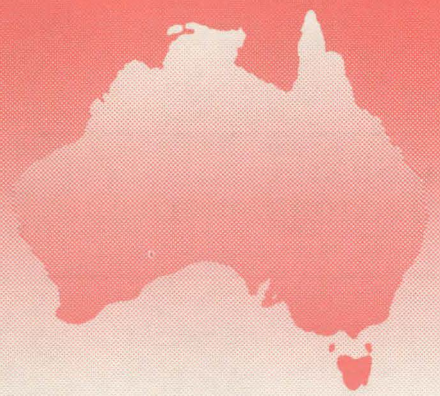


# COMMUNICABLE DISEASES INTELLIGENCE

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## CONTENTS




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**DEPARTMENT OF HEALTH, HOUSING AND COMMUNITY SERVICES**

**COMMUNICABLE DISEASES NETWORK-AUSTRALIA**
**A National Network for Communicable Diseases Surveillance**

# ALTERNATIVE HEPATITIS FOR ALTERNATIVE SEX: Recent Changes in Hepatitis A and Hepatitis B Infection Patterns in South Australia

*(A S Cameron (Senior Specialist) and P Weinstein (Registrar), Communicable Disease Control Unit, South Australian Health Commission)*

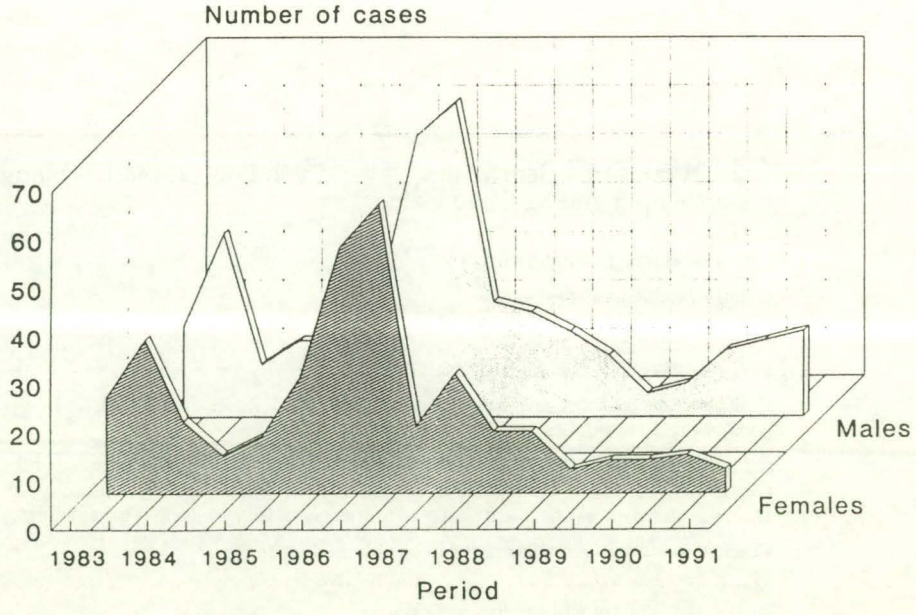
An epidemic of hepatitis A has recently been reported in the adult male populations in NSW and Victoria (*Advertiser* 28 June 1991, and *CDI*, this issue). Although the number of cases of hepatitis A notified to 20 June 1991 in South Australia is no greater than might be expected on the basis of reports for previous years (see Figure 1), the distribution of cases by sex has changed in a fashion that is consistent with the pattern in the eastern States.

The incidence of laboratory notified hepatitis A infections in South Australia has varied markedly since 1983, as is illustrated in Figure 1. The most notable increase in case numbers is that which occurred during 1985-1986, as part of a generalised phenomenon in South Australia and Western Australia. The cause of this increase is not known, but it is clear that during these years, and indeed since 1983, the sex ratio of infected males to females has remained constant at 1.16:1 in adults (age 20-66). In the last 18 months however, a marked predominance of males is apparent (sex ratio 2.62:1). These figures represents a statistically significant increase in the proportion of males infected ($z=2.48$, $p$ (one tail) = 0.0065).

A possible explanation for the increase of hepatitis A infection in the male population is an increase in oral-anal contact by way of an alternative sexual practice to AIDS-risk behaviours such as anal intercourse. It is therefore possible that the AIDS-awareness campaigns of the mid-1980's have indirectly affected the incidence of hepatitis A infection. It is therefore interesting to reflect upon the concurrent changes in the incidence of hepatitis B infection. Since 1985, based on laboratory notifications, there has been a continuous decrease in the incidence of acute, symptomatic hepatitis B infections in South Australia (see Figure 2). This decrease may also reflect a decrease in unprotected penetrative sex and needle-sharing as a result of successful AIDS education.

Figure 3 shows the age and sex distribution of people infected with hepatitis B in South Australia during 1989-90. There is a high proportion of females in the 15-19 year age group, but males form the overwhelming majority in 20-60 year olds. Factors of importance associated with infection in those males are IV drug use and unemployment.

**Figure 1. The number of notified cases of Hepatitis A in South Australia in 6 month periods since 1983, for males and females in the age group 20 - 60 years**



It is ironic that the behavioural changes associated with AIDS education may have decreased the risk of transmission for one hepatitis agent and increased the risk of contracting another. Nothing is absolute in medicine, and it is perhaps better to think in terms of "safer sex" than in terms of "safe sex".

Acknowledgements: We thank Adrian Esterman for his assistance, and Russell Waddell and Tony Stewart for useful discussion.

**Figure 2. The number of notified cases of Hepatitis B in South Australia by year since 1980**

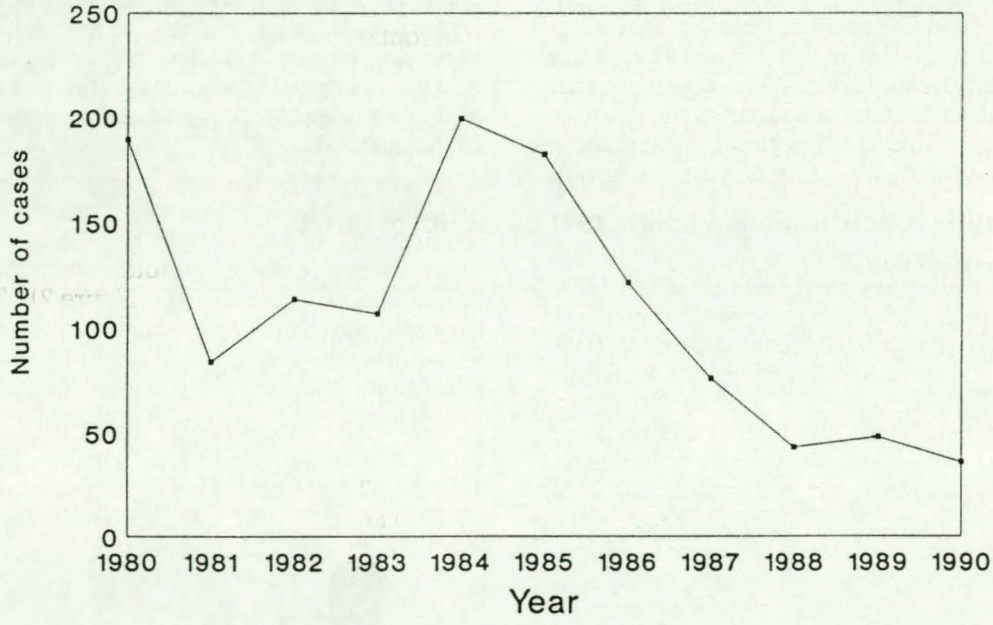
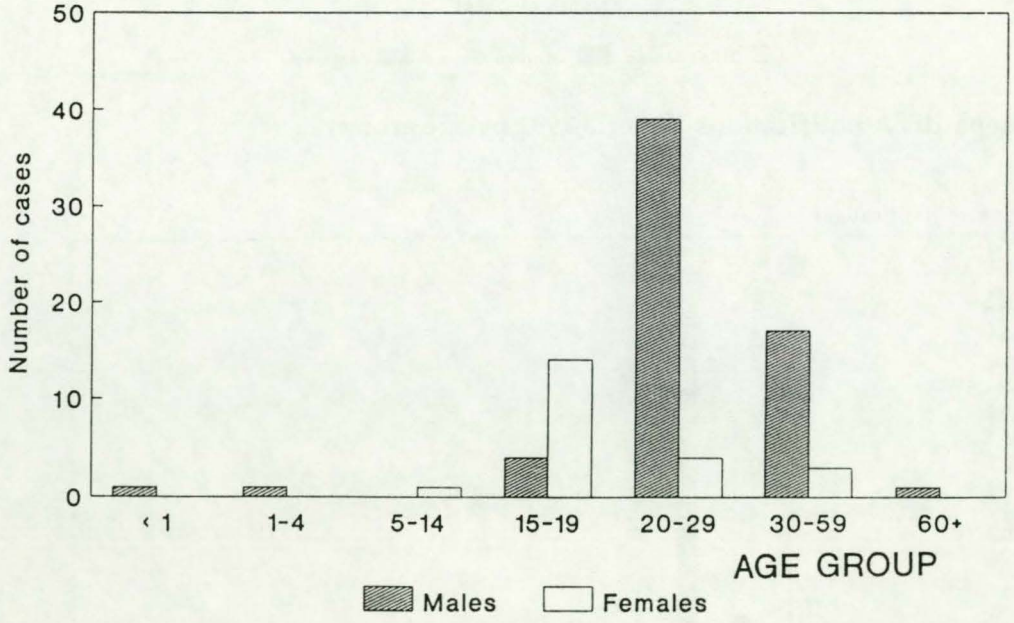



**Figure 3. The number of notified cases of Hepatitis B in South Australia by age group and sex for the years 1989-1990**



# A CLUSTER OF CASES OF HEPATITIS A IN VICTORIA

*(Dr Tony Stewart, Health Department, Victoria)*

As of 16 July 1991, there have been a total of 118 notifications of hepatitis A infection to the Health Department Victoria since 1 January 1991, with a sharp increase beginning in April (Figure 1).

Of these 118 reports, 84 (71.2%) were in males. Call back to Local Medical Officers has revealed that a significant proportion of the males (35 or 41.7%) are known to be of homosexual orientation, suggesting that sexual contact rather than food sources is the cause in some of the cases. This is supported by the peak in incidence for males in the 20 to 29 year old group (Figure 2). Of the 35 homosexual males so far identified, 15 reported onset of symptoms in May. Four men described a connection with Sydney.

We are now undertaking active case finding through contact with public and private laboratories and doctors with a large gay clientele. The Health Department is issuing advice to doctors regarding contact tracing and use of normal immunoglobulin (human) for sexual and other close contacts, and as a preventative measure in those at risk.

