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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 1,381 reports were processed during this period.

Poliovirus

- type 1 was isolated from the nasal aspirate of a 3 month old male with brochiolitis;
- mixed vaccinal strains were isolated from the nasal aspirates of 3 infants with symptoms of respiratory tract infections:
  - a 3 month old male with persistent cough,
  - a 2 month old female with severe rhinorrhea, and
  - a 2 month old female presenting with choking episodes.

Rubella antibodies were serologically detected in a 2 month old male with microcephaly. Congenital rubella infection was suspected.

Cytomegalovirus was isolated from the nasal aspirates of a 2 month old female and a 3 month old female with unspecified upper respiratory tract diseases. The significance of these isolates is yet to be determined.

*Chlamydia trachomatis* was isolated from vaginal swabs of a 22 year old female with amenorrhea.


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- Contributions are solicited, and do not preclude later publication elsewhere.


- Figures given may be subject to revision.

## OVERSEAS BRIEFS: DENGUE FEVER IN FIJI

An epidemic of dengue fever started in the Pacific reagion in April 1989.

To date Fiji reported:
. 15 serologically confirmed cases in July and August with a further
. 42 cases reported in September.

Cases of dengue-like illness are currently being reported from central and western divisions and include a few haemorrhagic cases. Some of these clinical cases are awaiting serological confirmations.

## AIDS AND HIV SURVEILLANCE, AUSTRALIA - 1 NOVEMBER 1989

### AIDS surveillance

To 1 November 1989, 1,481 cases of AIDS fulfilling the criteria of case definition were reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification (Table 1), by age group (Table 2), by risk category (Tables 3 A &3b) and by clinical presentation (Table 4) are shown below.

TABLE 1: AIDS patients by State or Territory of notification

| STATE/ TERRITORY | CASES | | | KNOWN DEATHS | | |
|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total |
| NSW | 923 | 30 | 953 | 526 | 20 | 546 |
| VIC | 317 | 7 | 324 | 141 | 2 | 143 |
| QLD | 98 | 4 | 102 | 59 | 4 | 63 |
| WA | 70 | 4 | 74 | 27 | 1 | 28 |
| SA | 51 | 2 | 53 | 20 | 1 | 21 |
| NT | 2 | 0 | 2 | 1 | 0 | 1 |
| TAS | 4 | 1 | 5 | 2 | 0 | 2 |
| ACT | 16 | 0 | 16 | 10 | 0 | 10 |
| TOTAL | 1481 | 48 | 1529 | 786 | 28 | 814 |

TABLE 2: AIDS patients by age group

| AGE (YEARS) | CASES | | | KNOWN DEATHS | | |
|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total |
| 0 - 9 | 10 | 2 | 12 | 6 | 1 | 7 |
| 10 - 19 | 7 | 2 | 9 | 3 | 1 | 4 |
| 20 - 29 | 302 | 15 | 317 | 156 | 4 | 160 |
| 30 - 39 | 639 | 6 | 645 | 334 | 2 | 336 |
| 40 - 49 | 378 | 5 | 383 | 196 | 4 | 200 |
| 50 - 59 | 117 | 7 | 124 | 66 | 6 | 72 |
| 60 + | 28 | 11 | 39 | 25 | 10 | 35 |
| TOTAL | 1481 | 48 | 1529 | 786 | 28 | 814 |

Beginning with this issue, tabulation of AIDS cases by transmission category will be presented separately for adults and adolescent cases (aged 14 yrs and older, Table 3A) and for paediatric cases (aged 13 yrs and younger, Table 3B). These categories broadly derive from the system of classification of cases used by the Centers for Disease Control, Atlanta, Georgia and are likely to be further modified in the future to reflect additional patterns of exposure evident in Australia.

Starting from this issue the category 'blood transfusion' has been expanded to include recipients of other blood products or tissue, and will be listed as 'Other blood/blood products/tissue recipients'.

TABLE 3A: AIDS patients by risk/transmission category: adults and adolescents 14 yrs and older

| TRANSMISSION/RISK CATEGORY | CASES | KNOWN DEATHS |
|---|---|---|
| Homosexual/Bisexual | 1352 | 711 |
| IV drug user: | (60) | (25) |
| . Homosexual/Bisexual IV drug user | 42 | 20 |
| . Heterosexual IV drug user | 18 | 5 |
| Person with haemophilia | 15 | 7 |
| Other blood/blood products/tissue recipient | 50 | 44 |
| Heterosexual transmission | 24 | 10 |
| None of the above | 7 | 4 |
| Under investigation | 6 | 4 |
| TOTAL | 1514 | 805 |

TABLE 3B: AIDS patients by risk/transmission category: Paediatric cases (13 years or less)

| TRANSMISSION CATEGORY | CASES | KNOWN DEATHS |
|---|---|---|
| Haemophiliac | 2 | 1 |
| Other blood/blood products/tissue recipeint | 10 | 7 |
| Mother with/at risk or AIDS/HIV | 3 | 1 |
| | 15 | 9 |

TABLE 4: AIDS patients by clinical presentation*

| INITIAL DISEASE REPORTED | CASES | KNOWN DEATHS |
|---|---|---|
| GROUP IV: | | |
| B: Neurological disease | 41 | 20 |
| C: Secondary infectious diseases | 1103 | 581 |
| D: Secondary cancers | 299 | 158 |
| E: Other conditions | 12 | 5 |
| BC: Neurological disease + infectious diseases | 11 | 9 |
| BD: Neurological disease + cancers | 1 | 1 |
| CD: Infectious diseases + cancers | 53 | 36 |
| TOTAL | 1520 | 810 |

* The data in Table 4 includes both presumptive (clinical) and definitive diagnoses. However, cases which have not been designated as one of these two categories have not been included.

