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Communicable Diseases Intelligence

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Contents:

SEASON'S GREETINGS TO ALL OUR READERS

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VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME:

In this period (22 November to 5 December 1990) there were 1199 reports processed.

There were 14 reports of Q-fever for the period (2 females, 11 males and 1 unstated). Case ages ranged from 17 to 64 years and occupational exposure details were given for one - a meatworker.

Rubella infection was diagnosed in a 33-year-old female who was 19 weeks pregnant.

Two cases of enterovirus type 71 - a 21-year-old female with meningitis (a CSF isolate) and a 60-year-old female with hand, foot and mouth infection. There has been increased reporting of this virus during the year (17 reports this year compared to 2-7 reports in the last 3 years).

There were 16 reports of measles for the period. A high titre to measles antibody was associated with a case of encephalitis in an 18-year-old male.

Coxsackievirus B2 was isolated from the cerebro-spinal fluid of a 1-month-old female with meningitis. Coxsackievirus B4 meningitis was confirmed by isolation from the CSF of a 1-year-old male and a 13-year-old female.

NON-VIRAL PATHOGEN REPORTS

Tables (2) summarising non-viral pathogen reports received for samples collected in July, August and September are included in this issue of CDI after the virus tables. Similar tables for the first six months of the year appeared in CDI 90/20.

Twelve additional blood culture reports have been received for November (3 from Nambour Hospital Laboratory, 4 from Toowoomba Base Hospital Laboratory and 5 from Dr Lynch's Rockhampton Pathology Laboratory). The following organisms were isolated:

- *Actinobacillus actinomycetemcomitans* from a 15-year-old male post-splenectomy;
- *Bacillus melaninogenicus* from a 74-year-old female with renal failure.
- *Escherichia coli* from 5 patients, a 61-year-old male with pyelonephritis, and 68-year-old female on chemotherapy for cancer, an 83-year-old male with nephrotic syndrome, a 82-year-old female and a patient over 65 years with no further clinical information.

. *Neisseria meningitidis* group Y from a 42-year-old male.

. *Staphyloccocus aureus* in 2 patients, a male aged 64 years and a female aged 71 years (now deceased).

. *Streptococcus milleri* from a 1-day-old female with *Streptococcus agalacticae* in the gastric aspirate.

Further interesting reports include:

Helicobacter pylori was isolated from digestive tract biopsies of 6 patients (5 males aged 43-70 years and one 71-year-old female).

Haemophilus influenzae type b from the joint fluid of a 9-month-old female with septic arthritis.

Enterobius vermicularis in lower gastrointestinal biopsies of 2 patients, a 12-year-old male with chronic appendicitis and a 31-year-old male with suppurative appendicitis.

Yersinia enterocolitica biotype 4 serotype 03 was isolated from the faeces of 4 children, 3 females aged 2,7 and 12 years and a 3-year -old male.

(The above 13 reports were received from Dr Lynch's Rockhampton Pathology Laboratory).

Shigella sonnei was isolated from the faeces of two siblings (brother and sister) aged 12 and 10 years. (Toowoomba Base Hospital Laboratory).

Eighteen reports of *Bordetella pertussis* have been received so far for November (14 from Dr Lynch's Rockhampton Pathology Laboratory and 4 from Toowoomba Hospital Laboratory) comprising 9 children (aged 5-14 years), and 9 adults (aged 19-44 years). The pertussis epidemic seems to be continuing in Queensland. If laboratories have noticed increased whooping cough activity in their area, reports would be appreciated.

OVERSEAS BRIEFS

1. YELLOW FEVER IN CAMEROON

The outbreak of yellow fever which began in September is continuing and 108 cases with 81 deaths have been reported to 20 November 1990. The two regions (departments) affected are Mayo Sava and Mayo Tsanaga in extreme-Nord province. Vaccination programs are being hampered by limited supplies.

2. CHOLERA IN GHANA

The Ministry of Health has reported an outbreak of cholera in and around Accra. As at 2 November 1990, suspected cases 572 with 94 confirmed and 19 associated deaths. Travellers are reminded to follow the normal precautions regarding food and drinking water.

NHMRC Childhood Immunisation Schedule, November 1990

Widespread immunisation of children with diphtheria, tetanus, pertussis, measles and poliomyelitis vaccines has led to a marked reduction in the number of cases and deaths from pertussis and the prevention of diphtheria, tetanus and poliomyelitis in those immunised. Schoolgirl rubella immunisation has been one of the key factors in the near-eradication of rubella embryopathy and more recently measles and mumps vaccination have greatly reduced morbidity and mortality from those diseases.

Notes on the Schedule:

1. **Combined vaccines** are used where possible in routine immunisation of infants: Diphtheria, tetanus, pertussis (DTP or triple antigen); Measles, mumps, rubella (MMR); CDT (Diphtheria, tetanus).

2. DTP should be used for primary immunisation commencing under the age of **four** years.

3. A combined diphtheria and tetanus (CDT) vaccine is used -

   a) for primary immunisation of infants where the pertussis component is contraindicated (see below). In this situation, CDT is recommended in three doses, i.e. at **two, four** and **eighteen** months.
   b) for booster doses in children to the age of **seven** years.
   c) for primary immunisation of infants who have already suffered bacteriologically confirmed pertussis.

4. A combined diphtheria and tetanus vaccine suitable for adults (ADT) is used from the age of **eight** years for primary immunisation and for intermittent booster doses (see tetanus section). This vaccine has a lower concentration of diphtheria toxoid and causes fewer side effects in older individuals.

5. Where primary immunisation has been with CDT and it is subsequently decided that pertussis immunisation should now be given, three injections of monovalent pertussis vaccine can be given in children aged less than two years a 2 month intervals, subject to the precautions as in (8) below and the availability of the vaccine.

6. Immunisation of premature infants should be commenced at two months after birth, providing there are no contraindications. These infants, which especially need protection, usually have adequate antibody responses and have a low incidence of adverse reactions.

7. Immunisation is commenced at two months because of the high morbidity and established mortality of pertussis in infancy.

NO CHILD SHOULD BE DENIED IMMUNISATION WITHOUT SERIOUS THOUGHT AS TO THE CONSEQUENCES BOTH FOR THE INDIVIDUAL CHILD AND THE COMMUNITY.

RECOMMENDED CHILDHOOD IMMUNISATION SCHEDULE (NHMRC) - 1990

AGE	DISEASE	VACCINE	ROUTE
2 months	Diphtheria, tetanus and pertussis	Triple antigen 'DTP'	Intramuscular
	Poliomyelitis	Sabin vaccine 'OPV'	Oral
4 months	Diphtheria, tetanus and pertussis	Triple antigen 'DTP'	Intramuscular
	Poliomyelitis	Sabin vaccine 'OPV'	Oral
6 months	Diphtheria, tetanus and pertussis	Triple antigen 'DTP'	Intramuscular
	Poliomyelitis	Sabin vaccine 'OPV'	Oral
12 months	Measles/mumps/ rubella	Measles/mumps/ rubella 'MMR'	Subcutaneous
18 months	Diphtheria, tetanus and pertussis	Triple antigen 'DTP'	Intramuscular
5 years or prior to school entry	Diphtheria and tetanus	Combined Diphtheria and Tetanus 'CDT'	Intramuscular
	Poliomyelitis	Sabin vaccine 'OPV'	Oral
10-16 years (females only)	Rubella (Preferably in last year of primary school or first year of high school).	Rubella vaccine	Subcutaneous
15 years or prior to leaving school	Diphtheria and tetanus	Adult Diphtheria and Tetanus 'ADT'	Intramuscular

8. **Precautions and contraindications:**

a) Immunisation should not be carried out during the course of a significant acute illness.

b) Any major reaction following DTP (triple antigen) is likely to be due to the pertussis component and any further DTP or monovalent pertussis vaccine is contraindicated. In this situation there is no indication to use a smaller dose of any pertussis vaccine. Major reactions include -

(i) fever above 40.5°C;
(ii) convulsions (uncommon);
(iii) hypotonic/hyporesponsive episodes (uncommon);
(iv) shock, anaphylaxis, thrombocytopaenia and encephalopathy (all extremely rare);
(v) severe local reactions;
(vi) persistent screaming (more than 3 hours).

Mild local reactions occur very frequently (up to 50%) following DTP and **do not** contraindicate further DTP. Rarely sterile abscesses have been observed. It is believed these are more likely to occur when the vaccine is given subcutaneously.

(c) Infants known to have active or progressive neurological disease including recent convulsions should not be given pertussis-containing vaccines. For infants with stable neurological disease (including controlled epilepsy) or a family history of idiopathic epilepsy or other familial neurological disorder, the risks of pertussis **infection** still greatly outweigh the risks of pertussis **immunisation**, so that routine immunisation including DTP should be commenced, subject to the normal precautions. If there is doubt in individual cases consultation with a paediatrician or paediatric neurologist may be appropriate.

