressive attitude to cases of Herpes zoster in hospitalised patients. Outbreaks of chickenpox in hospital can probably be prevented by isolating cases of chickenpox or zoster and only allowing immune nurses to care for them. The immune status of nurses can be determined from a previous history of chickenpox or, in those who give a negative history, by one of the available rapid tests for IgG antibody to Varicella zoster virus. There may also be an argument for screening immune status to varicella as part of all nurses' pre-employment medical examinations.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 20/7/89 TO 02/8/89

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
0100	ADENOVIRUS NOT TYPED	0	5	1	0	8	8	3	2	27
0101	ADENOVIRUS TYPE 1	1	1	0	0	1	0	0	0	3
0102	ADENOVIRUS TYPE 2	1	2	1	3	0	0	0	0	7
0103	ADENOVIRUS TYPE 3	9	1	0	0	3	0	0	0	13
0104	ADENOVIRUS TYPE 4	6	0	0	0	0	0	0	0	6
0105	ADENOVIRUS TYPE 5	1	0	0	1	0	0	0	0	2
0107	ADENOVIRUS TYPE 7	0	0	0	1	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	1	2	0	0	1	0	0	0	4
0110	ADENOVIRUS TYPE 10	0	1	0	0	0	1	0	0	2
0112	ADENOVIRUS TYPE 12	0	0	0	0	1	0	0	0	1
0113	ADENOVIRUS TYPE 13	0	0	0	0	1	0	0	0	1
0118	ADENOVIRUS TYPE 18	0	1	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	10	0	0	0	0	10
0201	INFLUENZA A VIRUS	0	1	1	0	2	0	0	0	4
0203	INFLUENZA B VIRUS	0	48	0	0	0	0	0	0	48
0301	PARAINFLUENZA VIRUS TYPE 1	1	0	0	3	0	0	0	0	4
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	2	1	0	1	0	0	5
0303	PARAINFLUENZA VIRUS TYPE 3	1	0	9	4	0	1	0	0	15
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	1	0	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	58	138	155	55	5	11	27	0	449
0500	RHINOVIRUS (ALL TYPES)	2	3	18	8	1	0	0	0	32
0600	MYCOPLASMA PNEUMONIAE	4	3	18	2	0	1	0	3	31
0700	ORNITHOSIS-PSITTACOSIS	1	0	0	0	0	0	0	0	1
0900	COXSACKIEVIRUS GROUP B - NOT T	0	0	1	0	0	0	0	0	1
0903	COXSACKIEVIRUS B3	0	0	0	0	2	0	0	0	2
0904	COXSACKIEVIRUS B4	0	0	5	0	0	0	0	0	5
0905	COXSACKIEVIRUS B5	2	0	0	0	0	0	0	0	2
1004	ECHOVIRUS TYPE 4	1	0	0	9	0	0	0	0	10
1009	ECHOVIRUS TYPE 9	1	0	0	1	1	0	0	0	3
1014	ECHOVIRUS TYPE 14	0	0	0	0	0	0	1	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	1	0	0	1
1022	ECHOVIRUS TYPE 22	1	0	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	1	2	0	0	3	0	0	0	6
1100	POLIOVIRUS NOT TYPED	0	0	0	5	0	5	0	0	10
1101	POLIOVIRUS TYPE 1	0	0	1	0	1	0	0	0	2
1102	POLIOVIRUS TYPE 2	0	0	0	0	2	0	2	0	4
1103	POLIOVIRUS TYPE 3	0	0	0	0	1	0	1	0	2
1104	POLIOVIRUS - MIXED VACCINAL ST	0	1	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	4	1	0	0	5
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	2	1	0	93	0	0	1	97
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	2	5	19	0	0	1	3	12	42
1303	VARICELLA-ZOSTER VIRUS	3	7	1	1	0	1	0	0	13
1306	HERPES SIMPLEX TYPE 1	47	44	26	0	8	6	0	0	131
1307	HERPES SIMPLEX TYPE 2	49	70	25	0	54	6	0	0	204
1399	HERPES VIRUS TYPING PENDING	1	0	0	3	0	0	0	0	4
1401	COXIELLA BURNETII	1	0	5	0	0	0	0	6	12
1502	PICORNIA VIRUS - NOT TYPED = E	0	1	0	0	0	10	0	0	11
1521	MEASLES VIRUS	1	0	0	0	0	0	0	0	1
1522	RUBELLA VIRUS	1	3	0	0	0	0	0	11	15
1532	HEPATITIS B ANTIGEN	3	27	24	2	57	11	2	0	126
1535	HEPATITIS A ANTIBODY	0	0	3	0	0	0	0	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	0	34	20	0	24	1	0	14	93
1556	CMV - CYTOMEGALOVIRUS	36	9	1	3	0	2	5	3	59
1564	ROTAVIRUS	0	40	2	0	4	3	4	0	53
1599	ENTEROVIRUS TYPING PENDING	0	0	0	9	0	7	2	0	18
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	0	0	0	0	0	0	0	4	4
9992	ROSS RIVER VIRUS	1	14	1	0	0	0	0	28	44
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	1	0	1
9995	DENGUE	0	0	0	0	0	0	0	2	2
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	0	7	7
TOTAL		238	465	340	122	277	78	51	93	1664