**Figure 1. Hepatitis A notifications Victoria 1991 by month of onset**

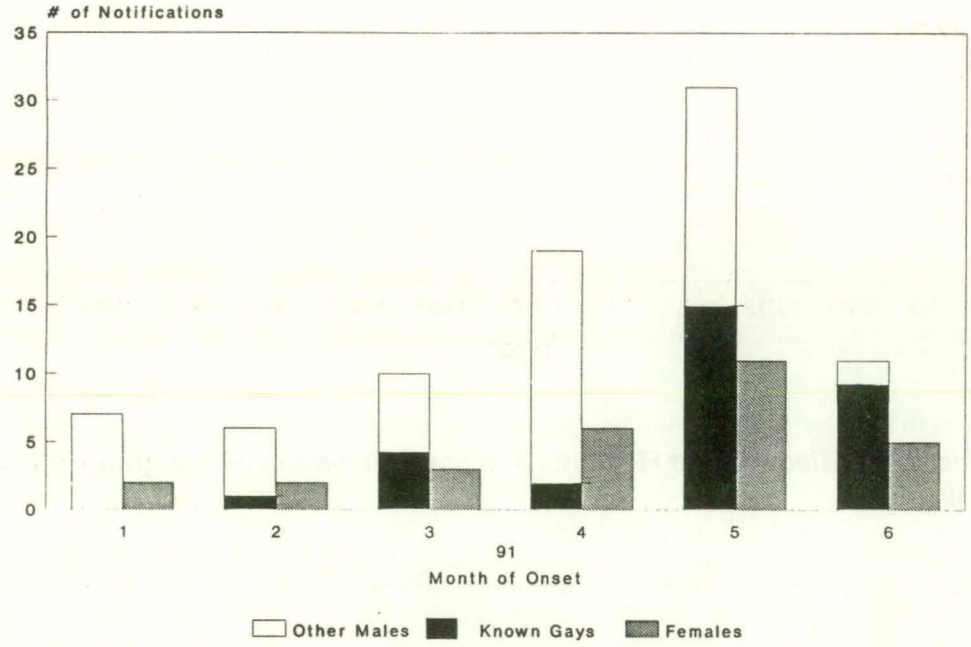
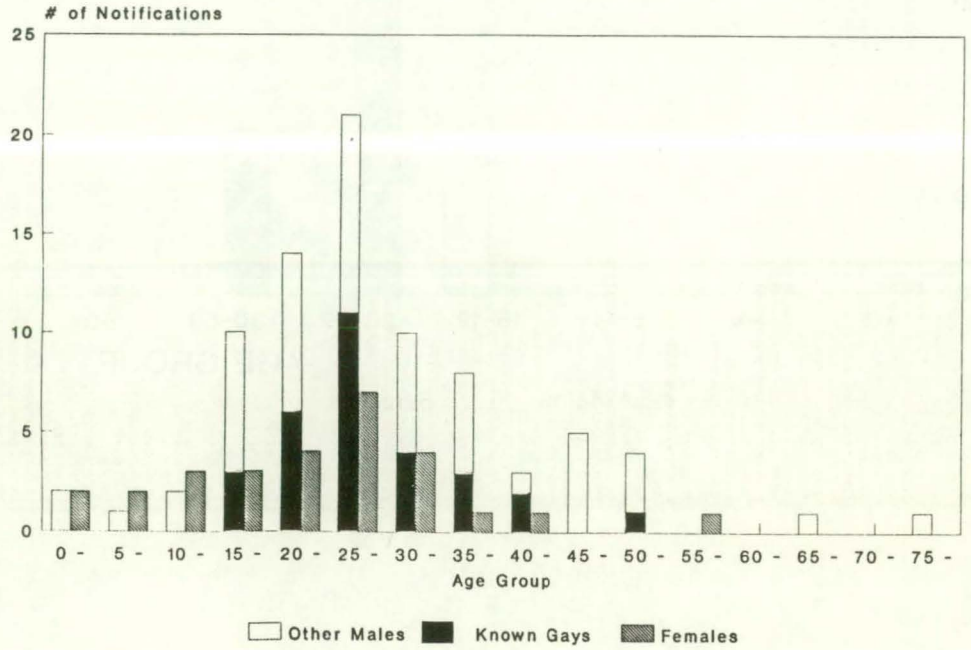



**Figure 2. Hepatitis A notifications Victoria 1991 by age group**



# A CLUSTER OF CASES OF HEPATITIS A IN WESTERN AUSTRALIA

*(Dr Robert Condon, NCEPH Epidemiology Registrar, Health Department of WA, and Mr Scott Dandridge, Health Surveyor, Shire of Harvey.)*

## Introduction:

Between January and May 1991, 136 cases of hepatitis A were notified in Western Australia. This is well in excess of the 35 notification of hepatitis A received during the same period in 1990. A review of hepatitis A surveillance in WA from 1985 to date shows that the incidence peaked in autumn in 1986, 1988 and 1991 (Figure 1). Because of the incubation period of hepatitis A, notifications in the autumn are likely to reflect infection acquired during the summer, when people are commonly engaged in outdoor activities (camping, swimming, etc.) and fly numbers are greater. The apparently cyclical phenomenon depicted in Figure 1 may just reflect the waxing and waning of herd immunity, largely from asymptomatic infections. Clear outbreaks have not been identified in recent years.

Towards the end of April 1991, two cases of hepatitis A were notified by a doctor practising in Harvey, 140 km south of Perth. Both patients lived in the same road in the nearby Town A.

The doctor was contacted and he said that he had seen two other cases of hepatitis A in residents of the Town A district earlier in the year: one lived in the next street north from the 2 latest cases, while the other was from Town B, about 5-6 km away.

Every case of a communicable disease notified from the Shire of Harvey is followed up by a Health Surveyor. Because of the clustering of three of these cases in the same town block and the presence of another case in a nearby town, it was suspected that a common source or a clear chain of person-to-person transmission would be found.

## Investigation:

The part of Town A in which the three cases live is a public housing estate. A schematic street plan of this section of town is shown in Figure 2. The houses labelled as 'B', 'C', 'D' and 'E' were all within 150m of House 'A', the home of the index case. The housing estate is regarded by health professionals as an economically depressed area with a highly mobile population who frequently visit friends and relatives in neighbouring houses and in the nearby towns.

**Figure 1. Hepatitis A in Western Australia 1985 to 1990**

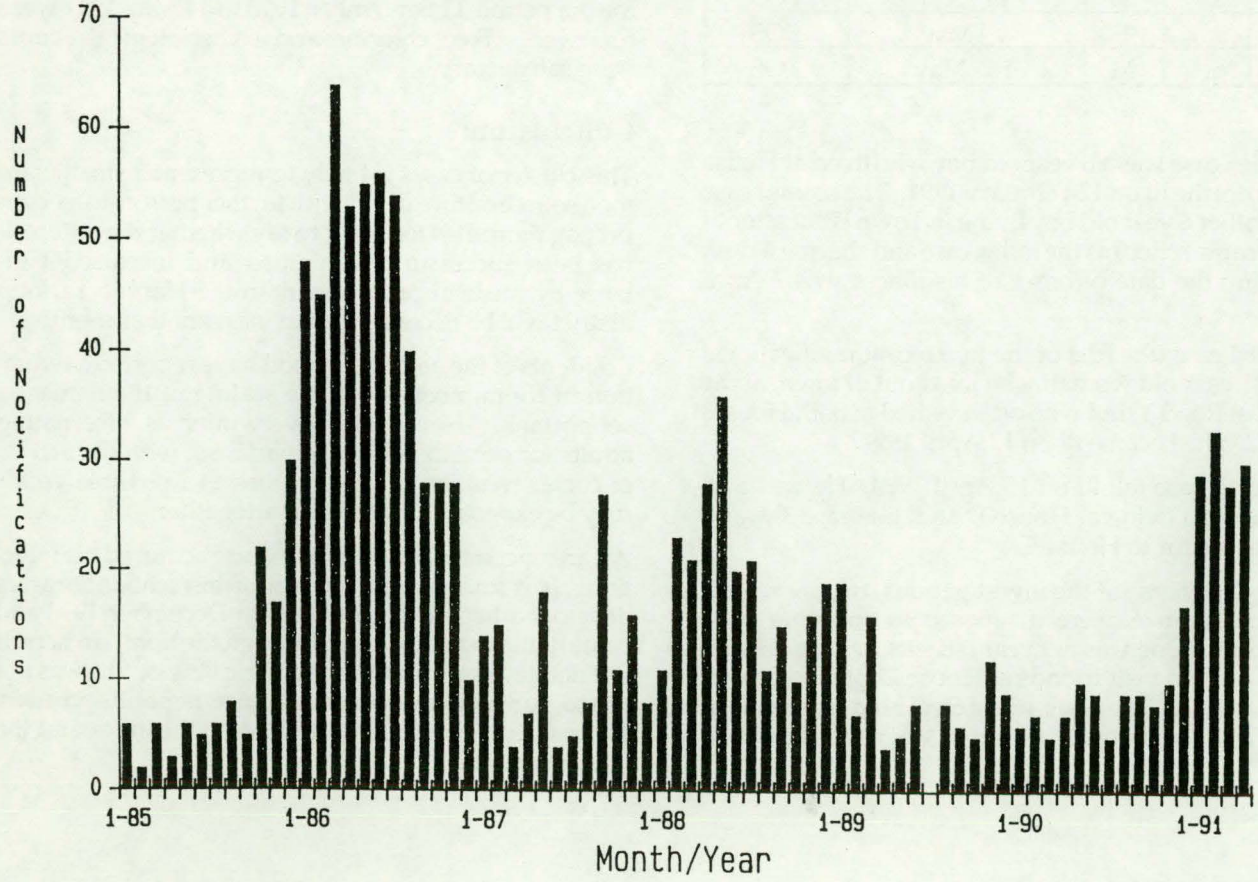
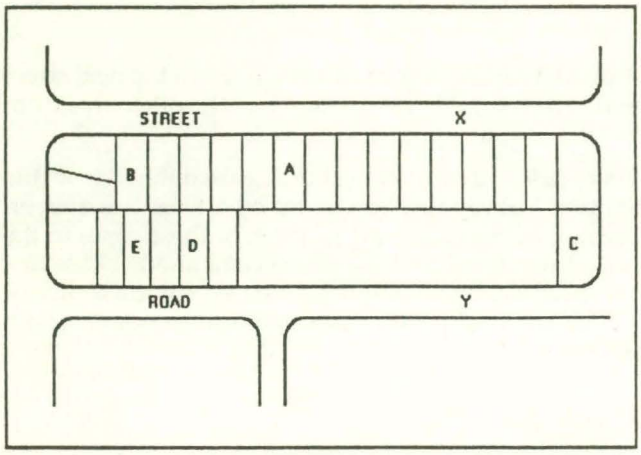


**Figure 2. Street plan of the neighbourhood where the cluster of cases of Hepatitis A occurred**




Details of the six cases are summarised in Table 1.

**Table 1. Hepatitis A cases in Town A February -May 1991**

| CASE | AGE | SEX | DATE OF ONSET OF SYMPTOMS | HAV-IgM |
|---|---|---|---|---|
| 1 | 6 | M | 12 - Feb | POS |
| 2 | 6 | M | 7 - Apr | POS |
| 3 | 31 | F | 12 - Apr | POS |
| 4 | 28 | M | 15 - Apr | POS |
| 5 | 9 | F | 1 - May | POS |
| 6 | 6 | M | 16 - May | POS |

The index case was a 6 year old boy who lived at House A and became ill on 12 February 1991. The second case was another 6 year old boy living in Town B but attending the same school as the index case and sharing a desk with him; the date of onset of his illness was 7 April 1991.

The third case (the first of the two recent notifications) was a 31 year old woman who lived out of town, at the far end of Road Y, but who often visited at both Houses B and C. She became ill on 12 April 1991.

The fourth case fell ill on 15 April 1990. He was a 28 year old man living at House D and, like case 3, was a frequent visitor to House C.

During the course of the investigations, two more residents of Town A were diagnosed as suffering from hepatitis A. One was a 9 year old girl living at House C who used to visit friends at House D; she became ill on 1 May 1991. The other was a 6 year old boy living at House E whose illness began on 16 May; he often visited friends at House A and had further contact with the index case at school.