## HIV surveillance

*Notifications of persons newly discovered to be infected with HIV*

TABLE 5: Notifications of persons newly diagnosed as infected with HIV by State and Territory, 1989

| STATE/ TERRITORY | 1989 Weeks 27-30 | 1989 Weeks 31-34 | 1985-1989 Cumulative to week 34 |
|---|---|---|---|
| NSW | N/A* | N/A* | N/A* |
| VIC | 23 | 34 | 2106 |
| QLD | 11 | 15 | 760 |
| WA | 9 | 6 | 466 |
| SA | 5 | 9 | 333 |
| TAS | 3 | 2 | 45 |
| ACT | N/A | N/A | 92 |
| NT | 1 | 1 | 91 |
| TOTAL | 52 | 67 | 3893 |

N/A = NOT AVAILABLE

* Notifications from New South Wales are not yet available, but will be available prospectibely and retrospectively by the start of 1990.

TABLE 6: Notifications of persons newly diagnosed as infected with HIV by presumed transmission category (data from NSW, ACT and NT not yet available

| PRESUMED TRANSMISSION CATEGORY | Weeks 27-30 | Weeks 31-34 | Cumulative Total 1985-1989 Number | (%) |
|---|---|---|---|---|
| Homo/bisexual males | 36 | 47 | 2966 | (80.0) |
| Heterosecual IVDU | 5 | 8 | 175 | ( 4.7) |
| ( - males) | (4) | (7) | (134) | |
| (- females) | (1) | (1) | (41) | |
| Homo/bisexual males IVDU | 4 | 2 | 97 | ( 2.6) |
| Haemophilia | - | - | 180 | ( 4.9) |
| Heterosexual contact | 1 | 2 | 94 | ( 2.5) |
| ( - males) | (1) | (2) | (54) | |
| (- females) | (-) | (-) | (40) | |
| Blood transfusion | 1 | - | 51 | ( 1.4) |
| Other/not specified | 5 | 6 | 147 | ( 4.0) |
| TOTAL | 52 | 67 | 3710 | (100.0) |

TABLE 7: Donations tested for antibody for HIV at Red Cross Blood Transfusion Services by State and Territory

| STATE/ TERRITORY | DONATIONS TESTED 1988 Cumulative to week 34 | DONATIONS TESTED 1988 Weeks 31-34 | CUMULATIVE HIV +VE DONORS, 1985-89 Cumulative to week 34 | CUMULATIVE HIV +VE DONORS, 1985-89 Weeks 31-34 | |
|---|---|---|---|---|---|
| NSW | 188,728 | 22,236 | 184,591 | 21,659 | 21 |
| VIC | 178,084 | 20,729 | 171,933 | -18,833 | 5 |
| QLD | 93,299 | 11,816 | 91,021 | N/A | 5 |
| WA | 47,528 | 5,705 | 47,910 | 2,890* | 1 |
| SA | 63,818 | 7,541 | 61,830 | 7,393 | - |
| TAS | 16,003 | 2,309 | 17,228 | 1,962 | - |
| ACT | 12,327 | 1,573 | 13,230 | 1,629 | - |
| NT | 5,993 | 679 | 6,096 | 825 | - |
| TOTAL | 605,780 | 72,189 | 593,839 | 55,191 | 32 |

* Weeks 31-32 only

| STATE/ TERRITORY | 1988 Cumulative to week 34 | 1988 Weeks 31-34 | 1989 Cumulative to week 34 | 1989 Weeks 31-34 |
|---|---|---|---|---|
| NSW | 110,207 | 12,473 | 70,223 | 6,365 |
| VIC | 45,284 | 5,323 | 44,571 | 5,112 |
| QLD | 35,538 | 4,577 | 46,722 | 6,803 |
| WA | 15,028 | 1,848 | 17,938 | 2,579 |
| SA | 20,255 | 1,848 | 17,938 | 2,579 |
| TAS | 4,238 | 569 | 4,576 | 691 |
| ACT | 3,284 | 414 | 3,436 | 369 |
| NT | 5,137 | 629 | 5,467 | 817 |
| TOTAL | 167,434 | 22,085 | 207,297 | 21,837 |

Date not yet available from:
- Royal Prince Alfred Hospital, Sydney: 1988, 1989 - all weeks
- Royal North Shore Hosptial, Sydney: 1989 - weeks 17 to 34
- St Vincent's Hospital, Sydney: 1989 - weeks 17 to 34
- St George Hospital, Sydney: 1989 - weeks 29 to 34
- RAAF, Richmond: 1989 - all weeks
- Army, Yeronga: 1989 - all weeks
- Royal Newcastle Hospital: 1989 - all weeks
- Greenslopes Hospital, Brisbane: 1989 - all weeks
- Mater Hospital, Brisbane: 1989 - weeks 25 to 32
- Princess Alexandra Hospital, Brisbane: 1989 - weeks 29 to 32
- Royal Perth Hospital: 1989 - weeks 33 to 34
- IMVS, Adelaide: 1989 - weeks 25 to 28

## HANTAAN VIRUS INFECTION OF LABORATORY RODENTS - AUSTRALIA

(Contributed by Margery Kennett, Virology Laboratory, Fairfield Hospital, Melbourne)

Hantaan and related viruses are the causative agents of haemorrhagic fever with renal syndrome (HFRS) which may occur when humans come into close contact with infected rodents. Although HFRS usually occurs in rural areas and is occasionally a hazard for laboratory staff handling rodents or rodent tumour and cell lines for research.