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 20/7/89 TO 02/8/89

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	5	5	0	0	0	14	0	0	0	0	24
0101	ADENOVIRUS TYPE 1	1	1	0	0	0	0	0	0	0	0	2
0102	ADENOVIRUS TYPE 2	1	3	0	0	1	0	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	1	3	0	0	0	0	0	0	0	0	4
0105	ADENOVIRUS TYPE 5	1	1	0	0	0	0	0	0	0	0	2
0107	ADENOVIRUS TYPE 7	0	1	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	2	0	0	0	0	0	0	0	0	0	2
0110	ADENOVIRUS TYPE 10	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
0113	ADENOVIRUS TYPE 13	0	0	0	0	0	1	0	0	0	0	1
0118	ADENOVIRUS TYPE 18	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	0	3	0	0	1	0	0	0	0	0	4
0203	INFLUENZA B VIRUS	2	39	0	0	0	0	0	1	0	0	42
0301	PARAINFLUENZA VIRUS TYPE 1	0	4	0	0	0	0	0	0	0	0	4
0302	PARAINFLUENZA VIRUS TYPE 2	0	4	0	0	0	0	0	0	0	0	4
0303	PARAINFLUENZA VIRUS TYPE 3	1	12	0	0	0	0	0	0	1	0	14
0399	PARAINFLUENZA VIRUS TYPING PEN	0	1	0	0	0	0	0	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	11	436	0	0	0	0	0	0	0	1	448
0500	RHINOVIRUS (ALL TYPES)	1	30	0	0	0	0	0	0	0	0	31
0600	MYCOPLASMA PNEUMONIAE	1	30	0	0	0	0	0	0	0	0	31
0700	ORNITHOSIS-PSITTACOSIS	0	1	0	0	0	0	0	0	0	0	1
0900	COXSACKIEVIRUS GROUP B - NOT T	0	1	0	0	0	0	0	0	0	0	1
0903	COXSACKIEVIRUS B3	1	0	0	0	0	0	0	1	0	0	2
0904	COXSACKIEVIRUS B4	1	3	0	0	0	0	0	0	0	0	4
0905	COXSACKIEVIRUS B5	0	0	0	2	0	0	0	0	0	0	2
1004	ECHOVIRUS TYPE 4	0	3	0	6	0	0	0	0	0	1	10
1009	ECHOVIRUS TYPE 9	2	1	0	0	0	0	0	0	0	0	3
1014	ECHOVIRUS TYPE 14	0	0	0	1	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	1	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	2	1	0	1	0	1	0	0	0	0	5
1100	POLIOVIRUS NOT TYPED	0	2	0	0	0	4	0	0	0	0	6
1101	POLIOVIRUS TYPE 1	1	0	0	0	0	1	0	0	0	0	2
1102	POLIOVIRUS TYPE 2	1	1	0	0	0	1	0	0	0	0	3
1103	POLIOVIRUS TYPE 3	1	0	0	0	0	1	0	0	0	0	2
1104	POLIOVIRUS - MIXED VACCINAL ST	1	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	0	0	0	0	0	0	0	1	2
1301	HERPES SIMPLEX VIRUS - NOT TYP	36	1	1	0	0	0	0	0	0	15	53
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	7	3	1	0	0	0	1	0	0	0	12
1303	VARICELLA-ZOSTER VIRUS	3	1	0	0	0	0	0	0	0	8	12
1306	HERPES SIMPLEX TYPE 1	2	10	0	0	0	1	1	0	0	64	78
1307	HERPES SIMPLEX TYPE 2	3	0	0	0	0	0	0	0	0	75	78
1399	HERPES VIRUS TYPING PENDING	0	2	0	0	0	0	0	0	1	1	4
1401	COXIELLA BURNETII	6	2	0	0	0	0	1	0	0	0	9
1502	PICORNIA VIRUS - NOT TYPED = E	1	0	0	0	0	10	0	0	0	0	11
1521	MEASLES VIRUS	1	0	0	0	0	0	0	0	0	0	1
1522	RUBELLA VIRUS	2	0	0	0	0	0	0	0	0	8	10
1532	HEPATITIS B ANTIGEN	64	0	0	0	0	2	46	0	1	1	114
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	3	0	0	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	15	0	0	0	0	0	0	0	0	0	15
1556	CMV - CYTOMEGALOVIRUS	4	11	0	0	1	1	1	0	5	0	23
1564	ROTAVIRUS	2	0	0	0	0	51	0	0	0	0	53
1599	ENTEROVIRUS TYPING PENDING	0	6	0	1	0	6	0	0	0	1	14
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	1	0	0	0	0	0	0	0	0	1	2
9992	ROSS RIVER VIRUS	9	0	0	0	0	0	0	0	0	4	13
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	1	0	0	0	0	1
9995	DENGUE	1	0	0	0	0	0	0	0	0	1	2
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	1	0	0	2	0	0	0	0	0	1	4
TOTAL		198	628	2	14	3	96	53	2	8	183	1187