## Public Health Action:

As cases were diagnosed, household and family contacts were offered a prophylactic dose of Human Immunoglobulin. The injections were administered by the reporting general practitioner. The infected children were excluded from school while they felt unwell, to a maximum of one week after the onset of jaundice.

A Health Surveyor visited the houses where the identified cases lived to assess domestic sewage systems and general environmental hygiene. The septic tank at the rear of the index case's house (House A) was found to have a badly cracked lid and the inspection openings on the drainage system were missing. A large number of flies were swarming in the vicinity of this house, but were also present in seasonally increased numbers throughout the neighbourhood.

The maintenance staff of the house authority were contacted and they promptly repaired the damaged septic tank and drainage system.

All other houses in Street X and Road Y were then visited by the Health Surveyor and the occupants told of the increased incidence of Hepatitis A in their neighbourhood. They were advised of possible symptoms of hepatitis and asked to visit a doctor if these symptoms appeared. Domestic sewage systems were inspected at the same time; a few minor faults were discovered and these were quickly repaired by the housing authority maintenance staff. Private residences were served with a works order.

The results of analysis of the water supply to Town A for the period 11 September 1990 to 14 May 1991 were reviewed. Free chlorine and bacteriological counts were satisfactory.

## Conclusion:

This cluster of cases is likely to represent a small common source outbreak but with further person-to-person propagation. It is too early to say whether the outbreak has been successfully contained and increased vigilance by medical practitioners in the Harvey-Yarloop district will be necessary for at least another month.

Residents of the neighbourhood have reported a reduction in fly numbers since the sealing of the damaged septic tank. House flies overwinter as hibernating adults (or occasionally as puparia) so, with the arrival of cooler weather, the role of flies as a possible vector may be expected to disappear altogether.

We are uncertain how the index case acquired his infection. It is known that the son of his school librarian developed hepatitis A in Perth in December 1990 and came home to recuperate, although the librarian herself did not develop an icteric or enteric illness. She was not tested for seroconversion against hepatitis viruses. There was no direct contact between the index and the librarian's son.

**Acknowledgement:** We would like to thank Dr Guy Buters for his vigilance and for his assistance with the passive immunisation of contacts.

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# HEPATITIS A - CDI LABORATORY REPORTING SCHEME DATA

The reports of hepatitis A received by the CDI Laboratory Reporting Scheme since the beginning of 1986 are shown in Figure 1 below. The pattern of reports has followed the patterns in South Australia and Western Australia (*CDI* , this issue), with a larger peak in 1986, and some evidence of increased activity in 1991.

The Scheme's data shows a distribution of cases between the sexes which is similar to those reported for South Australia and Victoria. Figures 2 and 3 show the reported cases, by age group and sex, for 1990 and 1991, respectively. A major difference between the two years is the larger number of cases in males, compared to females, especially in the 20-29 and 30-39 year age groups. Overall, the ratio of cases was 1.14:1 (Males to females)in 1990, and 1.90:1 (males to females) for 1991 to date. These data are consistent with the suspected spread of this disease within the homosexual community in South Australia, Victoria (CDI, this issue) and in NSW (*Sydney Morning Herald*, 28 June 1991).

Notifiable disease reports for 1991 are also consistent with these data. The distribution of cases by sex is in the ratio 1.85:1.0 (males to females) for this year, increased from 1.30:1.0 for 1990. Large differences in the numbers of cases in males and females were seen in the 20-39 age group (data not shown).

**Figure 1. Hepatitis A antibody reports 1986-1991, by month**

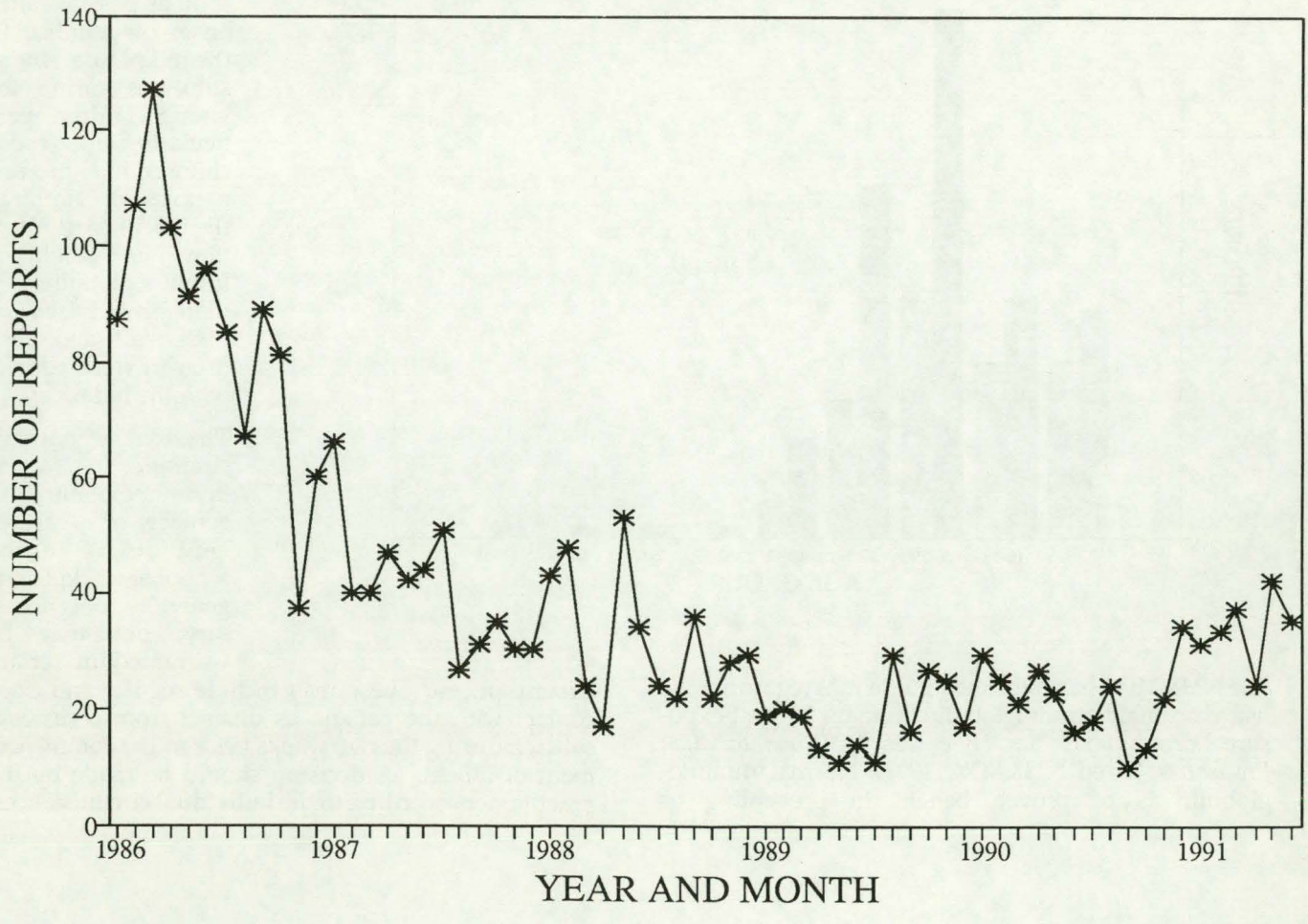


**Figure 2. Hepatitis A antibody reports, 1990 by age group and sex**

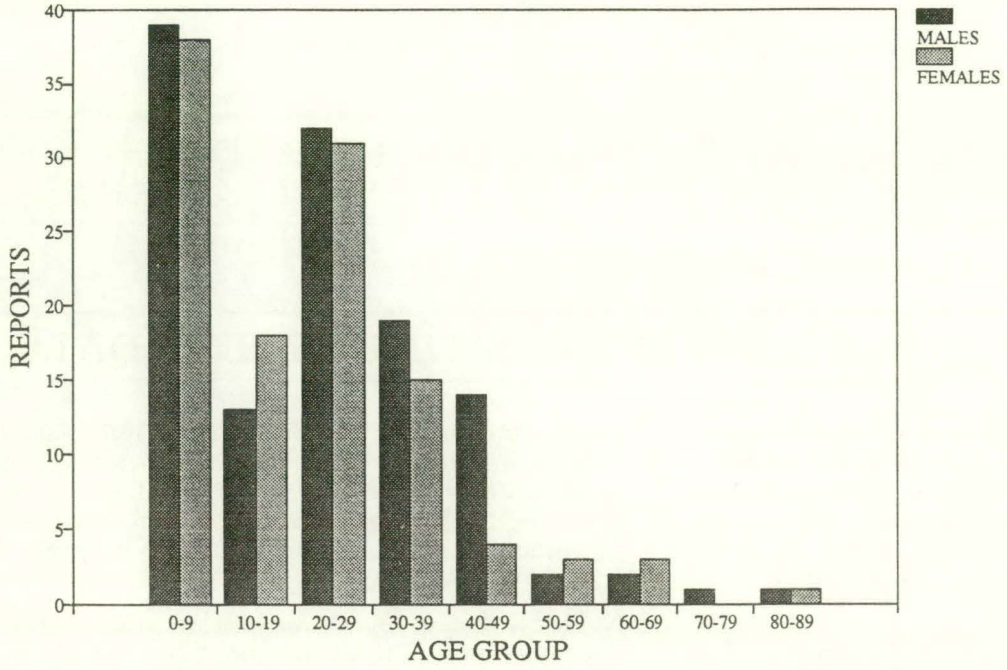
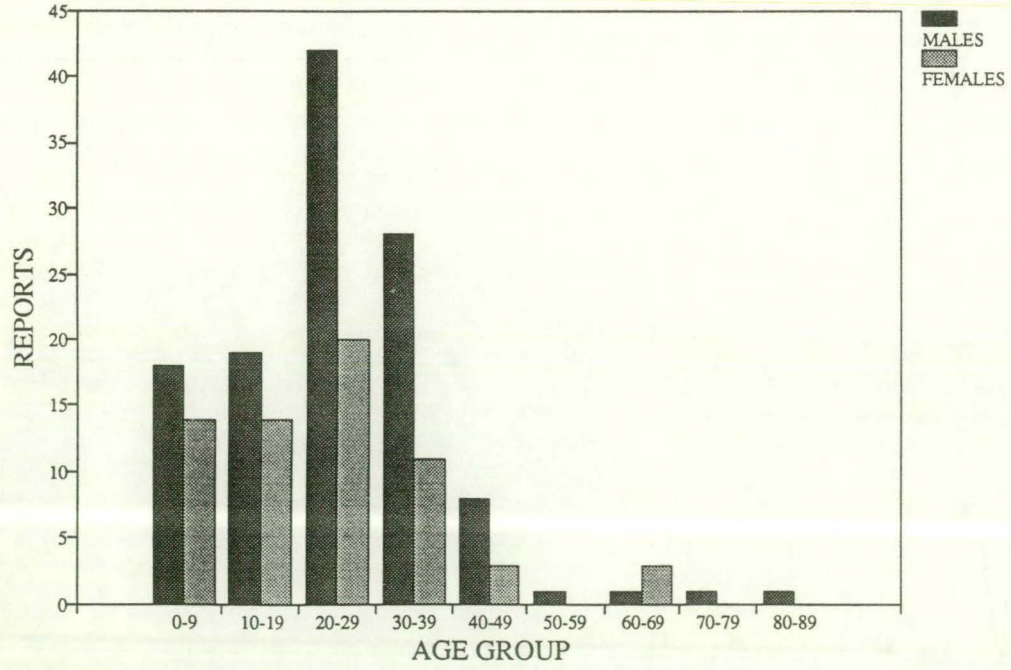



**Figure 3. Hepatitis A antibody reports, 1991 by age group and sex**




The NH&MRC has published guidelines regarding the use of normal immunoglobulin (human) for post-exposure prophylaxis for hepatitis A (*Immunisation Procedures*, 4th ed, NH&MRC, 1991). Normal Immunoglobulin is of proven benefit in preventing or modifying the severity of hepatitis A infection, providing it is given within 7-10 days after exposure. Thus by the time an index case of hepatitis A infection is clinically apparent, other individuals who were exposed simultaneously from a common source will not be protected by administration of normal immunoglobulin (human). It is, however, of value in protecting secondary contacts of an index case. In practice, the usefulness of immunoglobulin depends on identification of persons at risk within a few days of exposure.

