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Communicable Diseases Intelligence

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Contents:

Editor Dr I.F. Cook

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1 464 reports were processed during this period.

One case of Q fever was reported in a 23 year old male from New South Wales. No occupational details were available.

Two females 6 and 8 years had influenza A subtype H1N1 complicated by haemorrhagic gastritis. The 6 year old child died.

Cytomegalovirus was isolated from the blood of a 31 year old male.

OVERSEAS BRIEF: *SHIGELLA DYSENTERIAE* TYPE 1 IN TOURISTS VISITING MEXICO

(Based on MMWR 37:465)

CDC, Atlanta, has received 17 reports of *Shigella dysenteriae* type 1 diarrhoea in travellers to Mexico (mostly Cancun). There were no common exposures in hotels or restaurants. Thirteen (76%) of the patients required hospitalisation; two patients developed haemolytic-uremic syndrome. Six isolates

tested thus far at CDC were resistant to chloramphenicol and tetracycline; two isolates were also resistant to ampicillin and trimethoprim-sulfamethoxazole. An epidemiological and laboratory investigation is under way in Mexico.

CDC, Atlanta, recommends that physicians should:

- consider this diagnosis in persons with severe or bloody diarrhoeal illness who have recently returned from Mexico;
- obtain appropriate cultures from such patients; and
- report suspected cases of *S dysenteriae* to public health authorities.

CONVULSIONS ASSOCIATED WITH PROPHYLACTIC ANTIMALARIAL DRUGS: IMPLICATIONS FOR PEOPLE WITH EPILEPSY

A recent article in the *British Medical Journal* describes case reports of tonic-clonic convulsions in four epileptic women on prophylactic antimalarial drugs (chloroquine alone, and in association with dapsone-pyrimethamine or sulphadoxine-pyramethamine)[1].

The authors of this study conclude from the patients' histories, that the associations between the tonic-clonic seizures and the malaria prophylaxis are unlikely to be due to chance. They also reference other instances of convulsions associated with antimalarial drugs[2,3,4] although not at prophylactic doses. Consequently, the authors recommend:

> 'that specific inquiry should be made for a history of epilepsy when considering malaria prophylaxis and that people with epilepsy should be advised about the risk of antimalarial drugs provoking seizures. In view of the risk and the serious consequences if tonic-clonic seizures are provoked, some people with epilepsy may prefer to plan their itinerary so that they avoid the need to take antimalarial drugs'.

REFERENCES

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CLINICAL COMPLICATIONS OF *P. FALCIPARUM* INFECTION

(Contributed by Dr J. Hanna, Communicable Diseases Control Centre, and Dr N. Rajabalendran, Alice Springs Hospital, Alice Springs, N.T.)

Ten days following his return to Australia from a one-week visit to Timor (Indonesia), a 31 year old male resident of the Northern Territory presented to a medical practitioner with persistent fever, nausea, lethargy and myalgia. The patient had not taken malaria chemoprophylaxis during his overseas visit.

His full blood count and film showed:

. mild leukopaenia (3.1 x 10^9/L);
. mild thrombocytopaenia (94 x 10^9/L); and
. malarial parasites identified as *P vivax*.

Chloroquine was prescribed. However, due to severe emesis, he was admitted several hours later to Alice Springs Hospital. A blood film taken on admission and processed by a different laboratory identified *P falciparum* malarial parasites.

As there was further deterioration he was transferred to intensive care. Oral quinine therapy was initiated as no parenteral quinine was available. Seven hours after admission to ICU, the patient developed oliguria with black coloured urine. Intravenous quinine was commenced some twelve hours after *P falciparum* malaria was diagnosed. Approximately two and a half days had elapsed between the time of his initial presentation and this definitive treatment.

For the next 24 hours the patient remained critically ill with:

. anaemia (Hb 83 g/L);
. thrombocytopaenia (platelet count 23 x 10^9);
. hypoalbuminaemia (22g/L);
. hypocalcaemia (1.64 mmol/L); and
. hypophosphataemia (0.3 mmol/L),

which necessitated infusion of packed cells and albumin, calcium replacement and phosphate therapy.