Four distinct types of Hantaan-related viruses have been identified. These share antigenic and genetic characteristics and comprise the genus Hantavirus of the virus family Bunyaviridae. Hantaan virus was first isolated by Lee and his co-workers from the lungs of infected *Apodemus agrarius* mice captured in Korea, in the areas where Korean Haemorrhagic Fever (KHF) is endemic. They were able to demonstrate that this virus was probably the aetiological agent for KHF. The distribution of Hantaan virus while not known with certainty is likely to follow that of its primary host *Apodemus agrarius* ie. throughout much of Asia and the Soviet Union where epidemic haemorrhagic fever may occur.

The rat or urban-associated Hantaan-like virus Seoul has been associated with a mild form of HFRS in urban centres in Korea and China and with laboratory workers in several countries. A Hantaan-like virus isolated from domestic rats captured in the United States appears similar to Hantaan virus in cross immunfluorescence antibody tests (IFA) but distinct in plaque reduction neutralisation tests. Antibody studies in sera from rats captured in may areas of the world have shown that

rat-associated Hantaan-like virus is present in domestic rats - not only in ports but in habitats where rodent control programmes were inadequate or non existent. The various rat viruses which are similar regardless of georgraphic origin can be represented by the 'Seoul' type.

Puumala virus, another distinct Hanta virus has been isolated from bank voles (Clethrionomys glareolus) in Finland. This agent causes nephropathia epidemica (NE), a relatively mild form of HFRS in Scandinavia, the Western Soviet Union and much of Europe.

The fourth type, Prospect Hill virus was isolated from the meadow vole *Microtus pennsylvanicus* in Maryland, USA. At present no human disease has been attributed to infection with Prospect Hill although antibody to the virus has been identified among people working with mammals in the USA and in small mammals in areas in North America.

Infection with Hantaan virus is inapparent in laboratory rodents which may secrete large quantities of virus in saliva, urine and faeces for life. The route of transmission to man is by inhalation of aerosol although rodent bites may also be implicated.

In March 1989, the Murine Virus Monitoring Scheme (MVMS) of the South Australian Central Veterinary Laboratory (VETLAB) commenced screening sera from colonies of laboratory rodents using the indirect immunofluorescence test (IFAT) and inactivated Hantaan virus. Initially only small numbers of samples (not a statistically based sampling schedule) from a limited number of colonies were tested. Positive sera from several different colonies of rats and mice were identified. The significance of these findings is uncertain as the immunofluorescence test detects antibodies to Hantaan, known related Hanta viruses and possibly to unknown agents related to Hantavirus.

To determine whether the animals are infected with Hantaan or other viruses, the CSIRO Australian Animal Health Laboratory (AAHL) has now commenced virus isolation attempts (in cell culture and rats) from the tissues of animals with high IF titres of antibody. The work is being done under C3 containment conditions. Cell culture work is done in class III flexible film isolators and rat inoculation by workers in fully enclosed "suits". The work may take several months.

Institutions which handle laboratory animals, particularly rats ("stroke-prone" or other Japanese stock primarily) are urged to contact MVMS for advice on the collection of samples, so that a thorough screening programme can be achieved.

Provided an adequate sample of animals has been tested rats from antibody negative colonies can be used. Until stock have been shown to be antibody negative, personnel should handle animals with extreme care. Since inhalation of aerosol is the principal route of infection in humans, a barrier between the animals and personnel is required.

If Hanta infected animals are present in a colony, the most prudent method of control is to sacrifice the animals, disinfect the facility and restock it. If valuable rat strains are to be preserved, caesarean derivation using prescribed containment and surveillance methods should be instituted.

Personnel who have been in contact with rodents and are concerned about the possibility of exposure to Hanta virus may have sera tested by the Virus Laboratory, Fairfield Hosptial. To date we have no evidence of disease in Australia associated with Hantaan virus. Since 1983, the Virus Laboratory at Fairfield has been testing sera from patients suspected of having haemorrhagic fever with renal syndrome (HFRS) using IFAT. To date no patients have seroconverted, however sera from different patients had low titres during the acute or early convalescent stage of illness, while bleeds collected several months later were negative. In confirmed HFRS patients IF antibody appears during the first week of symptoms and lasts as long as 34 years.

The IF test perfomed at both MVMS and Fairfield detects a Hanta group antibody. The source and prior extraction of antigen affects the results of the enzyme-linked immunosorbent assay (ELISA) while plaque neutralization tests are type specific perhaps with minor cross reactions. Inactivated ELISA Hantaan antigen supplied by the United States Army Medical Institute of Infectious Diseases, Fort Detrick will be used for further testing of human and rodent sera at AAHL, MVMS and Fairfield.

For further information please contact:

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## LEPTOSPIROSIS, FRANCE

(Based on Wkly Epidem Rec 1989;64:310-1)

The prevalence of leptospirosis in France has increased considerably in the last 2 years [1]. The disease mainly affects people who do not belong to recognised groups routinely at risk for the disease. It might be advisable to in the near future, redefine groups at risk for the disease if the present epidemiological situation persists. The occupational risk is now diminishing on account of the precautions taken to prevent the disease, whereas the risk of infection associated with open-air activities such as bathing, fishing, hunting, country walks, etc is constantly increasing.