Normal immunoglobulin (human) is recommended for household contacts of a case of hepatitis A. It should be given as soon as possible after onset of jaundice in the index case. In institutions caring for young children, particularly those children likely to soil nappies or clothing, the risk of cross infection is high. Immunoglobulin is therefore advised for both staff and children in close contact with the index case.

The routine administration of immunoglobulin to contacts of a case of hepatitits A in the school or workplace is generally not necessary, but may be warranted in certain circumstances. These may include regular and close contact with the patient, as distinct from occasional contact, during the two weeks prior to the commencement of illness, A decision should be made by the practitioner according to the individual circumstances.

## Management of Outbreaks:

Control of hepatitis A outbreaks is achieved by enforcement of hygienic measures designed to limit faecal-oral spread, e.g. appropriate care with food preparation, water sources, faecal disposal, handwashing, and by isolation of cases using enteric precautions. In addition, individuals at risk can be protected from symptomatic infection by the administration of normal immunoglobulin (human) as described above.

# SHOULD ALL TRAVELLERS BE TESTED FOR HEPATITIS A ANTIBODY LEVELS PRIOR TO TRAVEL TO ENDEMIC AREAS?

*(Dr R B Kass, Traveller's Medical and Vaccination Centre, Queen Elizabeth Hospital, Woodville, SA; Mr A Caon and Dr D F M Looke, Department of Microbiology and Infectious Diseases, Queen Elizabeth Hospital, Woodville, SA)*

## Abstract:

We investigated the hypothesis that older people were more likely to have natural antibodies to hepatitis A and would not require immunisation prior to travel to highly endemic areas. Over a 15 month period we investigated hepatitis A seropositivity in two groups of travellers:

1. Corporate travellers undertaking frequent trips to endemic areas, and

2. Individuals resident in or travelling in endemic areas for periods longer than 6 months - in this group booster doses of normal immunoglobulin may be required.

We assessed 259 individuals. All travellers with a known or probable history of hepatitis A were excluded. Positivity rates were calculated for cohorts of ten years between 20 yrs and 59 yrs and also for those under 20 and those 60 yrs and over. Seropositivity increased from 13.3% in the 20 to 29 year age group to 44.2% for those 50-59 years. In those under 20 years we found no seropositive cases but the numbers were small. In those 60 years and over the rate was 59.5%. All travellers over 70 years were positive.

Travellers from Adelaide have a low rate of seropositivity in the younger age groups. Apart from those groups above it would seem reasonable to assume all travellers under 30 years of age are non-immune and prophylaxis should be advised according to travel risk. In those over 40 years, it may be cost effective to undertake antibody testing in order to exclude those who are already immune. Frequent travellers and those requiring booster doses while overseas should still be tested.

## Introduction:

Hepatitis A occurs commonly in many parts of the world, particularly in areas where personal hygiene and sanitation are poor. Expatriates resident in these areas have been shown to have high levels of risk[1,2]. Evidence suggests that normal human immune globulin is effective in reducing the risk[3] but it is often difficult to decide who should receive prophylaxis.

As the incidence of hepatitis A falls in many western countries the percentage of cases attributed to travelling increases. In the UK approximately 15% of all reported cases are attributed to travellers (4) whereas in Scandinavia and Western Europe the figure is closer to 60%. The number of contact cases is unknown but is probably significant.

Each year more than 900,000 Australians travel to areas of risk[5] however not all travellers are at risk of infection. Many are already immune from previous exposure. A number of recent studies suggest a strong correlation between increasing age and hepatitis A positivity rates[6,7]. There is, however, considerable regional variation. Together with other factors such as frequency of travel and length of stay, an understanding of the local patterns of age versus seropositivity may help to decide whether testing for hepatitis A antibody is indicated.

## Methods:

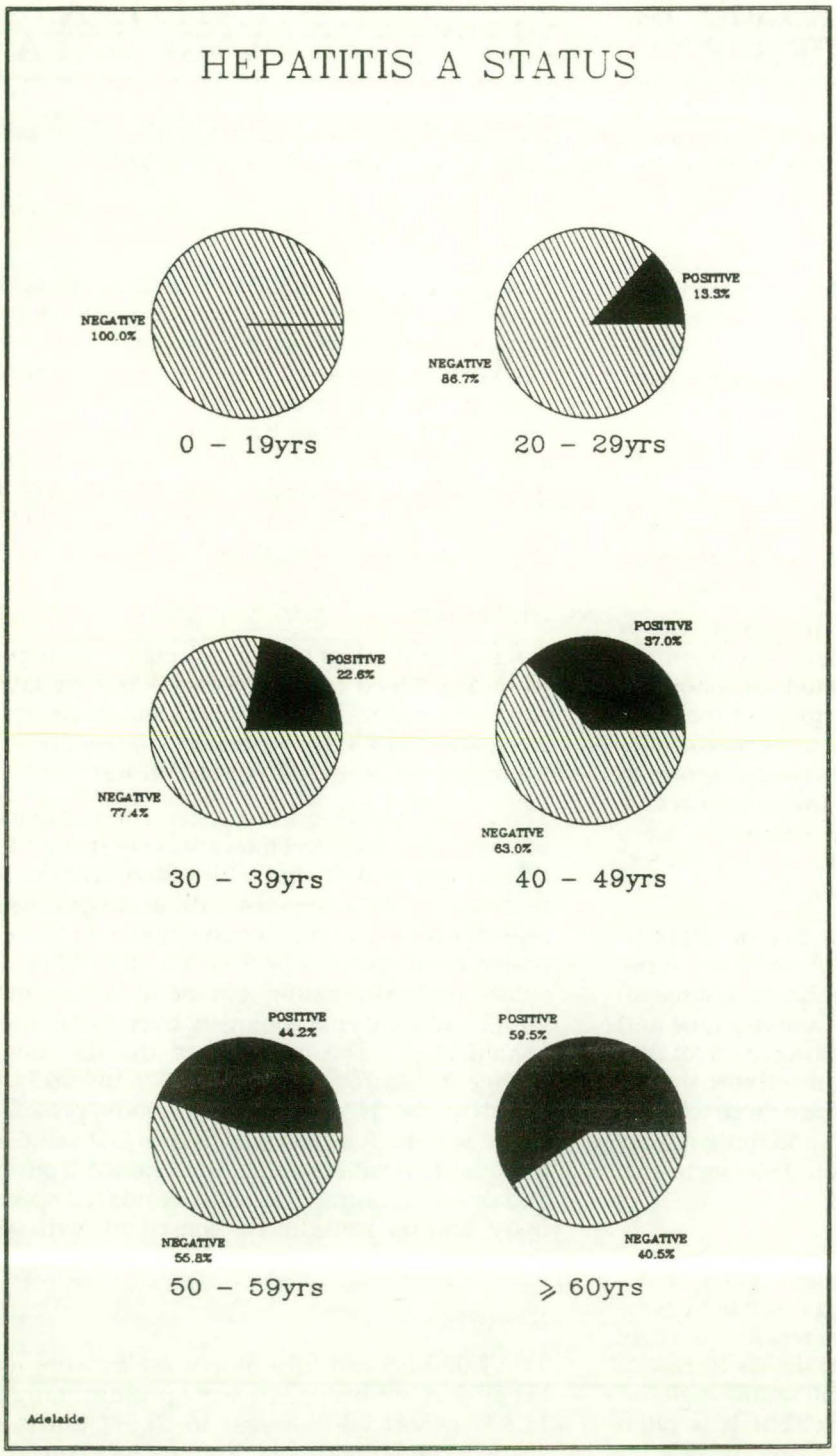
Samples of 10mL of blood, collected by venepuncture, were dispatched to the serology/virology laboratory. After separation by centrifugation, serum specimens were stored at 4°C for a week and longer at - 30°C until testing by the Sorin ETI-AB-HAVK test.

The ETI-A-HAVK test is a 2 step competitive enzyme immunoassay to detect total antibody specific for hepatitis A virus (HAV). In the first step, specific antibody in donor serum competes with anti-HAV IgG-horseradish peroxidase (enzyme conjugate) for HAV antigen coated onto micro-wells over 3 hours. After washing, substrate (incorporating tetramethyl benzidine) reacts with bound enzyme conjugate over 30 minutes in the second step. The intensity of the resulting colour change, measured as OD value, is inversely proportional to the HAV-specific antibody present in the donor serum. A serum showing an OD value less than or equal to a cut-off value, determined from negative and positive controls, is positive for HAV-specific antibody and is said to be consistent with previous exposure.

## Results:

Two hundred and fifty nine samples were tested for hepatitis A antibody; 34% were positive, ranging from 13.3% (cohort 20-29 years) to 59.5% (those over 60 years). The group under 20 years was considered too small. There was a steady increase in positivity rates for the five older groups; 13.3%, 22.6%, 37.0%, 44.2% and 59.5% (Figure 1).

**Figure 1. Hepatitis A Antibody Satus of Tested Travellers, by Age Groups**




## Discussion:

Hepatitis A vaccine is still unavailable for general use. Until it is available travel medicine departments will continue to use normal human immune globulin for prophylaxis. Rates of hepatitis A seropositivity vary from country to country. In Scotland 30% of travellers were found to be positive in the 10 to 19 years age group rising to 89% in those over 60 years (6). On the other hand it is likely that the levels of seropositivity in Scandinavian countries are low as the incidence of hepatitis A there is largely dependent on importation from abroad. Vaccination of those already seropositive is wasteful, costly and results in unnecessary inconvenience to the traveller.

In the group of travellers studied in Adelaide, the level of positivity is low in those under 30 years and it is likely that they are a representative group for Adelaide. The majority of the travellers tested were intending either to reside overseas or to travel for a long period of time. It would be reasonable to continue to test for hepatitis A antibody in these groups if only to avoid unnecessary booster doses in overseas countries. For travellers under 30 years testing would seem unnecessary. Antibody testing may be cost effective only in those over 40 years.

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7. Kudesia G, Follett E. Not all travellers need immunoglobulin for hep A. (Letter) *BMJ* 1987;**295**:118.

## CDI EDITORIAL COMMENT

The NH&MRC states that 40-70% of adults in Western countries are immune to hepatitis A. It recommends that normal immunoglobulin (human) is used for pre-exposure prophylaxis for travellers to areas where hepatitis is highly endemic, if the individual is likley to be eating and drinking from sources where hygiene is not strictly controlled. Areas currently considered to be highly endemic include many countries in Africa, Central and South America, and Asia (including India).