(d) A history of allergic symptoms is **NOT** a contraindication to childhood immunisation. In particular, eczema, hay fever, asthma and "snuffles" are not valid reasons for deferring immunisation.

(e) CDT is contraindicated only if there has been a severe local, or systemic reaction to previous CDT immunisation.

(f) For precautions and contraindications in regard to poliomyelitis, measles, mumps and rubella, see relevant sections below. Precautions and contraindications for MMR vaccine are the same as those for measles vaccine.

(g) **The use of paracetamol:** Paracetamol (15 mg/kg, for 3-4 doses at 3-4 hourly intervals), is effective in reducing febrile reactions following DTP.

9. **Site of injections:** Vaccines and antisera should be administered in an area where there is minimal opportunity for local, neural, vascular, or tissue injury. Subcutaneous injections are usually administered into the deltoid area. Intradermal injections are generally given on the volar surface of the forearms. The exception to this rule is human diploid cell rabies vaccine where reactions are less severe in the deltoid area.

Currently, the preferred site for intramuscular injections is the deltoid muscle of the upper arm in adults and children 12 months of age and over, and the anterolateral aspect of the upper thigh in infants less than 12 months. An individual decision must be made for each child, based on the volume of the material to be injected and the size of the muscle into which it is to be injected. In adults, the deltoid is generally used for routine intramuscular vaccine administration. Gluteal intramuscular injection is not recommended for vaccines.

10. If the recommended intervals between doses are exceeded, immunisation can be continued with subsequent doses; there is no need to recommence the schedule or give additional doses, because the immune response is not impaired by such delay.

11. Meticulous attention must be given to transport and storage of vaccines prior to administration. Storage at a temperature between $2\text{-}8^{0}C$ is the usual, unless otherwise stated on the product information sheet. Freezing of inactivated vaccines will result in a loss of potentcy.

12. Live vaccines may be given simultaneously at different sites without compromising antibody response.

13. Some states and territories are implementing the NHMRC recommendation of 1982, that evidence of immunisation, given to the child, should be provided prior to school entry so that unimmunised children may be excluded from school during disease outbreaks.

14. From time to time, NHMRC will vary the recommended immunisation schedule, depending upon changes in disease prevalence, available vaccines and other relevant factors.

15. In measles epidemics, public health officials may recommend measles vaccine for infants as young as six months of age. Although a smaller proportion of those given vaccine before the first birthday develop antibody to measles, compared with older infants, the higher risk of disease during an epidemic may justify earlier immunisation. Such infants should be re-immunised at the recommended age for measles vaccination to achieve protection.

MEASLES NOTIFICATIONS RECEIVED IN THE ACT - JANUARY TO OCTOBER 1990

(Contributed by R J Scott and I Passaris, Department of Health, ACT)

There has been a considerable increase in the number of measles notifications received in the ACT this year. 23 cases of measles were reported during the period January to October 1990, compared with 8 for the year 1989 and 1 in 1988. Of the 23 cases reported, 17 occurred between June and October. Serological confirmation of the diagnosis was received for 10 cases and 6 children were hospitalised for periods varying from 1 to 5 days.

The Chief Health Officer issued a Press Release on 4 September 1990 to inform the public of the increase in measles and pertussis notifications and to urge parents to have their children immunised.

The NSW Department of Health recently reported an increase in the incidence of measles this year, particularly in the Hunter and South East Regions of NSW. Victoria has also reported an increase in measles notifications. [CDI Note: articles on measles activity around Australia have appeared in CDI bulletin, 90/20 (8 October) and 90/23 (19 November)].

The increase in the reported incidence of measles is particularly disappointing as media campaigns promoting measles immunisation were conducted in the ACT in both 1988 and 1989 as part of the Commonwealth's Bicentennial initiative to eradicate this.

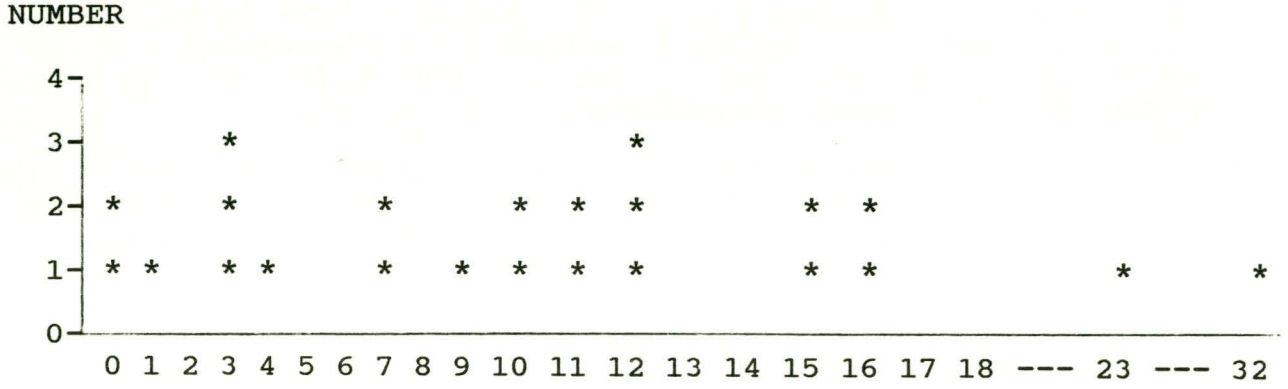
An investigation was conducted to establish the immunisation history of the 23 persons reported as having measles this year. ACT Child Health Immunisation records were checked in the first instance. If no record of measles immunisation was found the treating doctor was contacted. Parents of children were also contacted if the doctor was not able to verify immunisation status.

The categorisation of immunisation status (with respect to measles) is as follows:

Immunised	6
Immunisation claimed without evidence	7
Not immunised	9
Not known	1

The ages of the persons reported as having measles varied from 10 months to 32 years. Details of the age distribution and immunisation status are shown below.

Age distribution of persons with measles



Ages of persons reported to have measles (with respect to immunisation status)

Immunised	3	11	12	12	15	16				
Immunisation claimed:	3	7	9	10	10	12	15	16		
Not immunised:	10mths	10mths	10mths	3	7	11	16	23	32	
Not known	4									

LYME DISEASE IN AUSTRALIA - THE SEARCH FOR THE AETIOLOGICAL AGENT AND ITS VECTOR

Introduction

Lyme disease is a multisystem spirochaetal disease which has been recognised in Europe for many years, and was first described in the United States in 1977. It has protean manifestations and affects the skin, nervous system, joints and heart. Signs and symptoms include a rash, headache, malaise, fever, chills, stiff neck, arthralgia, myalgia, conjunctivitis, elevated transaminases, cranial nerve palsies, peripheral radiculoneuropathy, atrioventricular block and oligoarthritis.

The aetiological agent is *Borrelia burgdorferi* and is transmitted to man by *Ixodes* ticks belonging to the *Ixodes ricinus* group.

Although a clinical syndrome resembling Lyme disease has been described in Australia neither the aetiological agent(s) nor its vector(s) have been described in this country.

A report on the Australian and overseas experience with this syndrome has been published in CDI previously (1).

The search for the aetological agent and vector

As noted earlier (1) a research program is being undertaken by Dr R C Russell at Westmead Hospital, Westmead, NSW in an attempt to isolate the aetiological agent and identify the vector. A similar program is being undertaken by Mr R Silcock at the State health Laboratories, Brisbane, Queensland.

Medical practitioners in New South Wales, Victoria, South Australia, Western Australia, Tasmania and the Australian Capital Territory removing ticks from patients with suspected Lyme disease are encouraged to forward the tick to:-

Dr R C Russell
Head, Medical Entomology Unit
Department of Infectious Diseases and Microbiology
Westmead Hospital
WESTMEAD NSW 2145

Ticks collected from patients in Queensland and the Northern Territory should be forwarded to:-

Mr R Silcock
Serology Section
State Health Laboratories
63 George Street
BRISBANE QLD 4000

Ticks and other insects are more active during the warmer months and this, together with increased outdoor human activity renders man more susceptible to exposure to vectors of this disease. Practitioners are more likely to see patients with tick bites from the beginning of December until about the end of March.

Tick removal, handling and transportation.

The mouthparts of a tick are shaped like small barbs. The tick, which is about 1mm in size, should be pulled out gently, but firmly, at 90 degrees to the skin and without twisting. Experience in north America indicates that even if the pincers break off and remain embedded in the skin, the risk of infection is not great because the highest concentration of infectious agent is in the tick's stomach. However, because it is not possible to extrapolate these findings with confidence to the Australian situation, care should be taken to remove as much of the tick as possible.

The tick should not be squashed due to the possiblity of exposure to the infectious agent, and the skin of both patient and the person removing the tick should be washed thoroughly after removal. Tick removal should not be attempted using heated matches, alcohol, nail polish, vaseline or similar agents. The attendant stimulation of the tick may cause it to regurgitate stomach contents and infect the patient.