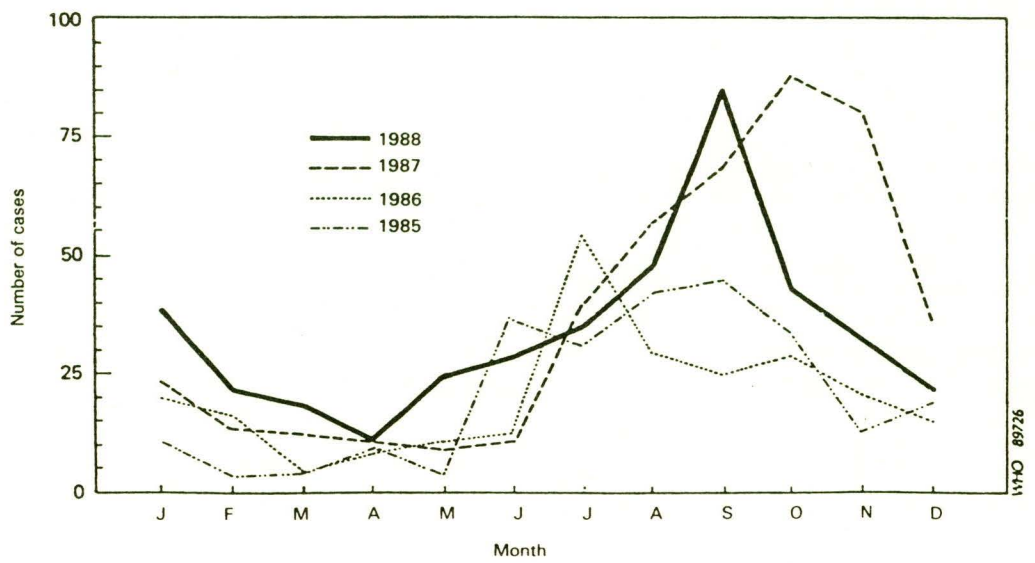
**Trends between 1985 and 1988**

Although the number of serum samples received at the reference centre remained relatively constant, the percentage of positive samples has almost doubled since 1985: with a total of 670 cases of leptospirosis reported in France in 1988 (400 cases in metropolitan France and 270 cases overseas).

In metropolitan France, the number of cases remained relatively constant between 1985 and 1986. An upsurge in the number of cases was observed in autumn 1987 and during the first 6 months of 1988. The increase in the number of cases was distributed fairly evenly over the whole country, but particularly marked in the south-west where the number of positive serum samples doubled between 1986 and 1987. Climatic conditions and possible variations in the public's habits (holiday periods, spare-time activities) do not satisfactorily explain this recrudescence of the disease, the cause of which may have to be sought in ecological changes affecting the biotopes of wild rodents.

The increase in the number of cases in overseas France is more artificial because the figures for the last 2 years include the diagnosis made in New Caledonia, which previously were not recorded.

Figure 1: Monthly distribution of leptospirosis cases, France, 1985-1988



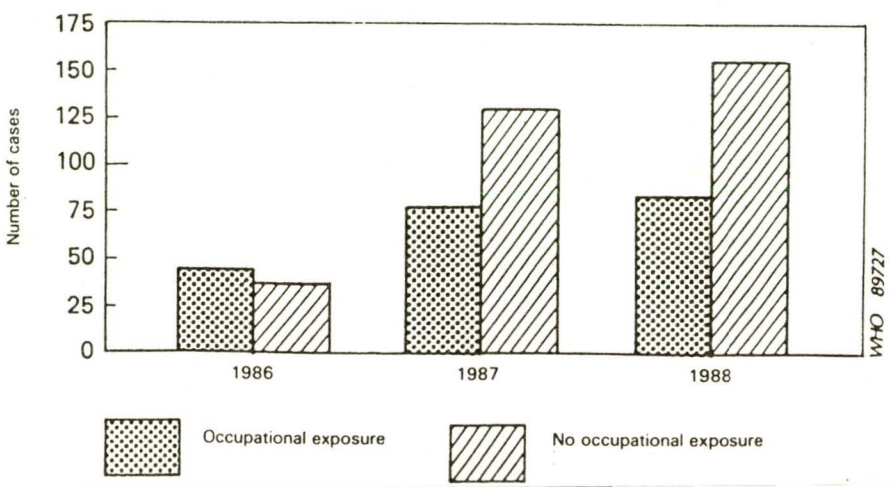
The monthly distribution (Fig 1) shows a summer/autumn peak which has a tendency to occur later (particularly late peak in 1987, not accounted for).

The distribution of serovars indicated an increased prevalence of serogroup *Grippotyphosa* which rose from 9.8% of leptospirosis cases in 1985 to 27% in 1988. *Grippotyphosa* is the second most important agent in metropolitan France since *Iceterohaemorrhagiae* is responsible for 35.7% of cases. Along the Atlantic coast and in the south-west, an area where the disease is firmly established, *Grippotyphosa* causes more cases than *Iceterohaemorrhagiae*. The serogroup *Australis* is in third place with 10% of cases, a prevalence that has remained constant for 3 years. In overseas France *Iceterohaemorrhagiae* is predominant, and little change has been noted in recent years.

**Occupations risk**

Against the background of annual increase in the number of cases of occupational leptospirosis, the increase in the number of cases in people not occupationally exposed is more marked, highlighting the importance of leisure activities (especially water sports) in the spread of the disease. The occupations traditionally regarded as exposed are agricultural workers, refuse collectors, sewage workers, occupations involving the handling of meat or foodstuffs and all occupations practised in a humid environment frequented by rats (plumbers, miners, seamen, waterworks and forestry employees, etc). It is still farm-workers who are most affected by the disease: they account for 59% of the cases occuring in the exposed occupations. In this occupational category *Grippotyphosa* was predominant in 1986 and 1987. *Iceterohaemorrhagiae* returned to the fore in 1988 with 50% of cases (Fig 2). These 2 serogroups clearly predominate in the occupational leptospiroses with 77% of cases. In patients with no occupational risk *Iceterohaemorrhagiae* and *Grippotyphosa* still predominate with 58% of all cases, but a wider range of serovars is involved on account of the variety of possible sources of infection. Schoolchildren and students are mainly infected with *Iceterohaemorrhagiae*, whereas retired people are more often infected by *Grippotyphosa*. The former, more involved in water sports, are more exposed to infection via water polluted with rat urine and thus to the serogroup *Iceterohaemorrhagiae*; the latter are more exposed to infection by serogroups carried by land rodents or cattle, which accounts for the greater importance of the *Grippotyphosa* group.