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 20/7/89 TO 02/8/89

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	2	0	0	0	0	0	0	1	0	0	3
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	0	1	0	0	1
0102	ADENOVIRUS TYPE 2	0	0	1	0	0	0	0	1	0	0	2
0103	ADENOVIRUS TYPE 3	8	0	0	0	0	0	0	0	1	0	9
0104	ADENOVIRUS TYPE 4	5	0	0	0	0	0	0	0	1	0	6
0108	ADENOVIRUS TYPE 8	0	0	0	0	0	1	0	0	1	0	2
0110	ADENOVIRUS TYPE 10	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	2	0	0	0	0	0	0	0	0	2	4
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	1	4	1	0	6
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	0	0	0	1	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	1	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	0	1	0	1
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	0	0	1
0904	COXSACKIEVIRUS B4	0	0	0	0	1	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	0	0	0	0	1	1
1030	ECHOVIRUS TYPE 30	0	0	0	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	0	1	0	3	4
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	1	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	3	0	0	0	0	0	0	0	0	3
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	43	0	0	0	0	0	0	0	0	44
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	19	3	4	1	0	0	3	0	30
1303	VARICELLA-ZOSTER VIRUS	1	0	0	0	0	0	0	0	0	0	1
1306	HERPES SIMPLEX TYPE 1	6	37	0	0	0	0	0	5	5	0	53
1307	HERPES SIMPLEX TYPE 2	0	120	0	1	0	0	0	1	4	0	126
1401	COXIELLA BURNETII	0	0	0	0	2	0	0	1	0	0	3
1522	RUBELLA VIRUS	0	0	0	2	1	0	0	0	2	0	5
1532	HEPATITIS B ANTIGEN	0	0	2	0	0	0	0	0	10	0	12
1541	CHLAMYDIA A - C. TRACHOMATIS	2	76	0	0	0	0	0	0	0	0	78
1556	CMV - CYTOMEGALOVIRUS	0	0	0	2	0	3	0	5	25	1	36
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	1	0	2	0	1	4
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	0	0	0	0	2	0	0	0	0	0	2
9992	ROSS RIVER VIRUS	0	0	0	1	25	0	0	2	3	0	31
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	1	0	0	1	1	0	3
TOTAL		28	279	22	9	36	6	1	30	58	8	477