In non-immune individuals taking up residence in highly endemic areas, four doses of normal immunoglobulin at 3 month intervals may be used to provide cover during the first 12 months when almost all those who are exposed develop subclinical infection followed by active immunity. The following dosages are recommended:

| Person's Weight (kg) | Dosage for Short - term Prophylaxis | Dose for Long - term Prophylaxis |
|---|---|---|
| under 25 | 0.5 | 1.0 |
| 25 to 50 | 1.0 | 2.5 |
| over 50 | 2.0 | 5.0 |

Further details regarding human immunoglobulin prophylaxis for hepatitis A can be found in *Immunisation Procedures* (4th ed, NH&MRC, 1991).

# AUSTRALIAN HIV SURVEILLANCE REPORT, VOLUME 7, NUMBER 5 (30 JUNE 1991)

The National Centre in HIV Epidemiology and Clinical Research reports that as of 31 May 1991 a total of 15,186 diagnoses of HIV infection and 2,643 cases of AIDS had been reported in Australia. For the most recent period, 1 May to 31 May 1991, 29 new cases of AIDS and 50 new diagnoses of HIV infection were reported.

The following tables provide more detailed information on a State/Territory basis.

Readers should note that cumulative figures are subject to retrospective revision, which may result in apparent discrepancies between the number of new cases for the reporting month and the increment in the cumulative figure from the previous report.

**Table 1. New diagnosis of AIDS and deaths from AIDS occurring in the period 1 May to 31 May, 1991, by sex and State/Territory in whinch the diagnosis was made**

| STATE/ TERRITORY | CASES | | | DEATHS | | |
|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total |
| ACT | 1 | 0 | 1 | 0 | 0 | 0 |
| NSW | 12 | 0 | 12 | 15 | 1 | 16 |
| NT | 0 | 0 | 0 | 0 | 0 | 0 |
| QLD | 0 | 0 | 0 | 0 | 0 | 0 |
| SA | 0 | 0 | 0 | 0 | 0 | 0 |
| TAS | 0 | 0 | 0 | 0 | 0 | 0 |
| VIC | 15 | 0 | 15 | 11 | 0 | 11 |
| WA | 1 | 0 | 1 | 1 | 0 | 1 |
| **TOTAL** | **29** | **0** | **29** | **27** | **1** | **28** |

**Table 2. Cumulative cases of AIDS and deaths from AIDS by sex and State/Terrtory in which diagnoses was made, to 31 May 1991**

| STATE/ TERRITORY | CASES | | | DEATHS | | |
|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total |
| ACT | 34 | 1 | 35 | 21 | 0 | 21 |
| NSW | 1588 | 48 | 1636 | 1019 | 34 | 1053 |
| NT | 7 | 0 | 7 | 3 | 0 | 3 |
| QLD | 190 | 8 | 198 | 127 | 6 | 133 |
| SA | 89 | 3 | 92 | 42 | 1 | 43 |
| TAS | 13 | 1 | 14 | 7 | 1 | 8 |
| VIC | 527 | 12 | 539 | 307 | 6 | 313 |
| WA | 115 | 7 | 122 | 73 | 3 | 76 |
| **TOTAL** | **2563** | **80** | **2643** | **1599** | **51** | **1650** |

**Table 3. Number of new diagnoses of HIV infection in the period 1 May to 31 May 1991 and cumulative since the introduction of HIV antibody testing to 31 May 1991 by sex and**

| STATE TERRITORY | MAY 1991[1] | | | CUMULATIVE TO 31 MAY 1991 | | | |
|---|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Sex not reported | Total |
| ACT | 1 | 1 | 1 | 16 | 0 | 97 | 113 |
| NSW | - | - | - | 7697 | 394 | 2152 | 10243 |
| NT | 1 | 0 | 1 | 58 | 5 | 0 | 63 |
| QLD | 21 | 4 | 25 | 1043 | 44 | 0 | 1087 |
| SA | - | - | - | 333 | 27 | 0 | 360 |
| TAS | 1 | 0 | 1 | 51 | 3 | 0 | 54 |
| VIC | 17 | 1 | 20 | 2570 | 79 | 3 | 2652 |
| WA | 2 | 0 | 2 | 583 | 31 | 0 | 614 |
| **TOTAL** | **43** | **5** | **50** | **12351** | **583** | **2252** | **15186** |

1. Dashes indicate that counts were unavailable for period.
2. Cumulative counts to 30 April 1991.
3. Cumulative counts to 18 May 1990.
4. Total for Victoria includes 2 people whose sex was not reported.
5. Total for May includes 2 people whose sex was not reported.

# OVERSEAS BRIEFS

## CHOLERA IN AFRICA UPDATE

The World Health Organization has supplied us with the following information regarding recent cholera cases in Africa.

In **Cameroon**, there has been a total of 1393 cases with 172 deaths from 1 January to 4 July 1991. They have occurred in Logone, Chari and Mayo Danai Departments.

In **Chad**, 1,886 cases (273 deaths) were reported for the period 25-30 June, and there were a further 1049 cases and 54 deaths reported from 1 to 7 July.

**Niger** has reported 683 cases with 83 deaths for the period 8 to 30 June. Maradi Department is the only area within Niger which is currently cholera-infected.

**Togo** has reported 682 cases with 30 deaths for the period 1 January to 8 July 1991. Cases occurred in Golfe, Kloto, Kozah, Lacs, Ogou, Sotougoua, Vo and Yoto Districts.

In **Angola**, there were 4038 cases (56 deaths) in the period 1 January to 10 June 1991.

**Mozambique** has reported 924 cases with 11 deaths for the period 24 March to 27 April 1991.

All or parts of the above countries were considered to be cholera-infected by the World Health Organization on 5 July 1991. In addition, all or parts of Benin, Burundi, Cote d'Ivoire, Ghana, Guinea, Kenya, Liberia, Malawi, Mali, Mauritania, Nigeria, Sao Tome and Principe, Tanzania, Zaire and Zambia are also considered to be currently cholera-infected.

## CHOLERA IN THE AMERICAS UPDATE

Recent reports from the World Health Organization regarding cholera in the Americas are as follows.

In **Brazil**, there have been a total of 18 cases reported up to 2 July 1991. Brazil's currently infected areas are the Amazonas State and Mato Grosso State.

In **Peru**, the totals to 22 June are 223,564 cases, 86,954 hospitalisations and 2,163 deaths.

**Ecuador** has reported 24,435 cases with 388 deaths for the period 28 February to 15 June 1991. A further 6,1000 cases with 113 deaths were reported from 16 June to 6 July.

**Columbia** reported 452 cases with 11 deaths for the period 11 to 17 July.

**Mexico** has reported 27 cases (no deaths) for the period 13-28 June, 1991. The cholera epidemic has only recently spread to this country, with the areas of Valle de Tula in Hidalgo State and Sultepec (including San Miguel Totolmaloya) in Mexico State, being declared infected since 27 June 1991.

All or parts of the above countries, and Chile are considered to be cholera-infected at present.

## CHOLERA IN ASIA UPDATE

The World Health Organization has supplied the following information regarding recent cholera cases in Asia.

In **Indonesia**, there was a total of 6,202 suspected cases (55 deaths) reported for the period 1 January to 30 April 1991.

In Singapore, a single non-fatal case was reported for the period 2-8 June 1991.

All or parts of the following Asian countries are considered to be cholera-infected at present: India, Indonesia, Iraq, Nepal and Vietnam.

## YELLOW FEVER SPREADING IN ECUADOR

Two further areas of Ecuador have recently been declared to be yellow fever infected: Morona-Santiago Province and Pastaza province now join the Humayacu District of Napo Province and the Zamoro-Chinchipe Province as the country's infected areas.

In the period 1 January to 28 February, 1991, there were reports of 4 fatal cases in Morona-Santiago Province, 9 cases (4 fatal) in Napo Province, 2 cases (1 fatal) in Pastaza Province and 4 cases (no deaths) in Zamoro-Chinchipe Province.

Australia's Quarantine requirements are that any person arriving in Australia within 6 days of being in any of the yellow-fever infected areas must have been vaccinated against the disease, or quarantine restrictions may be placed on them. Parts of Angola, Cameroon, Gambia, Guinea, Mali, Nigeria, Sudan, Zaire, Bolivia, Brazil, Colombia and Peru are also considered to be currently yellow fever-infected.

## *SHIGELLA DYSENTERIAE* IN GUATEMALA

In Guatemala during March and April, 1991, there were a large number of cases of dysentery, caused by *Shigella dysenteriae* type 1 [1]. In Guatemala City, 4 children from an orphanage were diagnosed with the disease, and 1 died. Stool cultures from 2 of the children yielded *S. dysenteriae* type 1, resistant to trimethoprim-sulfamethoxazole, ampicillin, chloramphenicol and tetracycline.

On March 21, over 100 cases of bacillary dysentery were reported from Rabinal, in the department of Baja Verapaz, over 180km from Guatemala city. Stool specimens from 11 of the cases yielded *S. dysenteriae* type 1, resistant to chloramphenicol and tetracycline. By April 10, at least 540 persons had developed dysentery in Rabinal (a town of about 10,000), and two infants had died. Strains isolated from 2 patients in April were resistant to ampicillin, chloramphenicol, tetracycline and trimethoprim-sulfamethoxazole. By the end of April, it was reported that the number of new cases was declining.

The detection of *S. dysenteriae* in 2 distant areas of Guatemala indicates that it could also be present elsewhere in the country. Because of this, and the presence of multiply-resistant strains of the organism, travellers to Guatemala should take care in the selection of foods and drinks, which can become contaminated by flies or by infected food handlers who do not clean their hands thoroughly after defaecation. Water should be treated chemically or boiled, if it is not clear that its source is safe [2].

## REFERENCES:

1. Centers for Disease Control. *Shigella dysenteriae* Type 1- Guatemala 1991. *MMWR* 1991;40:421-428.
2. Benenson, AS (Ed). *Control of Communicable Diseases in Man*. American Public Health Association, Washington. 1990.

# COMMUNICABLE DISEASES SURVEILLANCE

## CDI LABORATORY REPORTING SCHEMES

A total of 1303 reports were processed for the latest reporting period (3 July-16 July 1991) in the Virues, Chlamydias, Coxiellas Richettsias and Mycoplasmas Reporting Scheme.

A further 109 reports of **rotavirus** were made, bringing the total for 1991 to 661. There have been a lot more reports of this virus this year, compared to previous years (see Figure below). This indicates that there is either increased or earlier rotavirus activity than usual, this year.

There were 110 reports of **hepatitis C**, bringing the total for the year to 291. One report was from a 5 year old boy who was a haemophiliac. The boy's ALT levels were elevated in comparison to when they were investigated in 1988, indicating that the infection had been acquired since then. The source of the infection could have been Factor VIII Concentrate, as this product has only been treated to inactive Hepatitis C virus since 1990.

There were 12 cases of **rubella** reported. In one case, the virus was isolated from the urine of a baby with the clinical features of congenital rubella infection.

Exposure details were provided for two of the 5 cases of **Q fever** reported. One patient was a meatworker and the other was a farmer.