Figure 2: Distribution of leptospirosis cases accordingly to occupation exposure, French 1986-1988



Clinical symptoms

Fever is present in 87% of cases, associated with a pain syndrome in 44% of cases. There is liver or kidney involvement in 21% of cases; such involvement is twice as frequent in *Iceterohaemorrhagiae* patients as in *Grippotyphosa* patients. Meningeal involvement occurs frequently (35%), somewhat more frequently when *Grippotyphosa* is the cause. The pure febrile forms or pseudoinfluenzal forms are more common than the classical icterohaemorrhagic form with renal insufficiency. The pulmonary forms are rare (5%). These findings, which are for the year 1988, are very similar to those for 1987.

There is little change in the age and sex distribution of cases. This disease primarily affects males (80% of cases), and the average age of reported cases is approximately 43 years.

## HUMAN PLAGUE IN 1988

(Based on WER 1989;64:345-7 and WER 1988;63:360-2)

Human plague was reported from 9 countries in 1988, giving a total of 1363 cases with 134 deaths. This is the highest number of cases since 1977, but more deaths occured in 1987 (Table 1).

TABLE 1: HUMAN PLAGUE: NUMBER OF CASES (AND DEATHS) REPORTED IN THE WORLD, 1974-1988

| | 1974 | 1975 | 1976 | 1977 | 1978 | 1979 | 1980 | 1981 | 1982 | 1983 | 1984 | 1985 | 1986 | 1987 | 1988 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| AFRICA | 183 | 147 | 93 | 172 | 203 | 251 | 86 | 59 | 290 | 594 | 650 | 215 | 729 | 853 | 1109 |
| | (25) | (41) | (35) | (41) | (15) | (15) | (22) | (19) | (43) | (59) | (59) | (41) | (90) | (198) | (138) |
| AMERICAS | 321 | 521 | 146 | 48 | 97 | 23 | 142 | 128 | 182 | 225 | 500 | 128 | 162 | 88 | 52 |
| | (8) | (9) | (9) | (11) | (5) | (2) | (7) | (12) | (4) | (12) | (42) | (9) | (19) | (9) | (5) |
| ASIA | 2252 | 811 | 1266 | 1258 | 485 | 387 | 283 | 13 | 281 | 248 | 196 | 143 | 112 | 114 | 202 |
| | (130) | (52) | (60) | (26) | (14) | (16) | (29) | (-) | (1) | (21) | (6) | (8) | (6) | (8) | (10) |
| WORLD TOTAL | 2756 | 1479 | 1505 | 1478 | 785 | 661 | 511 | 200 | 753 | 1067 | 1346 | 486 | 1003 | 1055 | 1363 |
| | (163) | (102) | (104) | (78) | (34) | (33) | (58) | (31) | (48) | (92) | (107) | (58) | (115) | (215) | (134) |

In 1988, as in previous years, the epidemiological situation of plague in the world was determined by outbreaks in a number of African countries, with 75% of the world total from the United Republic of Tanzania and Zaire. The global case-fatality rate was also determined by the rate in those two countries as their number of deaths represented 78% of the world total (Table 2).

TABLE 2: HUMAN PLAGUE: NUMBER OF CASES (AND DEATHS) REPORTED IN 1988, BY COUNTRY

| | | | |
|---|---|---|---|
| Boliva | 2 (-) | United Republic of Tanzania | 647 (33) |
| Brazil | 25 (-) | United States of America | 15 (-) |
| China | 6 (4) | Vietnam | 196 (6) |
| Madagascar | 93 (19) | Zaire | 369 (86) |
| Peru | 10 (5) | | |

Americas

In Bolivia, cases were detected in February in one of the districts of Franz Tamayo Province (La Paz Department).

In Brazil, cases were recorded in Bahia State, especially in Serrinha municipality which recorded cases in January, March, June, August, September and November. This pattern of spread is typical of the epidemiology of plague occurring against the background of epizootics among wild rodents. In Peru, human plague occurred in 2 provinces of Piura Department; in Sayo District, 1 case was recorded on 12 January, 5 cases from 5 to 8 February, and 1 case on 10 February. This pattern of spread with an interval of 20-25 days between the first case and the following ones, was repeated in Canchaque district and is typical of the epidemiology of anthroponotic bubonic plague when the causative agent is transmitted through the bite of human fleas (in partucilar *Pulex irritans*). Although the territory of these Brazilian outbreaks is limited, there is cause for concern as they are manifestations of the form of plague which often leads to the appearance of large epidemics. It is necessary to take active measures for flea control in human dwellings, not only disinsection but measures which exclude the possibility of flea hatching (sweeping floors in houses, filling up cracks and chinks with clay etc).

In the United States of America, plague cases were recorded in Texas (1 case), Arizona (1 case), California (2 cases), Colorado (4 cases) and New Mexico (7 cases). Ten of the cases were diagnosed during July and August. The pattern of plague in the United States is consistent with that observed over many years, that is, of active foci of wild rodent infection.

Asia

As in previous years, human plague was recorded in Vietnam and in China. In Vietnam, cases were detected throughout the year in the well-known plague endemic Provinces of Dac Lac, Gua-Lai-Cong Tum, Lam Dong, Nghia Binh and Phu Khanh.

Africa

Human plague was reported from well-known enzootic areas of Madagascar: the Provinces of Antananarivo and Fianarantsoa.