The specimen should be sent, if possible in the live state, in a screw-capped glass or plastic vial up to about 50mL in size containing some loose, dry packing material such as facial tissue, with one drop of water added. Ticks can survive for many months without nutrients at room temperature. The water is added to increase humidity in the tube and assists in maintaining viability of the specimen. It is important that only one drop is added. Excess water will encourage fungal growth which will destroy the insect.

Reference

1. Communicable Diseases Intelligence 1990, 90/18, P4

AUSTRALIAN HIV SURVEILLANCE SUPPLEMENTARY REPORT - OCTOBER 1990

The following two tables have been taken from the Australian HIV Surveillance Report Volume 6, Supplement 2 (October 1990). Another two reports were published in CDI 90/24.

Table 1: Deaths following AIDS by sex and exposure category, cumulative to 30 September 1990, and for two previous yearly intervals.

Adults/adolescents (13 years and older at diagnosis of AIDS)

EXPOSURE CATEGORY	1 Oct 88 - 30 Sep 89		1 Oct 89 - 30 Sep 90		Cumulative to 30 Sep 90		
	Male	Female	Male	Female	Male	Female	Total(%)
Male homosexual/bisexual contact	280	-	351	-	1111	-	1111(87.5)
Male homosexual/bisexual contact and IV drug use	6	-	11	-	30	-	30(2.4)
IV drug use (female and heterosexual male	2	0	6	2	10	3	13(1.0)
Heterosexual contact:	1	4	0	2	2	7	9(0.7)
Sex with IV drug user	0	1	0	0	0	1	1(-)
Sex with bisexual male	-	2	-	1	-	4	4(-)
Sex with person from Pattern-II country	1	0	0	0	2	0	2(-)
Sex with transfusion recipient	0	1	0	0	0	1	1(-)
Sex with HIV-infected person of unknown exposure category	0	0	0	1	0	1	1(-)
Haemophilia/coagulation disorder	4	0	5	0	17	0	17(1.3)
Receipt of blood transfusion, blood components, or tissue	4	4	1	0	23	25	48(3.8)
Other/undetermined	9	0	10	2	28	2	30(2.4)
Total Adults/Adolescents	306	8	384	6	1221	37	1258(99.1)

Children (under 13 years at diagnosis of AIDS)

Mother with/at risk for HIV Infection	0	0	0	1	0	2	2(0.2)
Haemophilia/coagulation disorder	0	0	1	0	2	0	2(0.2)
Receipt of blood transfusion, blood components, or tissue	0	0	1	0	8	0	8(0.6)
Total Children	0	0	2	1	10	2	12(0.9)
TOTAL	306	8	386	7	1231	39	1270(100)

The number of cases of AIDS attributed to receipt of blood or other tissue does not appear to be decreasing.

Table 2: Cases of AIDS by AIDS-defining condition and sex, cumulative to 30 September 1990, and for two previous yearly intervals.

AIDS-DEFINING CONDITION	1 Oct 88 - 30 Sep 89		1 Oct 89 - 30 Sep 90		Cumulative to 30 Sep 90		
	Male	Female	Male	Female	Male	Female	Total(%)
Pneumocystis carinii pneumonia (PCP)	220	3	150	0	806	12	818(39.3)
Kaposi's sarcoma (KS) - skin	82	0	47	0	306	2	308(14.8)
KS and PCP only	6	0	2	0	23	0	23(1.1)
KS and other (not PCP)	6	0	7	0	33	0	33(1.6)
PCP and other (not KS)	29	0	34	1	135	8	143(6.9)
Candidiasis - oesophageal	31	2	16	0	97	6	103(5.0)
Toxoplasmosis - cerebral	20	0	18	1	78	2	80(3.8)
Cryptococcosis-meningeal	27	0	16	1	70	1	71(3.4)
Lymphoma - non-Hodgkin	19	1	15	0	63	3	66(3.2)
Mycobacterium - avium	12	0	16	2	54	3	57(2.7)
Herpes simplex virus	7	0	6	0	49	2	51(2.5)
HIV encephalopathy	21	1	15	0	48	2	50(2.4)
Cytomegalovirus	13	0	12	0	46	0	46(2.2)
HIV wasting disease	9	2	13	0	28	3	31(1.5)
Cryptosporidiosis - gut	11	1	4	0	28	2	30(1.4)
Mycobacterium - tuberculosis (TB)	1	1	0	1	9	2	11(0.5)
Other single diagnoses	8	0	7	0	29	3	32(1.5)
Other multiple diagnoses	16	2	33	3	116	12	128(6.1)
TOTAL	538	13	411	9	2018	63	2081(100)

The proportion of diagnoses of AIDS related to KS is slightly lower in the past two years than previously.

FOURTH NATIONAL CONFERENCE ON AIDS - ABSTRACTS

The following three abstracts have been taken from the proceedings of the 4th National Conference on AIDS held by the Department of Community Services and Health from 8-11 August 1990. Another four abstracts were published in CDI bulletin 90/23 (19 November).

Infection with Human Immunodeficiency Virus among Blood Donors in Australia

D J Castelino and B M Whyte

Transfusion transmitted acquired immunodeficiency syndrome (AIDS) was first recognised in the USA in 1982, and in Australia in 1984. Voluntary deferral of potential blood donors from "high risk groups" was introduced in 1983, and, once a screening test was available in 1985, a statutory declaration form was brought in. Donors have to declare that they have not indulged in "high risk" activities, and donations are, in addition, screened for antibody to HIV. No cases of post-transfusion HIV infection are known to have arisen from blood collected since testing began, in contrast to about 150 cases from transfusions prior to 1985.

Between May 1985 and 31 December 1988, 3,427,768 blood donations have been tested nationally for antibodies to HIV, and 26 confirmed positive results found. The crude cumulative seroprevalence rate per 100,000 was 0.8, ranging from 1.5 in New South Wales (NSW) to 0 in South Australia. Infected donors have been detected only in NSW, Victoria, Queensland and Western Australia.

The maximum sero-incidence per 100,000 was 2.9 in NSW in 1985, decreasing to 1.4 in 1988. Of the 26 donors, 18 were male and 8 female, with a mean age of 28 years overall, and no significant difference in mean age between the sexes, States, year of donation, risk group or whether they were donating for the first time or not. Risk factors are known for 22 donors - 6 homosexual - or bisexual males (one also an intravenous drug abuser [IVDA]); 14 heterosexuals (7 male and 7 female) four having had sex with an IVDA. Two donors did not have a sexual risk factor, but had been transfused while unconscious, and the transfusion history was only discovered after the HIV status was detected.

The Melbourne Prospective Study; Changes over Seven Years

B Mulhall, A Fuller, L Carter and B Donovan

OBJECTIVE: To determine rates of seroconversion and predictors of immunodeficiency and their relation to sexual life-style.

METHOD: In 1983 a cohort of 100 asymptomatic homosexual men were recruited for a prospective study at a major teaching hospital. HIV seroconversion and immune function assessments were performed

at six monthly intervals. In addition, all members were counselled and information on sexual life-style was elicited by questionnaire.

RESULTS: The prevalence of antibody to HIV rose from 22% in 1983 to 31% in 1985. There have been no further seroconversions. The cumulative incidence of AIDS is 23% (7/30) and of HIV-related complications (excluding lymphadenopathy) a further 13% (4/30). Hochterling analysis demonstrates the predictive value of repeated measurements in the seropositive group for CD4T cells ($t=2.2$, $p<0.01$) and immunoglobulin concentrations ($t=3.5$, $p<0.001$). Mean Beta 2 microglobulin levels were higher in the HIV + ve group, but the difference was not significant owing to the small number of stored serum samples. There has been a highly significant decrease in the number of sexual partners and the number of reported STDs ($p<0.01$). There has also been a significant fall in the frequency of unsafe sex, though not to zero, and an increase in the use of condoms. This applied to receptive and active anal intercourse, but not to oral intercourse.

CONCLUSIONS: 1. The spread of HIV into and within this group has been halted. 2. Although there are clear group trends; for a single individual, serial and not point-measurements are required to predict immunodeficiency. 3. The combination of motivation and counselling has resulted in a reduction of practices perceived to be unsafe.

Mycobacterial Diseases in HIV Infection

Dr Deborah Marriott

Immunodeficiency is a well-recognised risk factor for infection with tuberculous and non-tuberculous mycobacteria, and the association of disease caused by these organisms and the human immunodeficiency virus (HIV) has been appreciated since the first cases of AIDS were described in the early 1980's. Early studies linked tuberculosis with specific patient groups such as intravenous drug users or Haitians. It is now recognised that not only is the incidence of tuberculosis increased in all people with HIV infection, but that HIV infection is found in approximately one-third of new cases of tuberculosis. At St Vincent's Hospital 12/765 (1.6%) of AIDS diagnoses have *M. tuberculosis* infection which far exceeds the expected incidence for Caucasian males in New South Wales of approximately 2/100,000 per annum. Although many cases are due to reactivation of latent disease, the clinical presentation is more typical of progressive primary tuberculosis. Extrapulmonary disease occurs in 1/2 to 2/3 of patients, most frequently involving lymph nodes, pleura and bone marrow. Bacteraemia with *M. tuberculosis* has been described. Diagnosis is complicated by anergy to tuberculin skin testing in up to 90% of patients, depending on the degree of immunodeficiency. Treatment with standard anti-tuberculous therapy is usually successful although adverse drug reactions occur in approximately one-third.