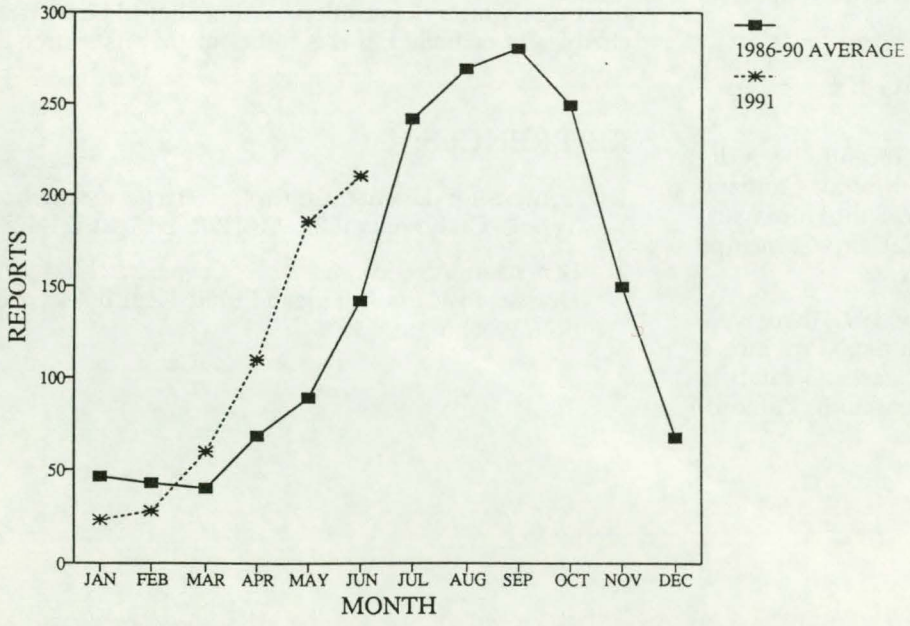
A case of Guillain-Barre Syndrome was reported. The patient was a 21 year old female, who had specific IgM to **cytomegalovirus**.

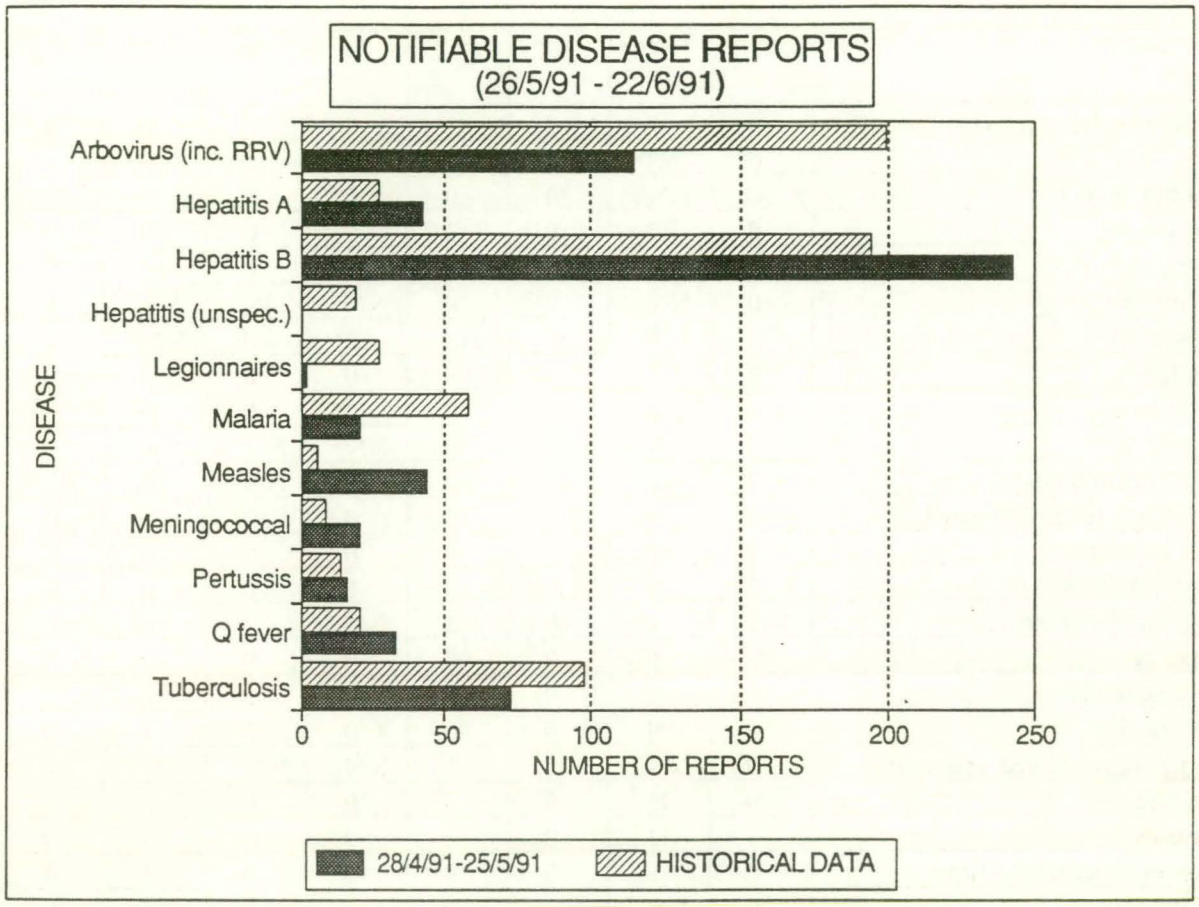
There has been an increased activity of **echovirus type 17** meningitis occurring in the Sydney area since March this year. Echovirus type 17 has been isolated from the cerebrospinal fluid of 9 patients and from faecal samples of another 2 patients with CNS symptoms. All except 2 patients (43 years and 18 years) were children under 8 years of age including 4 infants less than 2 months of age. Virus isolations have been reported by the Royal Alexandra Hospital for Children, Prince Henry Hospital and Westmead Hospital.

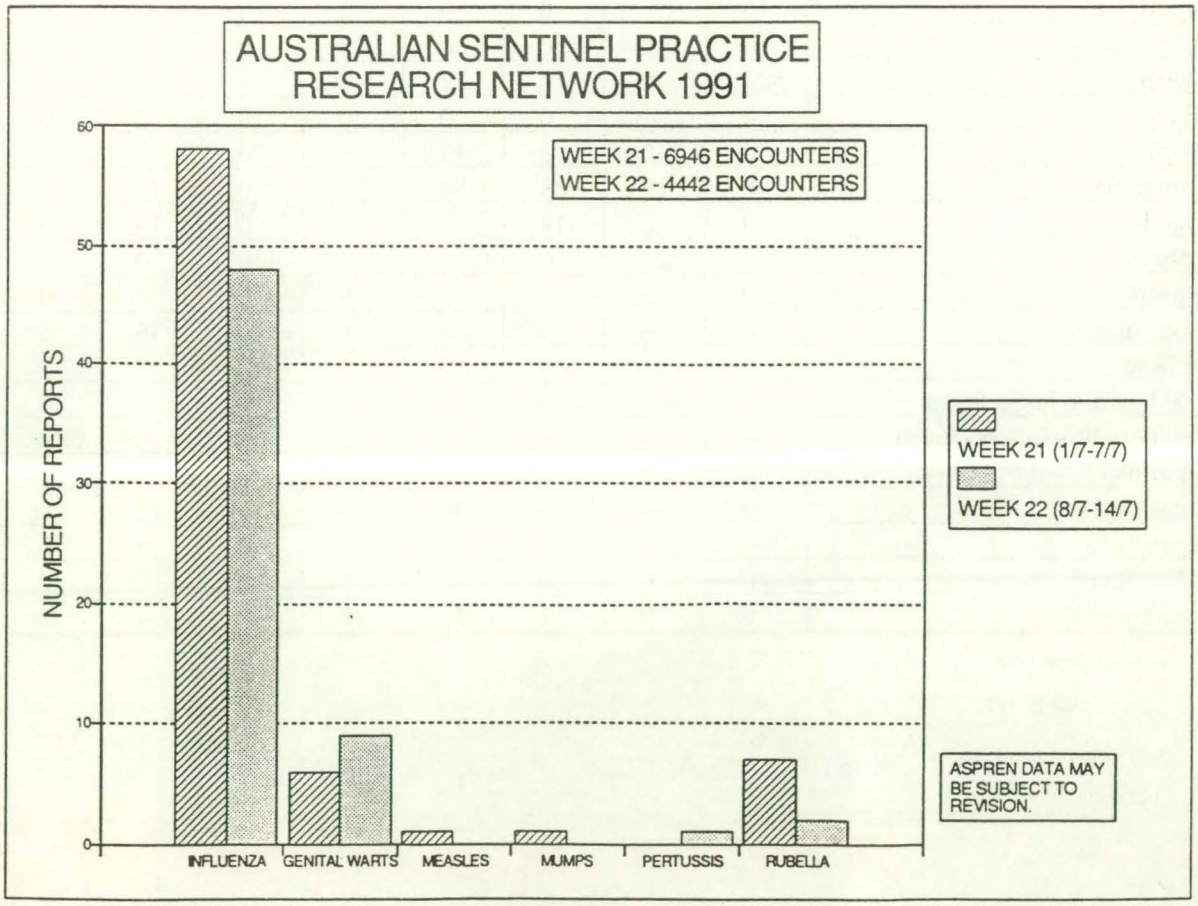
Other reports of echovirus type 17 this year have been from a biopsy sample from an infant with a distended bowel (Westmead Hospital) and from a nasal and a faecal sample from South Australia, making a total of 14 reports so far this year. Only six reports of this virus were received from 1985 to 1990, although 49, 20 and 19 reports were received in 1982, 1983 and 1984 respectively.

A further 3 cases of **meningococcal meningitis** have been reported from Western Australia, which reported 6 cases last fortnight. The first patient was from Katanning, a town in the rural soutwest of the State where there was a major outbreak of group C meningococcal disease in 1990. The other two cases were from the north of the State; one from Port Hedland and the other from Karratha.

**Figure 1. Rota virus reports 1986-90 average and 1991**




NOTIFIABLE DISEASE REPORTS
(26/5/91 - 22/6/91)
Arbovirus (inc. RRV)
Hepatitis A
Hepatitis B
Hepatitis (unspec.)
Legionnaires
Malaria
Measles
Meningococcal
Pertussis
Q fever
Tuberculosis
DISEASE
0
50
100
150
200
250
NUMBER OF REPORTS
28/4/91-25/5/91
HISTORICAL DATA


AUSTRALIAN SENTINEL PRACTICE
RESEARCH NETWORK 1991
WEEK 21 - 6946 ENCOUNTERS
WEEK 22 - 4442 ENCOUNTERS
NUMBER OF REPORTS
60
50
40
30
20
10
0
INFLUENZA
GENITAL WARTS
MEASLES
MUMPS
PERTUSSIS
RUBELLA
WEEK 21 (1/7-7/7)
WEEK 22 (8/7-14/7)
ASPREN DATA MAY BE SUBJECT TO REVISION.