In the United Republic of Tanzania, there have been non-stop outbreaks of human plague since 1983. This may be related to the low intensity of generally accepted measures taken there to detect and eliminate plague outbreaks. In Zaire, sporadic cases have been reported since 1958 but the apparant relaxation of epidemiological surveillance in recent years has lead to the outbreaks of 1987 (474 cases and 160 deaths) and 1988 (369 cases and 86 deaths). Preventive and prophylactic measures such as deratizaton and disinsection in settlements and surrounding areas regularly affected by plague in these countries should be intensified.

In conclusion, it should be stated that the current incidence of plague in the world generally reflects the pattern of plague epidemicity in its natural foci, with the appearance of relatively limited outbreaks against a background of epizootics among synanthropic rodents, rats in particular, in limited areas of several countries.

CDI Editorial Comment

Countries which reported human cases of plague to the WHO between 1 January and 16 November 1989 are Brazil, Madagascar, Mongolia, Myanmar (formerly Burma), Tanzania, USSR, USA, Vietnam and Zaire. On 16 November 1989, the countries listed by the WHO as having areas which were 'infected' with human plague were Bolivia, Brazil, Madagascar, Peru, Tanzania, Vietnam and Zaire. This list is compiled using information supplied to the WHO by signatories of the International Health Regulations; the criteria for being included on the list include occurrence of human cases and plague infection among domestic or wild rodents in sufficient proximity to ports and airports to be a threat to international traffic [1].

Human plague has a seasonal distribution that is related both to timing of epizootics in rodent populations and to periods where humans are in contact with rodent reservoirs. Thus in Tanzania, Zaire and Vietnam, most cases have occurred in January-April, which coincides with mild temperatures and rodent epizootics. In the USA, most cases have occurred during May-October, when people spend more time outdoors and are likely to have contact with rodent fleas.

In the epidemiology of plague, man is an accidental host for the plague bacillus, *Yersinia pestis*. Virtually all human cases derive from bites by infected rodent fleas. Human outbreaks coincide with epizootics, which are signalled by die-offs of the susceptible rodents; during die-off, fleas leave their dead hosts in search of a new host, and may find a human [2].

In endemic areas control measures for plague remain rodent control, insecticide use and public health education for people to avoid contact with rodents and their fleas.

The International Health Regulations also provide for controls to prevent the spread of plague by international travel to non-endemic areas. These include regular inspection and deratting of ships used in international traffic and the quarantine of infected travellers on arrival [1].

The plague vaccine, which has not been proved to be efficacious in properly controlled trials, is only recommended for laboratory and field personnel who are working with *Y. pestis* organisms resistant to antibiotics, and persons engaged in field operations in plague-enzootic areas, where preventing exposure can be difficult (such as in some disaster areas) [3]. Vaccination against plague is not required for entry into any country. Treatment of plague involves administration of streptomycin, tetracycline or chloramphenicol, and is highly effective, especially if commenced in the first 1-2 days of illness [2].

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## YELLOW FEVER POLICY - AUSTRALIA

As of 1 October 1989, Australia has introduced a revised yellow fever quarantine policy for international travellers arriving in Australia.

The policy reads as follows:

1. All persons over 1 year of age arriving in Australia and who have within the previous six (6) days been in that part of a country which has been reported as currently yellow fever infected by the World Health Organization in the *Weekly Epidemiological Record* must hold valid international yellow fever vaccination certificates, or they will be subject to quarantine.

2. Persons without a valid international certificate of vaccination against yellow fever are to be placed under quarantine surveillance for a period expiring 6 days after leaving a yellow fever infected area.

   Surveillance requirements are as follows:

   (a) Outside Queensland:
   (i) undertake to request permission of the QMO before travelling to Queensland.

   (ii) undertake to notify the QMO if diagnosed as suffering from yellow fever.

   (b) Inside Queensland:
   (i) undertake to telephone the QMO daily until released from quarantine surveillance to report any symptoms which might originate from yellow fever.

   (ii) undertake to notify the QMO if diagnosed as suffering from yellow fever.

3. Persons suffering from yellow fever are to be placed in quarantine.

This revised policy has been brought to the notice of WHO and airline carriers.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 09/11/89 TO 22/11/89