Disseminated *Mycobacterium avium* complex (MAC) infection was rare before the advent of AIDS. The organism is a common environmental saprophyte and has been isolated from 132/765 (17.2%) of AIDS patients at St Vincent's Hospital. However autopsy studies suggest that up to one-half may be infected with MAC, with colony counts up to 10^{10}cfu/gm of tissue. The role of MAC in disease is uncertain although several clinical syndromes have been described. Therapy of MAC is complicated by in vitro resistance to all standard anti-tuberculous agents and most secondline drugs, poor clinical and bacteriologic response, and the lack of data from controlled trials.

HEPATITIS C VIRUS AND BLOOD TRANSFUSION SERVICES

(Contributed by R J Kimber, National Blood Transfusion Committee)

Red Cross Transfusion Services have been routinely screening all blood donations for Hepatitis C virus (HCV) since about mid-February 1990. Approximately 0.6% of all donors are found to be reactive to the test and thus are potential carriers of HCV, although a positive antibody test does not necessarily indicate a risk of transmitting the infection. The HCV antibody test is subject to false positives.

Confirmatory tests are being developed which will improve the sensitivity and specificity of the screening test. Approximately one-third of samples which are positive on screening are positive with the available "confirmatory" tests.

Since July 1990 potential donors who are found to be HCV positive and who have one or ore abnormal liver function tests have been referred to a specialist clinician for advice and management. At the same time it was decided that HCV positive plasma would no longer be used for the preparation of any blood products.

Concerns regarding the exclusion of HCV positive donors have highlighted the need to maintain rigorous donor selection measures. The donor declaration forms designed to exclude donors 'at risk' of transmitting HIV infection have been further upgraded to address the risk presented by HCV.

GONOCOCCAL SURVEILLANCE - AUSTRALIA 1 APRIL - 30 JUNE 1990

(Contributed by the Australian Gonococcal Surveillance Programme - AGSP. Co-ordinator, J W Tapsall, The Prince of Wales Hospital, Sydney.

In the three months to 30th June 1990, 412 isolates of *N. gonorrhoeae* were examined in reference laboratories throughout Australia. This total is considerably less than the 513 strains isolated in the corresponding period in 1989. All centres, with the exception of Brisbane, recorded a decrease in the number of isolates examined.

It has been usual in past reports to show the percentage of isolates which fell into the categories of "fully sensitive" or "less sensitive" to penicillin or which were PPNG producers in a Table format. However, the number of isolates in some centres is now too low to validly express data in percentage terms and these are now shown for Sydney, Melbourne and Brisbane only (see Table 1). In a number of previous reports it has been noted that the percentage of isolates relatively resistant to penicillin (MIC>1.0mg/L) has been increasing in a number of centres and the percentage of isolates fully sensitive to penicillin was decreasing. This trend appears to have been reversed in this quarter. Only 10 strains falling into this category were isolated in Australia in this quarter whereas the number of fully sensitive percentage terms.

The total number of isolates of PPNG in this period was 76. In this quarter in 1988, 124 PPNG were isolated and 101 lactamase producers were reported last year. PPNG were isolated in low numbers in Darwin, Perth and Adelaide, however, strains isolated in Brisbane, Sydney and Melbourne accounted for 69 of the strains of this type. Locally-acquired PPNG accounted for 14% of all gonococci isolated in Sydney. For strains isolated in Melbourne, in 16 of 28 cases the infections were acquired overseas or were the result of contact with a patient who contracted the disease outside Australia. In a further 4 cases the disease was locally acquired. Most of the overseas contacts with PPNG were in S-E Asia, but in this quarter in addition there were three strains imported from different parts of Africa.

This report completes nine years of AGSP reports in the CDI. Changes in antibiotic sensitivity of gonococci continue to occur, these being mediated by both chromosomal and extra-chromosomal mechanisms. From time to time changes have occurred both in the total number of gonococcal isolates and the sites from which gonococci are isolated. There are indications from data reported here and also from additional AGSP material of further changes in gonococcal epidemiology in Australia. These will be the subject of more detailed analyses at a future date.

Reference

1. Med J Aust, 1988, 149: 609-612

Table 1: Penicillin sensitivity of isolates of *N. gonorrhoeae* 1 April - 30 June, 1990

Centre	Percentage of Isolates * Sensitive		** Less Sensitive		PPNG	
Brisbane	21	(14.7)	55.6	(56.8)	15	(17.9)
Sydney	11	(2.3)	47	(50)	25	(37.1)
Melbourne	6.2	(2.8)	59	(66.2)	24.7	(17.6)

* Sensitive, MIC = 0.004 - 0.016 mg/L

** Less Sensitive, MIC = 0.06 - 0.25 mg/L

PPNG = penicillinase producing *N. gonorrhoeae*

Figures in parenthesis represent data for the equivalent period in 1989.

NOSOCOMIAL CHICKEN POX

(Contributed by J L Faoagali, Director Medical Microbiology, Royal Brisbane Hospital, Brisbane and R Horhmann, Infection Control Nurse, Royal Children's Hospital, Brisbane).

Chicken pox is a highly infectious childhood disease with an incubation period of 2 to 3 weeks - commonly 13 to 17 days. The disease is caused by the varicella-zoster (VZ) virus, a member of the herpesvirus group (Family Herpetoviridae).

Asymptomatic children may be admitted to hospital in the incubation period of the disease with unrelated conditions and this may result in a widespread and prolonged epidemic unless the disease is quickly recognised and appropriate containment measures are instituted. This brief report details the recognition of a case and the containment of cross infection in contacts to one generation. The importance of surveillance in the control of nosocomial infections is highlighted.

On 26 March 1990 a 7½ year old boy was admitted to the orthopaedic ward of the Royal Children's Hospital, Brisbane with a fractured radius and ulna. These fractures were stabilised and immobilised on the day of admission and the child was discharged three days later (Table 1).

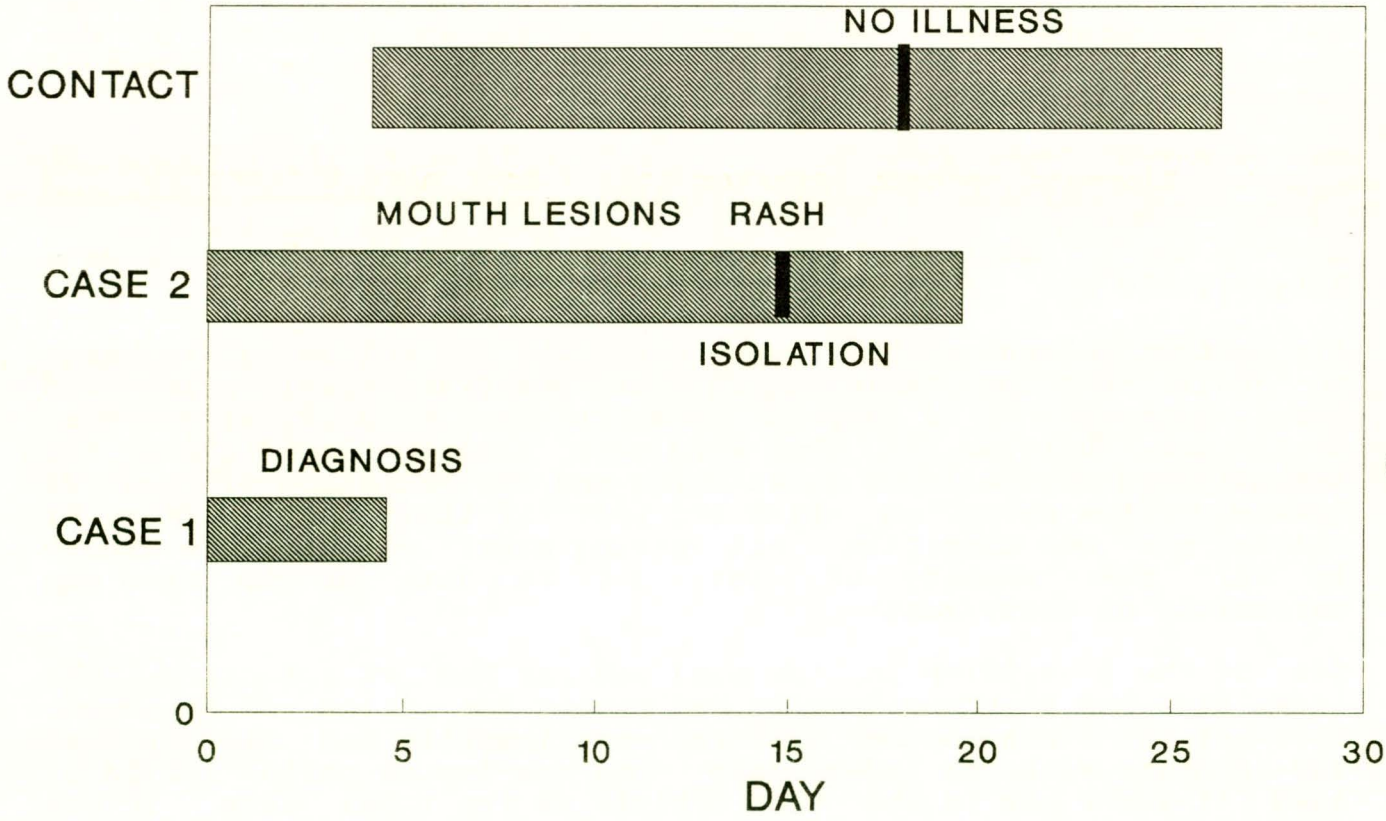
His mother telephoned the charge nurse the day following discharge to advise that the child had chicken pox (varicella), which had been diagnosed by a general practitioner on clinical grounds. Staff and children in the ward were assessed immediately for immunity to varicella by questioning and determination of their VZ immune status using an ELISA IgG test if their past history of chicken-pox was uncertain. All nursing staff on duty were immune to VZ. The immunity of staff not on duty at the time was determined on their return.