**National Notifiable Disease Reports 26/5/91-22/6/91**

| DISEASES | ACT | NSW* | NT | QLD | SA | TAS | VIC | WA | TOTAL |
|---|---|---|---|---|---|---|---|---|---|
| Arbovirus Infections (unspecified) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| Ross River Virus | 0 | 23 | 4 | 81 | 0 | 0 | 6 | 0 | 114 |
| Dengue fever | 0 | NN | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Brucellosis | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 3 |
| Campylobacter | 0 | 0 | 25 | 176 | 148 | 0 | 133 | 0 | 482 |
| Chancroid | 0 | NN | 0 | 0 | NN | NN | NN | 1 | 1 |
| Chlamydia | 0 | 0 | 32 | 148 | 82** | 0 | NN | 0 | 262 |
| Cholera | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Diphtheria | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 2 |
| Donovanosis | 0 | NN | 1 | 4 | NN | NN | NN | 0 | 5 |
| Gonoccocal diseases | 0 | 37 | 28 | 32 | 16** | NN | 0 | 28 | 141 |
| Haemophilus influenzae b | 0 | 3 | 0 | 15 | 0 | 0 | 24 | 0 | 42 |
| HIV infections | 1 | 17 | 0 | 0 | 0 | 0 | 0 | 0 | 18 |
| Hydatid disease | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Legionnaires disease | NN | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 2 |
| Leprosy | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Leptospirosis | 0 | 3 | 0 | 1 | 0 | 0 | 2 | 0 | 6 |
| Listeriosis | 0 | NN | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| Lymphogranuloma venereum | 0 | NN | NN | 0 | NN | NN | NN | NN | 0 |
| Malaria | 0 | 13 | 0 | 5 | 0 | 0 | 2 | 0 | 20 |
| Measles | 1 | 11 | 0 | 5 | 9 | 0 | 18 | 0 | 44 |
| Meningococcal infections | 0 | 9 | 0 | 0 | 0 | 0 | 11 | 0 | 20 |
| Ornithosis | 0 | NN | 0 | 0 | 0 | 0 | 5 | 0 | 5 |
| Pertussis | 0 | 4 | 0 | 8 | 1 | 0 | 3 | 0 | 16 |
| Plague | 0 | NN | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Poliomyelitis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Q fever | NN | 10 | 0 | 22 | 0 | 0 | 1 | 0 | 33 |
| Rabies | 0 | NN | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella | 0 | 0 | 0 | 12 | 6 | 0 | 7 | 0 | 25 |
| Salmonella | 0 | 99 | 38 | 89 | 38 | 0 | 46 | 0 | 272 |
| Shigella | 0 | 0 | 19 | 0 | 3 | 0 | 10 | 0 | 32 |
| Syphilis | 0 | 28 | 10 | 29 | 7** | 0 | 0 | 13 | 77 |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Tuberculosis | 0 | 48 | 1 | 6 | 4 | 0 | 15 | 0 | 73 |
| Typhoid | 0 | 3*** | 0 | 0 | 0 | 0 | 2 | 0 | 5 |
| Viral haemorrhagic fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Viral hepatitis (unspecified) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | NN | 0 |
| Hepatitis A | 0 | 3 | 0 | 12 | 2 | 0 | 25 | 0 | 42 |
| Hepatitis B | 0 | 34 | 0 | 131 | 0 | 0 | 78 | 0 | 243 |
| Hepatitis C | 0 | 1 | 0 | 69 | 0 | 0 | 18 | 0 | 88 |
| Yellow fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Yersiniosis | 0 | 0 | 2 | 21 | 17 | 0 | 1 | 0 | 41 |

* data for June 1991

** data to June 27 1991

*** typhoid and paratyphoid

NN not notifiable

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 03/07/91 TO 16/07/91

CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 065 - STATE HEALTH LABORATORY SERVICES, PERTH (WA)
CODE 066 - PRINCESS MARGARET HOSPITAL, PERTH (WA)
CODE 110 - INSTITUTE OF MEDICAL & VETERINARY SCIENCE, ADELAIDE (SA)
CODE 111 - ROYAL CHIDLDRENS HOSPITAL, MELBOURNE (VIC)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)
CODE 270 - TAMWOTH LAB, NEW ENGLAND PATHOLOGY (NSW)
CODE 400 - DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON (QLD)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

| | 019 | 065 | 066 | 110 | 111 | 112 | 114 | 115 | 116 | 270 | 400 | RHH | TPL | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 0100 ADENOVIRUS NOT TYPED | 0 | 3 | 2 | 6 | 10 | 7 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 30 |
| 0101 ADENOVIRUS TYPE 1 | 3 | 0 | 0 | 2 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9 |
| 0102 ADENOVIRUS TYPE 2 | 3 | 0 | 0 | 1 | 0 | 5 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 10 |
| 0103 ADENOVIRUS TYPE 3 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 6 |
| 0104 ADENOVIRUS TYPE 4 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0105 ADENOVIRUS TYPE 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0108 ADENOVIRUS TYPE 8 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0129 ADENOVIRUS TYPE 29 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0135 ADENOVIRUS TYPE 35 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0199 ADENOVIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5 |
| 0201 INFLUENZA A VIRUS | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 |
| 0202 INFLUENZA A VIRUS SUBTYPE H3N2 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0203 INFLUENZA B VIRUS | 0 | 0 | 0 | 4 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 6 |
| 0301 PARAINFLUENZA VIRUS TYPE 1 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0302 PARAINFLUENZA VIRUS TYPE 2 | 0 | 0 | 0 | 8 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 10 |
| 0303 PARAINFLUENZA VIRUS TYPE 3 | 6 | 0 | 1 | 12 | 1 | 5 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 28 |
| 0399 PARAINFLUENZA VIRUS TYPING PEN | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (R | 20 | 0 | 6 | 10 | 48 | 35 | 55 | 0 | 19 | 6 | 0 | 1 | 0 | 200 |
| 0500 RHINOVIRUS (ALL TYPES) | 9 | 0 | 0 | 1 | 16 | 6 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 32 |
| 0600 MYCOPLASMA PNEUMONIAE | 3 | 1 | 0 | 0 | 5 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12 |
| 0650 MYCOPLASMA HOMINIS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0700 ORNITHOSIS-PSITTACOSIS | 7 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 9 |
| 0809 COXSACKIEVIRUS A9 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 0902 COXSACKIEVIRUS B2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2 |
| 0903 COXSACKIEVIRUS B3 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0904 COXSACKIEVIRUS B4 | 1 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1001 ECHOVIRUS TYPE 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 1009 ECHOVIRUS TYPE 9 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1017 ECHOVIRUS TYPE 17 | 0 | 0 | 0 | 0 | 0 | 7 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| 1022 ECHOVIRUS TYPE 22 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1025 ECHOVIRUS TYPE 25 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1101 POLIOVIRUS TYPE 1 | 2 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1102 POLIOVIRUS TYPE 2 | 1 | 0 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5 |
| 1103 POLIOVIRUS TYPE 3 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1300 HERPES VIRUS GROUP - NOT TYPED | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1301 HERPES SIMPLEX VIRUS - NOT TYP | 0 | 0 | 1 | 1 | 0 | 16 | 3 | 0 | 13 | 0 | 0 | 0 | 0 | 34 |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS) | 3 | 9 | 0 | 16 | 0 | 29 | 1 | 0 | 0 | 0 | 11 | 1 | 0 | 70 |
| 1303 VARICELLA-ZOSTER VIRUS | 3 | 8 | 0 | 2 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 17 |
| 1306 HERPES SIMPLEX TYPE 1 | 15 | 28 | 1 | 21 | 1 | 6 | 0 | 0 | 1 | 1 | 0 | 6 | 0 | 80 |
| 1307 HERPES SIMPLEX TYPE 2 | 18 | 51 | 0 | 18 | 0 | 24 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 114 |
| 1399 HERPES VIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1401 COXIELLA BURNETII | 0 | 0 | 0 | 1 | 0 | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 5 |
| 1502 PICORNIA VIRUS - NOT TYPED = E | 0 | 9 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 |
| 1514 MOLLUSCUM CONTAGIOSUM | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 3 |
| 1521 MEASLES VIRUS | 1 | 0 | 0 | 8 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9 |
| 1522 RUBELLA VIRUS | 1 | 1 | 0 | 2 | 0 | 4 | 0 | 0 | 2 | 0 | 2 | 0 | 0 | 12 |
| 1532 HEPATITIS B ANTIGEN | 6 | 19 | 0 | 3 | 0 | 35 | 0 | 0 | 11 | 0 | 2 | 2 | 0 | 78 |
| 1535 HEPATITIS A ANTIBODY | 1 | 6 | 0 | 2 | 0 | 4 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 15 |
| 1536 HEPATITIS C VIRUS | 53 | 16 | 0 | 38 | 0 | 0 | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 110 |
| 1541 CHLAMYDIA TRACHOMATIS - UNSPEC | 0 | 28 | 0 | 16 | 1 | 21 | 2 | 16 | 9 | 4 | 0 | 9 | 6 | 112 |
| 1542 CHLAMYDIA TRACHOMATIS - A-K | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 | 0 | 0 | 8 |
| 1556 CMV - CYTOMEGALOVIRUS | 21 | 1 | 1 | 5 | 4 | 16 | 2 | 1 | 0 | 0 | 4 | 1 | 0 | 56 |
| 1563 CORONAVIRUS | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1564 ROTAVIRUS | 6 | 2 | 6 | 13 | 33 | 13 | 6 | 0 | 0 | 6 | 15 | 2 | 7 | 109 |
| 1565 CALICI VIRUS | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1566 NORWALK AGENT | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1571 ENTEROVIRUS TYPE 71 (BCR) | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1599 ENTEROVIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 5 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| 9903 NON-A, NON-B HEPATITIS (OTHER) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 14 | 0 | 14 |
| 9992 ROSS RIVER VIRUS | 0 | 0 | 0 | 1 | 0 | 4 | 0 | 0 | 0 | 0 | 9 | 0 | 0 | 14 |
| 9993 ASTROVIRUS | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 9995 DENGUE NOT TYPED | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| TOTAL | 195 | 184 | 19 | 192 | 132 | 278 | 80 | 17 | 68 | 18 | 57 | 41 | 14 | 1295 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 03/07/91 TO 16/07/91