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

| | | 019 | 065 | 110 | 111 | 112 | 113 | 114 | 115 | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|
| 0100 | ADENOVIRUS NOT TYPED | 4 | 2 | 2 | 0 | 2 | 4 | 0 | 20 | 34 |
| 0101 | ADENOVIRUS TYPE 1 | 3 | 1 | 4 | 0 | 5 | 0 | 0 | 0 | 13 |
| 0102 | ADENOVIRUS TYPE 2 | 3 | 0 | 5 | 0 | 7 | 0 | 0 | 0 | 15 |
| 0103 | ADENOVIRUS TYPE 3 | 3 | 1 | 6 | 0 | 1 | 0 | 0 | 0 | 11 |
| 0104 | ADENOVIRUS TYPE 4 | 5 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 8 |
| 0105 | ADENOVIRUS TYPE 5 | 0 | 0 | 1 | 0 | 2 | 0 | 0 | 0 | 3 |
| 0111 | ADENOVIRUS TYPE 11 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0119 | ADENOVIRUS TYPE 19 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0130 | ADENOVIRUS TYPE 30 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0199 | ADENOVIRUS TYPING PENDING | 0 | 0 | 0 | 11 | 0 | 0 | 0 | 0 | 11 |
| 0201 | INFLUENZA A VIRUS | 0 | 7 | 0 | 0 | 5 | 1 | 0 | 0 | 13 |
| 0203 | INFLUENZA B VIRUS | 2 | 2 | 1 | 2 | 2 | 0 | 0 | 2 | 11 |
| 0301 | PARAINFLUENZA VIRUS TYPE 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 0302 | PARAINFLUENZA VIRUS TYPE 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0303 | PARAINFLUENZA VIRUS TYPE 3 | 2 | 2 | 10 | 18 | 11 | 5 | 3 | 8 | 59 |
| 0399 | PARAINFLUENZA VIRUS TYPING PEN | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 4 |
| 0400 | RESPIRATORY SYNCYTIAL VIRUS (R | 8 | 0 | 6 | 0 | 0 | 1 | 1 | 3 | 19 |
| 0500 | RHINOVIRUS (ALL TYPES) | 6 | 8 | 6 | 11 | 2 | 1 | 0 | 0 | 34 |
| 0600 | MYCOPLASMA PNEUMONIAE | 17 | 2 | 11 | 1 | 2 | 0 | 0 | 0 | 33 |
| 0700 | ORNITHOSIS-PSITTACOSIS | 3 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 4 |
| 0899 | COXSACKIEVIRUS GROUP A TYPING | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0903 | COXSACKIEVIRUS B3 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1009 | ECHOVIRUS TYPE 9 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1011 | ECHOVIRUS TYPE 11 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1022 | ECHOVIRUS TYPE 22 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 1030 | ECHOVIRUS TYPE 30 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1100 | POLIOVIRUS NOT TYPED | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 2 |
| 1101 | POLIOVIRUS TYPE 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1103 | POLIOVIRUS TYPE 3 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 |
| 1104 | POLIOVIRUS - MIXED VACCINAL ST | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1200 | MUMPS VIRUS | 2 | 1 | 0 | 0 | 0 | 3 | 0 | 0 | 6 |
| 1300 | HERPES VIRUS GROUP - NOT TYPED | 0 | 1 | 0 | 0 | 4 | 2 | 0 | 0 | 7 |
| 1301 | HERPES SIMPLEX VIRUS - NOT TYP | 10 | 0 | 3 | 0 | 44 | 0 | 4 | 88 | 149 |
| 1302 | EPSTEIN-BARR VIRUS (EB VIRUS) | 5 | 10 | 25 | 3 | 0 | 1 | 0 | 1 | 45 |
| 1303 | VARICELLA-ZOSTER VIRUS | 7 | 5 | 1 | 1 | 4 | 6 | 0 | 0 | 24 |
| 1306 | HERPES SIMPLEX TYPE 1 | 50 | 26 | 29 | 0 | 5 | 4 | 0 | 1 | 115 |
| 1307 | HERPES SIMPLEX TYPE 2 | 62 | 73 | 11 | 0 | 37 | 17 | 0 | 0 | 200 |
| 1399 | HERPES VIRUS TYPING PENDING | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 5 |
| 1401 | COXIELLA BURNETII | 0 | 0 | 1 | 0 | 5 | 0 | 0 | 0 | 6 |
| 1502 | PICORNIA VIRUS - NOT TYPED = E | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 10 | 13 |
| 1514 | MOLLUSCUM CONTAGIOSUM | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1521 | MEASLES VIRUS | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1522 | RUBELLA VIRUS | 11 | 4 | 22 | 0 | 8 | 0 | 2 | 0 | 47 |
| 1532 | HEPATITIS B ANTIGEN | 10 | 11 | 15 | 0 | 39 | 7 | 1 | 24 | 107 |
| 1535 | HEPATITIS A ANTIBODY | 0 | 2 | 5 | 0 | 1 | 0 | 0 | 0 | 8 |
| 1541 | CHLAMYDIA A - C. TRACHOMATIS | 33 | 41 | 30 | 0 | 3 | 3 | 0 | 12 | 122 |
| 1556 | CMV - CYTOMEGALOVIRUS | 46 | 1 | 3 | 6 | 3 | 6 | 3 | 3 | 71 |
| 1564 | ROTAVIRUS | 15 | 0 | 63 | 0 | 13 | 18 | 2 | 0 | 111 |
| 1566 | NORWALK AGENT | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 2 |
| 1599 | ENTEROVIRUS TYPING PENDING | 0 | 0 | 0 | 7 | 0 | 3 | 2 | 0 | 12 |
| 9992 | ROSS RIVER VIRUS | 3 | 3 | 23 | 0 | 0 | 4 | 0 | 0 | 33 |
| 9994 | SMALL VIRUS (LIKE) PARTICLE | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 3 |
| TOTAL | | 318 | 210 | 287 | 66 | 211 | 91 | 22 | 176 | 1381 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 09/11/89 TO 22/11/89