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Notifiable Diseases - 1 January to 31 December 1988

The notifiable diseases data for the year have been referred back to State and Territory health authorities so that the correctness of data can be verified. In some instances these data will differ from the cumulative totals published in Period 13 (CDI 89/10).

DISEASE	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	TOTAL
Amoebiasis	8	6	2	38	4		2		60
Ankylostomiasis			1	17	15	2	NN		35
Anthrax									
Arbovirus infection	136	150	511	8	63		29		897
Brucellosis	1		13	1	1				16
Campylobacter infection	1875	NN	NN	1560	372	NN	265	10	4082
Chancroid		NN	1	NN	2	NN		1	4
Cholera	1				1				2
Congenital rubella syndrome			2			NN			2
Diphtheria	1						#60		61
Donovanosis	1	NN	62	NN	46		24		133
Giardiasis	497	NN	NN	967	284	NN	NN	5	1753
Genital herpes	954	227	852	NN	NN	NN	34	62	2129
Gonococcal ophthalmia neonatorum		NN		0	NN	NN	3	NN	3
Gonorrhoea	746	629	831	278	979	28	569	19	4079
Hepatitis A (infectious)	89	65	87	101	231	3	23	1	600
Hepatitis B (serum)	388	185	555	43	443	28	23	18	1683
Hepatitis - unspecified	15	16	24	11	NN	NN	1	2	69
Hydatid disease	5	1	3	2	1	2		1	15
Lassa fever									
Legionnaires disease	26	8	4	26	2	NN	1	NN	67
Leprosy	7		3	1	5		4		20
Leptospirosis	36	25	22	6	4	11			104
Lymphogranuloma venereum		NN		NN	NN	NN	NN		

Cutaneous cases only

DISEASE	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	TOTAL
Malaria	84	65	332	30	42	2	20	26	601
Marburg disease									
Measles	43	NN	174	14	16	NN	NN	1	248
Meningococcal infections	18	20	21	29	13	NN	23	2	126
Non-specific urethritis	3136	NN	1	NN	NN	NN	73	NN	3210
Ornithosis	5	5	1	8				2	21
Pertussis (whooping cough)	25	40	NN	57	31	NN			153
Plague									
Poliomyelitis									
Q fever	232	1	167	21	3				424
Rabies						NN		NN	
Salmonella infections	1048	179	1116	398	366	113	240	24	3484
Shigella infections	99	16	152	53	76	3	181	1	581
Smallpox									
Syphilis	1158	65	939	92	194	2	598	8	3056
Tetanus	1		1	2	1				5
Trachoma		NN	NN	130	138		NN		268
Tuberculosis (all forms)	406	307	167	95	127	19	26	18	1165
Typhoid fever	25	8	4		3			1	40
Typhus (all forms)		1	3	1	3				8
Vibrio parahaemolyticus infections	1	NN	NN		1	NN		NN	2
Yellow fever									
Yersinia infections	124		NN	44	3	NN	1	NN	172

NN - Not notifiable

(Note: Data collected under the National Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon reporting by medical practitioners etc.)