Six of the 8 children in the ward had not had VZ infection. The index case had been confined to bed except for toilet and bathroom. Five of the 6 non-immune children were immobile and did not have any contact with the index case. One non-immune child (child 2) aged 11 years was in the same cubicle as the index case. It was decided to keep this child in a side room in the main ward because there was a requirement for intensive physiotherapy which could not be provided as an out-patient as the normal domicile was distant from these facilities. Part of the physiotherapy included the use of a passive knee mobiliser. This mobiliser was shared with another child who had previously had varicella.

It was decided that as the contact child had to remain in hospital he would be confined in isolation from day 16 to day 21 after first contact with the index case, but would be allowed to attend physiotherapy and hydrotherapy until day 16. The contact was able to be so defined because of the short hospital stay of the index case. Any other child in the orthopaedic ward during the period of admission of the index case and requiring re-admission during the next 21 days was to be re-admitted to the isolation ward. The orthopaedic ward and the Accident and Emergency staff were informed of this decision, but no 'at risk' child was re-admitted. On 9 April, (day 15), the contact child developed mouth lesions. This was the day before he was due to be fully isolated. On day 16, typical VZ lesions were present and he was transferred to the isolation as previously planned. One other child (7 months old) admitted on 29 April 1990 was non-immune to VZ and had been mobile during the 'at risk' period. There was concern that this child may have been in unprotected contact with child 2 or his fomites. He was confined for a further 5 days, but no lesions developed prior to or after discharge.

No other children became infected.

NOSOCOMIAL VARICELLA-ZOSTER



The successful containment of this infection was due to the early involvement of the Infection Control Nurse and appropriate containment measures which not only enabled the contact child to continue with the necessary treatments, but removed the potential risk of contracting this infection from other children and staff.

CDI EDITORIAL COMMENT.

This report highlights the effectiveness of infection control procedures in the prevention of nosocomial infection with varicella -zoster virus. Infection with this virus may result in potentially serious sequelae in immunocompromised persons.

Early consideration should be given to treatment with Zoster Immunoglobulin (ZIG) of any high risk patient who is exposed to infection with VZ virus. ZIG is prepared from screened plasma donations obtained by the Australian Red Cross Society from donors who have recently recovered from VZ infection.

ZIG has been shown to prevent varicella in susceptible contacts of an index case, and has been used successfully to prevent the spread of this infection in high risk patients. Greatest effectiveness is to be expected when treatment is begun within 96 hours of exposure. Treatment after 96 hours is of uncertain value.

Supplies of ZIG are limited:- administration should be restricted to individuals meeting the following criteria-

1. One of the following underlying illnesses or conditions:-
   a. Neoplastic disease (leukaemia or lymphoma);
   b. Congenital or acquired immunodeficiency;
   c. Immunosuppressive therapy with steroids or antimetabolites.

2. One of the following types of exposure to chickenpox or zoster patients:-
   a. Household contact;
   b. Playmate contact (greater than 1 hour play indoors);
   c. Hospital contact (in same 2 to 4 bed room or adjacent beds in a large ward;
   d. Newborn contact (newborn of mother who had onset of chickenpox less than 5 days before delivery or within 48 hours after delivery);
   e. Premature infant (28 weeks' gestation or greater) whose mother lacks a prior history of chickenpox;
   f. Premature infant (less than 28 weeks' gestation, or 1,000g or less regardless of maternal history.

3. Negative or unknown prior history of chickenpox.

4. If ZIG can be administered within 96 hours after exposure.

TYPHOID AND PARATYPHOID REPORT, MDU, 1986-1989

(contributed by D Lightfoot, Phage Typing Reference Laboratory, Microbiological Diagnostic Unit, University of Melbourne)

Phage typing results of Salmonella Typhi and Paratyphi sent to the Australian Phage Typing Reference Laboratory between 1 January 1986 and 31 December 1989 are listed below.

This report was presented at the meeting of the International Federation of Enteric Phage Typing held during the XV International Congress of Bacteriology and Mycology, Osaka, Japan, September 1990.

Explanation of Tables:

The percentage of occurrence of each phage type is given in brackets and they are listed in decreasing order of frequency.

Each grouping is divided into a maximum of three parts:

a) includes the most frequent phage types (those for which the cumulative percentage reaches about 90%).

b) the less common types whose individual percentages are still over 0.5.

c) indicates the rare phage types whose individual percentages are less than 0.5.

The recognised Vi-phage types have either lettered or numerical designation.

The "I+IV" strains are resistant to all the specific phage preparations derived from Vi-phage II of Craigie and Yen, but they are susceptible to Vi-phage I or Vi-phage IV.

The degraded Vi-strains (DVS) react widely with the typing phages but their phage sensitivity patterns do not conform to a recognised Vi-phage type.

RDNC(Paratyphi A and B scheme), strains react with the typing phages but do not conform to designated phage types.

Salmonella Typhi:

AUSTRALIA

Type distribution by cases: 268

a) E1(18.3);DVS(14.5);A(9.7);B1(9.3);I+IV(7.8);46(5.6); D2(4.9);B1-variant(3.4);B2(3.4);28(3.0);32(2.6); Vi-negative z66(2.6);J1(2.6);0(2.6);C1(1.9).

b) E2(1.5);51(1.1);D1(0.7);D9(0.7);E7(0.7);M1(0.7); N(0.7);27(0.7).

c) D6(0.4);E10(0.4);G1(0.4);M4(0.4);T(0.4);34(0.4) 60(0.4);Vi-negative(0.4).

PAPUA NEW GUINEA

Type distribution by cases: 134

a) D2(80.6);E1(14.2)

b) B1(2.2);Vi-negative(1.5);60(0.7);B1-degraded(0.7).

PHILIPPINES

Type distribution by cases: 328

a) B1-degraded(26.2);B1(25.3);A(19.2);E2(18.9);DVS(7.0).

b) C1(1.2);I+IV(1.2).

c) Vi-negative(0.3).

Salmonella Paratyphi A

AUSTRALIA

Type distribution by cases: 96

a) RDNC(30.2);1(29.2);untypable(20.8);2(10.4);6(5.2).

b) 3(1.0);4(1.0);5(1.0);9(1.0).

Salmonella Paratyphi B

AUSTRALIA

Type distribution by cases: 42

a) Taunton(40.5);3a1 var.1(19.0);Dundee(7.1);1(4.8);
3a var.4(4.8);Dundee var.1(4.8);Dundee var.2(4.8);
untypable(4.8);RDNC(4.8).

b) 1 var.3(2.4);Battersea(2.4).

FOOTNOTE:

Phage typing is an excellent epidemiological marker for isolates of S.Typhi, S.paratyphi A, S.Paratyphi B and S.Java.

Please send isolates of these organisms to the Microbiological Diagnostic Unit, Melbourne University for phage typing.

NOTES FROM THE LITERATURE

AN ECOLOGICAL NICHE FOR CRYPTOCOCCUS NEOFORMANS VAR. GATTII?

Cryptococcus neoformans is a yeast which causes pulmonary infections and meningoencephalitis, particularly in immunocompromised persons.

Two varieties of this organism have been recognised;- *C. neoformans var. neoformans* and *C. neoformans var. gattii*. The former variety

is commonly found in bird excreta, but until recently a natural habitat for *C. neoformans var. gattii* has not been identified.