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

| | | NSW | VIC | QLD | WA | SA | TAS | ACT | TOTAL |
|---|---|---|---|---|---|---|---|---|---|
| 0100 | ADENOVIRUS NOT TYPED | 7 | 10 | 0 | 5 | 6 | 1 | 1 | 30 |
| 0101 | ADENOVIRUS TYPE 1 | 4 | 3 | 0 | 0 | 2 | 0 | 0 | 9 |
| 0102 | ADENOVIRUS TYPE 2 | 5 | 3 | 0 | 0 | 1 | 0 | 1 | 10 |
| 0103 | ADENOVIRUS TYPE 3 | 0 | 5 | 0 | 0 | 0 | 1 | 0 | 6 |
| 0104 | ADENOVIRUS TYPE 4 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0105 | ADENOVIRUS TYPE 5 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0108 | ADENOVIRUS TYPE 8 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0129 | ADENOVIRUS TYPE 29 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0135 | ADENOVIRUS TYPE 35 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0199 | ADENOVIRUS TYPING PENDING | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 5 |
| 0201 | INFLUENZA A VIRUS | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 2 |
| 0202 | INFLUENZA A VIRUS SUBTYPE H3N2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0203 | INFLUENZA B VIRUS | 1 | 0 | 0 | 0 | 4 | 0 | 1 | 6 |
| 0301 | PARAINFLUENZA VIRUS TYPE 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 2 |
| 0302 | PARAINFLUENZA VIRUS TYPE 2 | 1 | 0 | 0 | 0 | 8 | 0 | 1 | 10 |
| 0303 | PARAINFLUENZA VIRUS TYPE 3 | 7 | 7 | 0 | 1 | 12 | 0 | 1 | 28 |
| 0399 | PARAINFLUENZA VIRUS TYPING PEN | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0400 | RESPIRATORY SYNCYTIAL VIRUS (R | 96 | 68 | 0 | 6 | 10 | 1 | 19 | 200 |
| 0500 | RHINOVIRUS (ALL TYPES) | 6 | 25 | 0 | 0 | 1 | 0 | 0 | 32 |
| 0600 | MYCOPLASMA PNEUMONIAE | 3 | 8 | 0 | 1 | 0 | 0 | 0 | 12 |
| 0650 | MYCOPLASMA HOMINIS | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 0700 | ORNITHOSIS-PSITTACOSIS | 0 | 7 | 0 | 0 | 1 | 0 | 1 | 9 |
| 0809 | COXSACKIEVIRUS A9 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 3 |
| 0902 | COXSACKIEVIRUS B2 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 2 |
| 0903 | COXSACKIEVIRUS B3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0904 | COXSACKIEVIRUS B4 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1001 | ECHOVIRUS TYPE 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 1009 | ECHOVIRUS TYPE 9 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1017 | ECHOVIRUS TYPE 17 | 8 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| 1022 | ECHOVIRUS TYPE 22 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1025 | ECHOVIRUS TYPE 25 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1101 | POLIOVIRUS TYPE 1 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1102 | POLIOVIRUS TYPE 2 | 4 | 1 | 0 | 0 | 0 | 0 | 0 | 5 |
| 1103 | POLIOVIRUS TYPE 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1300 | HERPES VIRUS GROUP - NOT TYPED | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 2 |
| 1301 | HERPES SIMPLEX VIRUS - NOT TYP | 19 | 0 | 0 | 1 | 1 | 0 | 13 | 34 |
| 1302 | EPSTEIN-BARR VIRUS (EB VIRUS) | 30 | 3 | 11 | 9 | 16 | 1 | 0 | 70 |
| 1303 | VARICELLA-ZOSTER VIRUS | 3 | 3 | 0 | 8 | 2 | 1 | 0 | 17 |
| 1306 | HERPES SIMPLEX TYPE 1 | 7 | 16 | 0 | 29 | 21 | 6 | 1 | 80 |
| 1307 | HERPES SIMPLEX TYPE 2 | 25 | 18 | 0 | 51 | 18 | 0 | 2 | 114 |
| 1399 | HERPES VIRUS TYPING PENDING | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1401 | COXIELLA BURNETII | 2 | 0 | 2 | 0 | 1 | 0 | 0 | 5 |
| 1502 | PICORNIA VIRUS - NOT TYPED = E | 2 | 0 | 0 | 9 | 0 | 0 | 0 | 11 |
| 1514 | MOLLUSCUM CONTAGIOSUM | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 |
| 1521 | MEASLES VIRUS | 0 | 1 | 0 | 0 | 8 | 0 | 0 | 9 |
| 1522 | RUBELLA VIRUS | 4 | 1 | 2 | 1 | 2 | 0 | 2 | 12 |
| 1532 | HEPATITIS B ANTIGEN | 35 | 6 | 2 | 19 | 3 | 2 | 11 | 78 |
| 1535 | HEPATITIS A ANTIBODY | 4 | 1 | 0 | 6 | 2 | 0 | 2 | 15 |
| 1536 | HEPATITIS C VIRUS | 1 | 53 | 1 | 16 | 38 | 0 | 1 | 110 |
| 1541 | CHLAMYDIA TRACHOMATIS - UNSPEC | 27 | 1 | 22 | 28 | 16 | 9 | 9 | 112 |
| 1542 | CHLAMYDIA TRACHOMATIS - A-K | 0 | 0 | 8 | 0 | 0 | 0 | 0 | 8 |
| 1556 | CMV - CYTOMEGALOVIRUS | 18 | 25 | 5 | 2 | 5 | 1 | 0 | 56 |
| 1563 | CORONAVIRUS | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1564 | ROTAVIRUS | 25 | 39 | 22 | 8 | 13 | 2 | 0 | 109 |
| 1565 | CALICI VIRUS | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1566 | NORWALK AGENT | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1571 | ENTEROVIRUS TYPE 71 (BCR) | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1599 | ENTEROVIRUS TYPING PENDING | 3 | 5 | 0 | 0 | 0 | 0 | 0 | 8 |
| 9903 | NON-A, NON-B HEPATITIS (OTHER) | 0 | 0 | 0 | 0 | 0 | 14 | 0 | 14 |
| 9992 | ROSS RIVER VIRUS | 4 | 0 | 9 | 0 | 1 | 0 | 0 | 14 |
| 9993 | ASTROVIRUS | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 9995 | DENGUE NOT TYPED | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| TOTAL | | 376 | 327 | 88 | 203 | 192 | 41 | 68 | 1295 |

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

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 03/07/91 TO 16/07/91

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

| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 0100 | ADENOVIRUS NOT TYPED | 0 | 2 | 0 | 0 | 0 | 0 | 26 | 0 | 0 | 0 | 0 | 28 |
| 0101 | ADENOVIRUS TYPE 1 | 2 | 4 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 7 |
| 0102 | ADENOVIRUS TYPE 2 | 1 | 5 | 0 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 10 |
| 0103 | ADENOVIRUS TYPE 3 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 0105 | ADENOVIRUS TYPE 5 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0129 | ADENOVIRUS TYPE 29 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 0130 | ADENOVIRUS TYPE 30 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| 0199 | ADENOVIRUS TYPING PENDING | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 4 |
| 0201 | INFLUENZA A VIRUS | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0202 | INFLUENZA A VIRUS SUBTYPE H3N2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0203 | INFLUENZA B VIRUS | 1 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 0301 | PARAINFLUENZA VIRUS TYPE 1 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0302 | PARAINFLUENZA VIRUS TYPE 2 | 0 | 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10 |
| 0303 | PARAINFLUENZA VIRUS TYPE 3 | 0 | 20 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 20 |
| 0399 | PARAINFLUENZA VIRUS TYPING PEN | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0400 | RESPIRATORY SYNCYTIAL VIRUS (R | 2 | 188 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 191 |
| 0500 | RHINOVIRUS (ALL TYPES) | 2 | 28 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 32 |
| 0600 | MYCOPLASMA PNEUMONIAE | 1 | 8 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9 |
| 0700 | ORNITHOSIS-PSITTACOSIS | 3 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| 0809 | COXSACKIEVIRUS A9 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0902 | COXSACKIEVIRUS B2 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0903 | COXSACKIEVIRUS B3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0904 | COXSACKIEVIRUS B4 | 2 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1001 | ECHOVIRUS TYPE 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1009 | ECHOVIRUS TYPE 9 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1017 | ECHOVIRUS TYPE 17 | 4 | 0 | 0 | 3 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 8 |
| 1022 | ECHOVIRUS TYPE 22 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 |
| 1025 | ECHOVIRUS TYPE 25 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1101 | POLIOVIRUS TYPE 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 3 |
| 1102 | POLIOVIRUS TYPE 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 2 |
| 1103 | POLIOVIRUS TYPE 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1300 | HERPES VIRUS GROUP - NOT TYPED | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 |
| 1301 | HERPES SIMPLEX VIRUS - NOT TYP | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9 | 12 |
| 1302 | EPSTEIN-BARR VIRUS (EB VIRUS) | 12 | 10 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 3 | 28 |
| 1303 | VARICELLA-ZOSTER VIRUS | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12 | 15 |
| 1306 | HERPES SIMPLEX TYPE 1 | 2 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 55 | 63 |
| 1307 | HERPES SIMPLEX TYPE 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 57 | 57 |
| 1502 | PICORNIA VIRUS - NOT TYPED = E | 1 | 3 | 1 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 1 | 9 |
| 1514 | MOLLUSCUM CONTAGIOSUM | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 |
| 1521 | MEASLES VIRUS | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 1522 | RUBELLA VIRUS | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 7 |
| 1532 | HEPATITIS B ANTIGEN | 61 | 0 | 0 | 0 | 0 | 0 | 0 | 15 | 0 | 0 | 0 | 76 |
| 1535 | HEPATITIS A ANTIBODY | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 12 | 0 | 0 | 0 | 15 |
| 1536 | HEPATITIS C VIRUS | 103 | 0 | 0 | 0 | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 108 |
| 1541 | CHLAMYDIA TRACHOMATIS - UNSPEC | 13 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 17 |
| 1542 | CHLAMYDIA TRACHOMATIS - A-K | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1556 | CMV - CYTOMEGALOVIRUS | 3 | 6 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 12 |
| 1563 | CORONAVIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 1564 | ROTAVIRUS | 4 | 1 | 0 | 0 | 0 | 0 | 103 | 0 | 0 | 0 | 0 | 108 |
| 1565 | CALICI VIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 |
| 1566 | NORWALK AGENT | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 2 |
| 1571 | ENTEROVIRUS TYPE 71 (BCR) | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1599 | ENTEROVIRUS TYPING PENDING | 0 | 2 | 0 | 1 | 0 | 0 | 3 | 0 | 0 | 0 | 1 | 7 |
| 9903 | NON-A, NON-B HEPATITIS (OTHER) | 14 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 14 |
| 9993 | ASTROVIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 9995 | DENGUE NOT TYPED | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| TOTAL | | 253 | 321 | 4 | 8 | 1 | 1 | 155 | 35 | 3 | 1 | 149 | 931 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 03/07/91 TO 16/07/91

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 |
| 0101 | ADENOVIRUS TYPE 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 2 |
| 0103 | ADENOVIRUS TYPE 3 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 0104 | ADENOVIRUS TYPE 4 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0108 | ADENOVIRUS TYPE 8 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0135 | ADENOVIRUS TYPE 35 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0199 | ADENOVIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0203 | INFLUENZA B VIRUS | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 2 |
| 0303 | PARAINFLUENZA VIRUS TYPE 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 | 0 | 8 |
| 0400 | RESPIRATORY SYNCYTIAL VIRUS (R | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 7 | 0 | 9 |
| 0600 | MYCOPLASMA PNEUMONIAE | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 3 |
| 0650 | MYCOPLASMA HOMINIS | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0700 | ORNITHOSIS-PSITTACOSIS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0809 | COXSACKIEVIRUS A9 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 1101 | POLIOVIRUS TYPE 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1102 | POLIOVIRUS TYPE 2 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 3 |
| 1301 | HERPES SIMPLEX VIRUS - NOT TYP | 1 | 17 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 21 |
| 1302 | EPSTEIN-BARR VIRUS (EB VIRUS) | 0 | 0 | 23 | 7 | 0 | 0 | 2 | 9 | 2 | 0 | 43 |
| 1303 | VARICELLA-ZOSTER VIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 2 |
| 1306 | HERPES SIMPLEX TYPE 1 | 2 | 11 | 0 | 0 | 0 | 1 | 1 | 0 | 2 | 0 | 17 |
| 1307 | HERPES SIMPLEX TYPE 2 | 0 | 57 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 57 |
| 1399 | HERPES VIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1401 | COXIELLA BURNETII | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 4 | 0 | 6 |
| 1502 | PICORNIA VIRUS - NOT TYPED = E | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| 1521 | MEASLES VIRUS | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 6 | 0 | 7 |
| 1522 | RUBELLA VIRUS | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 2 | 0 | 5 |
| 1532 | HEPATITIS B ANTIGEN | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 2 |
| 1536 | HEPATITIS C VIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 2 |
| 1541 | CHLAMYDIA TRACHOMATIS - UNSPEC | 6 | 86 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 94 |
| 1542 | CHLAMYDIA TRACHOMATIS - A-K | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1556 | CMV - CYTOMEGALOVIRUS | 0 | 0 | 0 | 2 | 1 | 0 | 6 | 8 | 30 | 0 | 47 |
| 1564 | ROTAVIRUS | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 1599 | ENTEROVIRUS TYPING PENDING | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 9992 | ROSS RIVER VIRUS | 0 | 0 | 0 | 0 | 11 | 0 | 0 | 2 | 0 | 0 | 13 |
| TOTAL | | 16 | 175 | 25 | 9 | 14 | 3 | 18 | 25 | 77 | 2 | 364 |