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 | 0 | 14 | 0 | 0 | 0 | 13 | 0 | 0 | 0 | 3 | 30 |
| 0101 | ADENOVIRUS TYPE 1 | 2 | 6 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 9 |
| 0102 | ADENOVIRUS TYPE 2 | 4 | 9 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 15 |
| 0103 | ADENOVIRUS TYPE 3 | 1 | 4 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 6 |
| 0104 | ADENOVIRUS TYPE 4 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 |
| 0105 | ADENOVIRUS TYPE 5 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 0130 | ADENOVIRUS TYPE 30 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0199 | ADENOVIRUS TYPING PENDING | 0 | 9 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10 |
| 02[illegible] | INFLUENZA A VIRUS | 0 | 12 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12 |
| 02[illegible] | INFLUENZA B VIRUS | 1 | 9 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10 |
| 0301 | PARAINFLUENZA VIRUS TYPE 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0302 | PARAINFLUENZA VIRUS TYPE 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0303 | PARAINFLUENZA VIRUS TYPE 3 | 8 | 50 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 58 |
| 0399 | PARAINFLUENZA VIRUS TYPING PEN | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 0400 | RESPIRATORY SYNCYTIAL VIRUS (R | 0 | 19 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 19 |
| 0500 | RHINOVIRUS (ALL TYPES) | 4 | 30 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 34 |
| 0600 | MYCOPLASMA PNEUMONIAE | 0 | 32 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 32 |
| 0700 | ORNITHOSIS-PSITTACOSIS | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 0899 | COXSACKIEVIRUS GROUP A TYPING | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0903 | COXSACKIEVIRUS B3 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1009 | ECHOVIRUS TYPE 9 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1011 | ECHOVIRUS TYPE 11 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1022 | ECHOVIRUS TYPE 22 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 1030 | ECHOVIRUS TYPE 30 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1100 | POLIOVIRUS NOT TYPED | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 2 |
| 1101 | POLIOVIRUS TYPE 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1103 | POLIOVIRUS TYPE 3 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 2 |
| 1104 | POLIOVIRUS - MIXED VACCINAL ST | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1200 | MUMPS VIRUS | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1300 | HERPES VIRUS GROUP - NOT TYPED | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 4 | 6 |
| 1301 | HERPES SIMPLEX VIRUS - NOT TYP | 13 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 66 | 82 |
| 1302 | EPSTEIN-BARR VIRUS (EB VIRUS) | 8 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 9 |
| 1303 | VARICELLA-ZOSTER VIRUS | 7 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9 | 16 |
| 13[illegible] | HERPES SIMPLEX TYPE 1 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 69 | 73 |
| 13[illegible] | HERPES SIMPLEX TYPE 2 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 84 | 87 |
| 1399 | HERPES VIRUS TYPING PENDING | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 5 |
| 1401 | COXIELLA BURNETII | 4 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 1502 | PICORNIA VIRUS - NOT TYPED = E | 0 | 6 | 0 | 0 | 0 | 6 | 0 | 0 | 0 | 0 | 12 |
| 1514 | MOLLUSCUM CONTAGIOSUM | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 1521 | MEASLES VIRUS | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1522 | RUBELLA VIRUS | 5 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 27 | 34 |
| 1532 | HEPATITIS B ANTIGEN | 53 | 0 | 0 | 0 | 0 | 0 | 42 | 0 | 0 | 1 | 96 |
| 1535 | HEPATITIS A ANTIBODY | 1 | 0 | 0 | 0 | 0 | 0 | 7 | 0 | 0 | 0 | 8 |
| 1541 | CHLAMYDIA A - C. TRACHOMATIS | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1556 | CMV - CYTOMEGALOVIRUS | 4 | 13 | 0 | 0 | 1 | 1 | 2 | 0 | 4 | 0 | 25 |
| 1564 | ROTAVIRUS | 0 | 0 | 0 | 0 | 0 | 110 | 0 | 0 | 0 | 0 | 110 |
| 1566 | NORWALK AGENT | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 2 |
| 1599 | ENTEROVIRUS TYPING PENDING | 0 | 5 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 1 | 10 |
| 9992 | ROSS RIVER VIRUS | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 16 | 17 |
| 9994 | SMALL VIRUS (LIKE) PARTICLE | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 |
| TOTAL | | 138 | 239 | 1 | 3 | 2 | 148 | 52 | 1 | 5 | 285 | 874 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 09/11/89 TO 22/11/89

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 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 1 | 0 | 4 |
| 0101 | ADENOVIRUS TYPE 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 4 |
| 0103 | ADENOVIRUS TYPE 3 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5 |
| 0104 | ADENOVIRUS TYPE 4 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 6 |
| 0111 | ADENOVIRUS TYPE 11 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0119 | ADENOVIRUS TYPE 19 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 0199 | ADENOVIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| 0201 | INFLUENZA A VIRUS | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0203 | INFLUENZA B VIRUS | 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 |
| 0600 | MYCOPLASMA PNEUMONIAE | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1200 | MUMPS VIRUS | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1300 | HERPES VIRUS GROUP - NOT TYPED | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1301 | HERPES SIMPLEX VIRUS - NOT TYP | 3 | 64 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 67 |
| 1302 | EPSTEIN-BARR VIRUS (EB VIRUS) | 0 | 1 | 30 | 0 | 1 | 0 | 0 | 2 | 2 | 0 | 36 |
| 1303 | VARICELLA-ZOSTER VIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 6 | 0 | 8 |
| 1306 | HERPES SIMPLEX TYPE 1 | 5 | 27 | 0 | 0 | 0 | 0 | 0 | 0 | 10 | 0 | 42 |
| 1307 | HERPES SIMPLEX TYPE 2 | 0 | 113 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 113 |
| 1401 | COXIELLA BURNETII | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| 1502 | PICORNIA VIRUS - NOT TYPED = E | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 1522 | RUBELLA VIRUS | 0 | 0 | 1 | 1 | 1 | 0 | 0 | 3 | 7 | 0 | 13 |
| 1532 | HEPATITIS B ANTIGEN | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 | 0 | 11 |
| 1541 | CHLAMYDIA A - C. TRACHOMATIS | 1 | 119 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 120 |
| 1556 | CMV - CYTOMEGALOVIRUS | 0 | 0 | 0 | 1 | 1 | 1 | 0 | 6 | 37 | 0 | 46 |
| 1564 | ROTAVIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 1599 | ENTEROVIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 2 |
| 9992 | ROSS RIVER VIRUS | 0 | 0 | 0 | 0 | 15 | 0 | 1 | 0 | 0 | 0 | 16 |
| TOTAL | | 20 | 325 | 32 | 4 | 19 | 2 | 7 | 19 | 77 | 2 | 507 |