Ellis and Pfeiffer (1990a) have published evidence that *C. neoformans var. gattii* is environmentally associated with *Eucalyptus camaldulensis* (common name, the River Red Gum). This tree is native to Australia, although it is now grown widely in other parts of the world. The authors indicate that the global distribution of *E. camaldulensis* appears to correspond to cases of infection with *C neoformans var. gattii*, and that this may explain the high incidence of infection with this variety in some areas of Australia, and the low incidence in AIDS patients world-wide.

In later report, Ellis and Pfeiffer (1990b) expand on these findings and propose that exposure to this species of tree is required to initiate infections with *C. neoformans var. gattii* in both man and animals. They suggest that the principal infectious propagule is the sexual form of the fungus. They further propose that its ecology includes the koala (*Phascolarctos cinereus*) as an intermediate host, with infected plant debris and animal dung in certain sheltered habitats acting as a reservoir for the organism.

A preliminary report of this work has been published elsewhere by Pfeiffer and Ellis (1990).

References

1. Ellis DH and Pfeiffer TJ (1990a). Natural habitat of *Cryptococcus neoformans var. gattii*. J. Clin. Micro. **28:** (7) 1642 - 4.

2. Ellis DH and Pfeiffer TJ (1990b). Ecology, life cycle, and infectious propagule of *Cryptococcus neoformans*. Lancet **336:** 923-25.

3. Pfeiffer TJ and Ellis DH (1990) The natural habitat of *Cryptococcus neoformans var. gattiicfl..* *Aust Microbiologist* **11:** (3) 338.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 22/11/90 TO 05/12/90

CODE 018 - MICROBIOLOGICAL DIAGNOSTIC UNIT, UNIVERSITY OF MELBOURNE (VIC)
CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 065 - STATE HEALTH LABORATORY SERVICES, PERTH (WA)
CODE 066 - PRINCESS MARGARET HOSPITAL, PERTH (WA)
CODE 110 - INSTITUTE OF MEDICAL & VETERINARY SCIENCE, ADELAIDE (SA)
CODE 111 - ROYAL CHIDLDRENS HOSPITAL, MELBOURNE (VIC)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)

		018	019	065	066	110	111	112	114	115	116	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	6	4	2	7	2	1	9	0	31
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	3	0	0	1	4
0102	ADENOVIRUS TYPE 2	0	1	0	0	0	0	1	0	0	0	2
0103	ADENOVIRUS TYPE 3	0	2	0	0	0	0	5	1	0	1	9
0104	ADENOVIRUS TYPE 4	0	0	0	0	0	0	1	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	0	1	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	1	0	0	0	1
0144	ADENOVIRUS TYPE 44	0	1	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	3	0	0	0	0	3
0201	INFLUENZA A VIRUS	0	2	0	0	3	0	2	0	4	1	12
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	1	0	0	0	0	0	0	2	0	3
0203	INFLUENZA B VIRUS	0	1	0	0	1	0	1	0	0	0	3
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	1	1	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	3	0	1	4	4	0	1	2	0	15
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	1	0	0	2	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	1	2	1	2	0	3	0	1	0	10
0500	RHINOVIRUS (ALL TYPES)	0	4	0	0	1	4	2	0	9	0	20
0600	MYCOPLASMA PNEUMONIAE	0	1	0	0	10	4	2	1	1	0	19
0700	ORNITHOSIS-PSITTACOSIS	0	7	0	0	0	0	3	0	0	0	10
0816	COXSACKIEVIRUS A16	0	2	0	0	0	0	0	0	0	0	2
0902	COXSACKIEVIRUS B2	0	2	0	0	0	0	0	0	0	0	2
0903	COXSACKIEVIRUS B3	0	0	0	0	1	0	1	0	0	0	2
0904	COXSACKIEVIRUS B4	0	6	0	0	0	0	0	0	0	0	6
1025	ECHOVIRUS TYPE 25	0	0	0	0	0	0	1	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	1	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	1	0	2	1	0	0	4
1103	POLIOVIRUS TYPE 3	0	1	0	0	0	0	2	1	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	2	0	0	0	12	0	0	8	23
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	2	3	1	0	6	2	0	0	14
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	1	6	0	34	3	0	1	11	1	57
1303	VARICELLA-ZOSTER VIRUS	0	12	4	0	1	1	0	1	2	0	21
1306	HERPES SIMPLEX TYPE 1	0	42	46	1	37	2	2	0	41	0	171
1307	HERPES SIMPLEX TYPE 2	0	50	77	0	49	0	27	0	36	0	239
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	4	0	0	0	0	4
1401	COXIELLA BURNETII	0	0	0	0	1	0	5	0	7	1	14
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	3	1	1	0	0	0	7	0	12
1521	MEASLES VIRUS	0	8	0	0	1	3	3	0	0	1	16
1522	RUBELLA VIRUS	0	3	0	0	4	0	0	0	2	1	10
1532	HEPATITIS B ANTIGEN	0	15	14	0	18	0	31	2	39	13	132
1535	HEPATITIS A ANTIBODY	0	0	1	0	1	0	0	0	1	0	3
1536	HEPATITIS C VIRUS	0	0	11	0	0	0	0	0	0	0	11
1541	CHLAMYDIA A - C. TRACHOMATIS	16	0	44	1	34	0	24	1	12	3	135
1556	CMV - CYTOMEGALOVIRUS	0	26	2	6	5	0	8	1	25	2	75
1563	CORONAVIRUS	0	2	0	0	0	0	0	0	0	0	2
1564	ROTAVIRUS	0	1	2	2	12	4	35	5	0	0	61
1571	ENTEROVIRUS TYPE 71 (BCR)	0	2	0	0	0	0	0	0	0	0	2
1599	ENTEROVIRUS TYPING PENDING	0	1	0	0	0	6	0	1	0	0	8
9992	ROSS RIVER VIRUS	0	0	0	0	0	0	0	0	10	0	10
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	3	3	0	0	6
TOTAL		16	201	222	20	226	47	188	23	223	33	1199

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 22/11/90 TO 05/12/90

NSW: ICPMR; PHH/POW; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP.
VIC: FAIRFIELD; RCH; MDU, UNI MELB.
QLD: STATE LAB, BRIS; TOOWOOMBA PATH LAB; ROYAL BRIS HOSP; DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON.
WA: STATE LAB, PERTH; PMH.
SA: 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	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	3	7	9	10	2	0	31
0101	ADENOVIRUS TYPE 1	3	0	0	0	0	1	4
0102	ADENOVIRUS TYPE 2	1	1	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	6	2	0	0	0	1	9
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	1	0	0	0	0	0	1
0144	ADENOVIRUS TYPE 44	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	3	0	0	0	0	3
0201	INFLUENZA A VIRUS	2	2	4	0	3	1	12
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	1	2	0	0	0	3
0203	INFLUENZA B VIRUS	1	1	0	0	1	0	3
0302	PARAINFLUENZA VIRUS TYPE 2	0	1	0	0	1	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	1	7	2	1	4	0	15
0399	PARAINFLUENZA VIRUS TYPING PEN	0	1	2	0	0	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	3	1	1	3	2	0	10
0500	RHINOVIRUS (ALL TYPES)	2	8	9	0	1	0	20
0600	MYCOPLASMA PNEUMONIAE	3	5	1	0	10	0	19
0700	ORNITHOSIS-PSITTACOSIS	3	7	0	0	0	0	10
0816	COXSACKIEVIRUS A16	0	2	0	0	0	0	2
0902	COXSACKIEVIRUS B2	0	2	0	0	0	0	2
0903	COXSACKIEVIRUS B3	1	0	0	0	1	0	2
0904	COXSACKIEVIRUS B4	0	6	0	0	0	0	6
1025	ECHOVIRUS TYPE 25	1	0	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	1	0	1
1102	POLIOVIRUS TYPE 2	3	0	0	0	1	0	4
1103	POLIOVIRUS TYPE 3	3	1	0	0	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	12	1	0	2	0	8	23
1301	HERPES SIMPLEX VIRUS - NOT TYP	8	0	0	5	1	0	14
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	1	4	11	6	34	1	57
1303	VARICELLA-ZOSTER VIRUS	1	13	2	4	1	0	21
1306	HERPES SIMPLEX TYPE 1	2	44	41	47	37	0	171
1307	HERPES SIMPLEX TYPE 2	27	50	36	77	49	0	239
1399	HERPES VIRUS TYPING PENDING	0	4	0	0	0	0	4
1401	COXIELLA BURNETII	5	0	7	0	1	1	14
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	7	4	1	0	12
1521	MEASLES VIRUS	3	11	0	0	1	1	16
1522	RUBELLA VIRUS	0	3	2	0	4	1	10
1532	HEPATITIS B ANTIGEN	33	15	39	14	18	13	132
1535	HEPATITIS A ANTIBODY	0	0	1	1	1	0	3
1536	HEPATITIS C VIRUS	0	0	0	11	0	0	11
1541	CHLAMYDIA A - C. TRACHOMATIS	25	16	12	45	34	3	135
1556	CMV - CYTOMEGALOVIRUS	9	26	25	8	5	2	75
1563	CORONAVIRUS	0	2	0	0	0	0	2
1564	ROTAVIRUS	40	5	0	4	12	0	61
1571	ENTEROVIRUS TYPE 71 (BCR)	0	2	0	0	0	0	2
1599	ENTEROVIRUS TYPING PENDING	1	7	0	0	0	0	8
9992	ROSS RIVER VIRUS	0	0	10	0	0	0	10
9994	SMALL VIRUS (LIKE) PARTICLE	6	0	0	0	0	0	6
TOTAL		211	264	223	242	226	33	1199

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 22/11/90 TO 05/12/90

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

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	9	0	0	0	17	0	0	0	0	27
0101	ADENOVIRUS TYPE 1	2	0	0	0	0	0	0	0	0	0	2
0102	ADENOVIRUS TYPE 2	0	1	0	0	0	0	0	0	0	0	1
0103	ADENOVIRUS TYPE 3	1	1	0	0	0	5	0	0	0	0	7
0105	ADENOVIRUS TYPE 5	0	0	0	0	0	0	0	0	0	1	1
0144	ADENOVIRUS TYPE 44	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	3	0	0	0	0	0	0	0	0	3
0201	INFLUENZA A VIRUS	1	5	0	0	0	0	0	1	0	0	7
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	2	0	0	0	0	0	0	0	0	2
0203	INFLUENZA B VIRUS	1	1	0	0	0	0	0	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	14	0	0	0	0	0	0	0	0	14
0399	PARAINFLUENZA VIRUS TYPING PEN	0	3	0	0	0	0	0	0	0	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	2	7	0	0	0	0	0	0	0	0	9
0500	RHINOVIRUS (ALL TYPES)	0	19	0	0	0	1	0	0	0	0	20
0600	MYCOPLASMA PNEUMONIAE	3	14	0	0	0	0	0	0	0	0	17
0700	ORNITHOSIS-PSITTACOSIS	1	8	0	0	0	0	0	0	0	0	9
0816	COXSACKIEVIRUS A16	0	1	0	0	0	0	0	0	0	1	2
0902	COXSACKIEVIRUS B2	0	1	0	1	0	0	0	0	0	0	2
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	2	0	2	0	0	0	0	0	0	4
1025	ECHOVIRUS TYPE 25	0	0	0	0	0	1	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	2	0	0	0	0	3
1103	POLIOVIRUS TYPE 3	1	1	0	0	0	2	0	0	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	5	0	0	0	0	0	0	0	0	11	16
1301	HERPES SIMPLEX VIRUS - NOT TYP	3	1	1	0	0	0	0	0	0	5	10
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	10	0	0	0	0	1	2	0	0	0	13
1303	VARICELLA-ZOSTER VIRUS	0	0	1	0	0	0	0	0	0	19	20
1306	HERPES SIMPLEX TYPE 1	1	10	0	0	0	0	1	1	2	110	125
1307	HERPES SIMPLEX TYPE 2	1	0	0	0	0	0	0	0	0	117	118
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	3	3
1401	COXIELLA BURNETII	4	0	0	0	0	1	0	0	0	0	5
1502	PICORNIA VIRUS - NOT TYPED = E	1	6	0	0	1	4	0	0	0	0	12
1521	MEASLES VIRUS	3	0	1	0	0	0	0	0	0	10	14
1522	RUBELLA VIRUS	1	0	0	0	0	0	0	0	0	4	5
1532	HEPATITIS B ANTIGEN	59	0	0	0	0	0	54	0	0	0	113
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	3	0	0	0	3
1536	HEPATITIS C VIRUS	6	0	0	0	0	0	5	0	0	0	11
1541	CHLAMYDIA A - C. TRACHOMATIS	12	1	0	0	0	0	0	0	0	0	13
1556	CMV - CYTOMEGALOVIRUS	11	14	0	0	2	0	3	1	7	1	39
1563	CORONAVIRUS	0	0	0	0	0	2	0	0	0	0	2
1564	ROTAVIRUS	1	0	0	0	0	60	0	0	0	0	61
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	1	0	0	0	0	0	1	2
1599	ENTEROVIRUS TYPING PENDING	1	5	0	0	0	1	0	0	0	0	7
9992	ROSS RIVER VIRUS	6	0	0	0	0	0	0	0	0	0	6
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	6	0	0	0	0	6
TOTAL		138	134	3	4	3	104	68	3	9	283	749

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 22/11/90 TO 05/12/90

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	3	0	0	0	0	0	0	1	0	0	4
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	2	0	0	0	2
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	1	0	0	0	1
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	0	0	0	0	0	2
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	0	1	0	0	1
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	1	2	2	0	5
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	0	0	0	0	1	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
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	0	1	0	1
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	1	0	1	0	2
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	0	1	0	0	1
0903	COXSACKIEVIRUS B3	0	0	0	0	0	0	0	0	0	1	1
0904	COXSACKIEVIRUS B4	0	0	0	0	0	0	0	2	0	0	2
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	1	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	4	0	0	0	0	0	1	2	0	7
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	2	0	0	0	0	0	1	1	0	4
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	34	1	0	0	0	3	6	0	44
1303	VARICELLA-ZOSTER VIRUS	1	0	0	0	0	0	0	0	0	0	1
1306	HERPES SIMPLEX TYPE 1	5	34	0	0	0	0	0	1	6	0	46
1307	HERPES SIMPLEX TYPE 2	0	121	0	0	0	0	0	0	0	0	121
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	0	1
1401	COXIELLA BURNETII	0	0	0	0	0	0	2	6	1	0	9
1521	MEASLES VIRUS	0	0	1	0	0	0	0	0	1	0	2
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	1	4	0	5
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	19	0	19
1541	CHLAMYDIA A - C. TRACHOMATIS	1	120	0	0	0	0	0	0	1	0	122
1556	CMV - CYTOMEGALOVIRUS	0	4	1	2	0	1	3	7	18	0	36
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	0	1
9992	ROSS RIVER VIRUS	0	0	0	0	1	0	1	1	1	0	4
TOTAL		14	285	36	3	1	2	11	33	64	1	450

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

NON-VIRAL PATHOGEN IDENTIFICATIONS CATEGORISED BY SOURCE SPECIMENS

SAMPLE COLLECTION DATE: JULY TO SEPTEMBER 1990

BL - WHOLE BLOOD; BR - BRONCHIAL WASHINGS OR ASPIRATE; CS - CEREBROSPINAL FLUID;
EY - EYE; FA - FAECES/RECTUM; GE - GENITAL SWAB; LE - LEUCOCYTES;
NA - NASOPHARYNGEAL SWAB; PD - PERITONEAL DIALYSIS FLUID; PF - PERICARDIAL,
PLEURAL OR JOINT FLUID; PU - PUS; SA - SALIVA; SK - SKIN; SM - SERUM;
SP - SPUTUM; SS - SKIN SCRAPINGS; TH - THROAT; UR - URINE;

POSTMORTEM OR BIOPSY SPECIMENS: MB - BLOOD, BONE MARROW; MD - DIGESTIVE TRACT;
MH - HEART; MK - KIDNEY; ML - LIVER; MN - BRAIN, SPINAL CORD; MP - LUNGS;
MR - RESPIRATORY TRACT; MS - SPLEEN, LYMPH NODES; MO - OTHER POSTMORTEM/BIOPSY
SPECIMEN

		BL	CS	FA	GE	LE	NA	PF	PU	SK	SM	SP	SS	TH	UR	OT	MD	MO	TOTAL
AE00	AEROMONAS SPECIES	0	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3
AS00	ASPERGILLUS SPECIES	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
BO01	BORDETELLA PERTUSSIS	2	0	0	0	0	0	0	0	0	31	0	0	0	0	0	0	0	33
BR00	BRUCELLA SPECIES	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
BR01	BRUCELLA ABORTUS	0	0	0	0	0	0	0	0	0	6	0	0	0	0	0	0	0	6
BT00	BACTEROIDES SPECIES	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
CA00	CANDIDA SPECIES	0	0	1	118	4	1	0	0	5	0	1	0	5	2	0	0	0	137
CL00	CLOSTRIDIUM SPECIES	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1
CM00	CAMPYLOBACTER SPECIES	0	0	12	0	0	0	0	0	0	0	0	0	0	0	0	0	0	12
CM01	CAMPYLOBACTER JEJUNI	0	0	44	0	0	0	0	0	0	0	0	0	0	0	0	0	0	44
CR00	CRYPTOCOCCUS SPECIES	0	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	3
CT00	CRYPTOSPORIDIUM SPECIES	0	0	13	0	0	0	0	0	0	0	0	0	0	0	0	0	0	13
DI01	DIPHYLLOBOTHRIUM LATUM	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
EA01	ENTAMOEBA HISTOLYTICA	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
EC01	ECHINOCOCCUS GRANULOSUS	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
EN00	ENTEROBACTER SPECIES	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2
EP00	EPIDERMIDOPHYTON SPECIES	0	0	0	0	0	0	0	0	3	0	0	2	0	0	1	0	0	6
ES01	ESCHERICHIA COLI	6	0	6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	12
ET01	ENTEROBIUS VERMICULARIS	0	0	1	0	0	0	0	0	0	0	0	0	0	1	0	1	0	3
GI01	GIARDIA LAMBLIA	0	0	37	0	0	0	0	0	0	0	0	0	0	1	0	0	0	38
HM02	HAEMOPHILUS INFLUENZAE	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
KL00	KLEBSIELLA SPECIES	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
LE01	LEGIONELLA PNEUMOPHILA	0	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	3
LS00	LEPTOSPIRA SPECIES	0	0	0	0	0	0	0	0	0	5	0	0	0	0	0	0	0	5
LS03	LEPTOSPIRA ICTEROHAEMORRHAGIAE	0	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	3
LS04	LEPTOSPIRA POMONA	0	0	0	0	0	0	0	0	0	14	0	0	0	0	0	0	0	14
LS05	LEPTOSPIRA AUTUMNALIS	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
LS07	LEPTOSPIRA HARDJO	0	0	0	0	0	0	0	0	0	8	0	0	0	0	0	0	0	8
LS10	LEPTOSPIRA AUSTRALIS	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
MI00	MICROSPORUM SPECIES	0	0	0	0	0	0	0	0	0	0	0	13	0	0	0	0	0	13
NE01	NEISSERIA GONORRHOEAE	0	0	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	4
NE02	NEISSERIA MENINGITIDIS	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
NOTL	NOT LISTED	0	0	3	0	0	0	0	0	0	0	0	3	2	0	1	0	1	10
PA01	PASTEURELLA MULTOCIDA	0	0	0	0	0	0	0	4	2	0	0	0	0	0	0	0	0	6
PL00	PLASMODIUM SPECIES	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4
PL01	PLASMODIUM FALCIPARUM	30	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	30
PL02	PLASMODIUM VIVAX	48	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	48
PS00	PSEUDOMONAS SPECIES	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
SA00	STAPHYLOCOCCUS SPECIES	7	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	9
SA01	STAPHYLOCOCCUS AUREUS	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
SE00	STREPTOCOCCUS SPECIES	9	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	10
SE01	STREPTOCOCCUS PNEUMONIAE	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2
SH04	SHIGELLA SONNEI	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
SL00	SALMONELLA SPECIES	1	0	7	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8
TC01	TRICHOMONAS VAGINALE	0	0	0	12	0	0	0	0	0	0	0	0	0	1	1	0	0	14
TI00	TRICHOPHYTON SPECIES	0	0	0	0	0	0	0	0	8	0	0	17	0	0	0	0	0	25
TP01	TOXOPLASMA GONDI	0	1	0	0	0	0	0	0	0	21	0	0	0	0	0	0	0	22
TR01	TREPONEMA PALLIDUM	0	1	0	0	0	0	0	0	0	95	0	0	0	0	0	0	0	96
YE01	YERSINIA ENTEROCOLITICA	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2
TOTAL		116	4	131	135	4	1	1	4	19	194	1	35	7	5	4	1	1	663

NB: NUMBERS MAY CHANGE AT A LATER DATE AS A RESULT OF LATE REPORTING

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

NON-VIRAL PATHOGEN IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

SAMPLE COLLECTION DATE: JULY TO SEPTEMBER 1990

CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 400 - DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON (QLD)
CODE HOB - HOBART PATHOLOGY LABORATORY (TAS)
CODE LDS - DIAGNOSTIC SERVICES LTD, LAUNCESTON (TAS)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

		019	112	115	400	HOB	LDS	RHH	TPL	TOTAL
AE00	AEROMONAS SPECIES	0	0	0	3	0	0	0	0	3
AS00	ASPERGILLUS SPECIES	0	0	0	0	1	0	0	0	1
BO01	BORDETELLA PERTUSSIS	0	0	0	21	0	0	0	12	33
BR00	BRUCELLA SPECIES	0	0	0	1	0	0	0	0	1
BR01	BRUCELLA ABORTUS	0	0	3	3	0	0	0	0	6
BT00	BACTEROIDES SPECIES	0	0	0	0	0	0	0	1	1
CA00	CANDIDA SPECIES	0	0	134	0	3	0	0	0	137
CL00	CLOSTRIDIUM SPECIES	0	0	0	1	0	0	0	0	1
CM00	CAMPYLOBACTER SPECIES	0	0	0	8	4	0	0	0	12
CM01	CAMPYLOBACTER JEJUNI	0	0	0	0	0	30	0	14	44
CR00	CRYPTOCOCCUS SPECIES	0	1	2	0	0	0	0	0	3
CT00	CRYPTOSPORIDIUM SPECIES	0	0	0	1	0	2	0	10	13
DI01	DIPHYLLOBOTHRIUM LATUM	0	0	0	1	0	0	0	0	1
EA01	ENTAMOEBA HISTOLYTICA	0	0	0	1	0	0	0	0	1
EC01	ECHINOCOCCUS GRANULOSUS	0	0	1	0	0	0	0	0	1
EN00	ENTEROBACTER SPECIES	0	0	0	0	0	0	0	2	2
EP00	EPIDERMIDOPHYTON SPECIES	0	0	0	2	3	0	0	1	6
ES01	ESCHERICHIA COLI	0	0	0	7	0	0	0	5	12
ET01	ENTEROBIUS VERMICULARIS	0	0	0	2	0	0	0	1	3
GI01	GIARDIA LAMBLIA	0	0	0	17	2	2	0	17	38
HM02	HAEMOPHILUS INFLUENZAE	0	0	0	0	0	0	0	1	1
KL00	KLEBSIELLA SPECIES	0	0	0	0	0	0	0	1	1
LE01	LEGIONELLA PNEUMOPHILA	0	0	3	0	0	0	0	0	3
LS00	LEPTOSPIRA SPECIES	0	0	5	0	0	0	0	0	5
LS03	LEPTOSPIRA ICTEROHAEMORRHAGIAE	0	0	3	0	0	0	0	0	3
LS04	LEPTOSPIRA POMONA	0	2	11	1	0	0	0	0	14
LS05	LEPTOSPIRA AUTUMNALIS	0	0	1	0	0	0	0	0	1
LS07	LEPTOSPIRA HARDJO	0	3	5	0	0	0	0	0	8
LS10	LEPTOSPIRA AUSTRALIS	0	0	1	0	0	0	0	0	1
MI00	MICROSPORUM SPECIES	0	0	0	12	0	0	0	1	13
NE01	NEISSERIA GONORRHOEAE	0	0	0	0	1	0	0	3	4
NE02	NEISSERIA MENINGITIDIS	0	0	0	0	0	0	0	1	1
NOTL	NOT LISTED	0	0	0	10	0	0	0	0	10
PA01	PASTEURELLA MULTOCIDA	0	0	0	2	4	0	0	0	6
PL00	PLASMODIUM SPECIES	0	0	4	0	0	0	0	0	4
PL01	PLASMODIUM FALCIPARUM	0	0	30	0	0	0	0	0	30
PL02	PLASMODIUM VIVAX	0	0	48	0	0	0	0	0	48
PS00	PSEUDOMONAS SPECIES	0	0	0	0	0	0	0	1	1
SA00	STAPHYLOCOCCUS SPECIES	0	0	0	2	4	0	0	3	9
SA01	STAPHYLOCOCCUS AUREUS	0	0	0	0	0	0	0	1	1
SE00	STREPTOCOCCUS SPECIES	0	0	0	3	0	0	0	7	10
SE01	STREPTOCOCCUS PNEUMONIAE	0	0	0	0	0	0	0	2	2
SH04	SHIGELLA SONNEI (A	0	0	0	0	0	0	0	1	1
SL00	SALMONELLA SPECIES	0	0	0	3	0	0	0	5	8
TC01	TRICHOMONAS VAGINALE	0	0	6	3	3	0	0	2	14
TI00	TRICHOPHYTON SPECIES	0	0	0	16	8	0	0	1	25
TP01	TOXOPLASMA GONDI	6	5	8	2	0	0	1	0	22
TR01	TREPONEMA PALLIDUM	0	0	88	4	0	0	0	4	96
YE01	YERSINIA ENTEROCOLITICA	0	0	0	1	0	0	0	1	2
TOTAL		6	11	353	127	33	34	1	98	663

NB: NUMBERS MAY CHANGE AT A LATER DATE AS A RESULT OF LATE REPORTING