Over the next 36 hours there was a gradual improvement with return of normal urine output and mental state and the cessation of vomiting. Therapy was then changed to oral quinine and Fansidar. The patient was discharged after 9 days of hospitalisation.

CDI Editorial Comment

This report summarises the management of a *P falciparum* malarial infection complicated by a blackwater fever-like syndrome.

The pathogenesis of blackwater fever is unknown. It is generally considered that following repeated attacks of malaria and the consequent destruction of red cells, an autoimmune factor is formed which induces haemolysis. It has also been suggested that the presence of the malarial parasite inside the erythrocyte changes the antigenic structure of this cell stimulating the production of autoantibodies. These, in the presence of complement can lead to haemolysis. It should be noted however that autoantibodies are rarely found in patients with blackwater fever. The predisposing factor for blackwater fever is repeated infection with *P falciparum*. Precipitating factors include:

. cold exposure;
. excessive physical exertion;
. over indulgence in alcohol; and
. irregular therapy and prophylaxis with antimalarial drugs, particularly quinine.

The syndrome is charactarised clinically by the abrupt onset of headache, fever, nausea, vomiting of bile-stained fluid, severe loin pains and marked prostration. The urine is typically dark red to almost black, with the colour depending on the proportions of oxyhaemoglobin and methaemoglobin. Albumin is present in high concentrations in the urine throughout the lytic period.

The appearance of haemoglobinuria associated with febrile paroxysm in a patient with likely exposure to *P falciparum* malaria is highly suggestive of blackwater fever. Other causes of haemoglobinuria are:

. quinine in susceptible individuals;
. 8-aminoquinolines (primaquine) and sulfones (dapsone) in persons with glucose-6-phosphate dehydrogenase deficiency
. paroxymal haematuria; and
. favism.

These can usually be excluded by careful consideration of the patient's history and clinical condition.

The essential feature of blackwater fever management is cardiovascular support, including intravenous fluids, absolute rest and careful nursing. Blood transfusion is often required and corticosteriods may sometimes help to contain haemolysis. Renal failure may necessitate renal dialysis. Antimalarial medications are rarely required during the haemolytic crisis and quinine should preferably be avoided. The mortality from blackwater fever is up to 40%, with most fatalities resulting from renal failure.

Q FEVER 1979-1987 IN NOVA SCOTIA AND ONTARIO

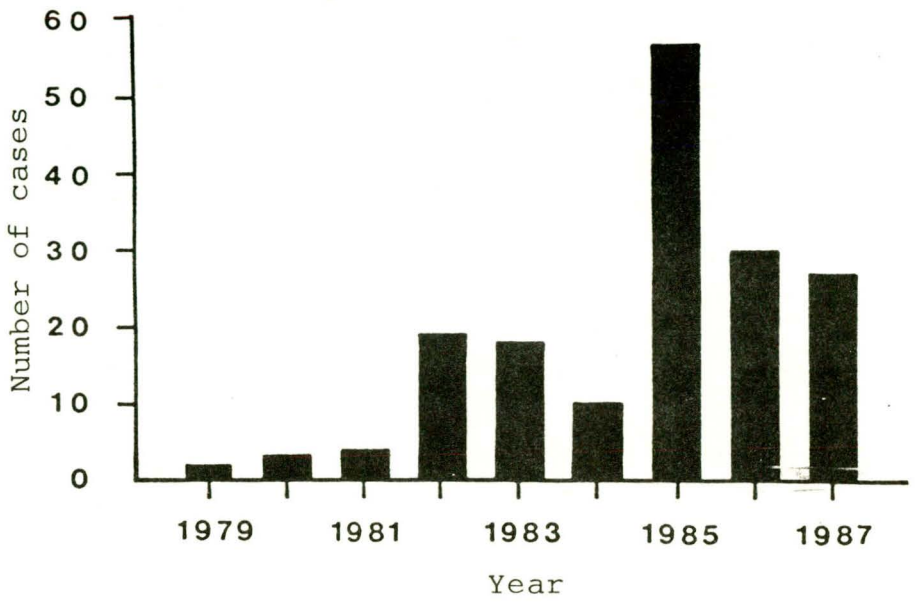
(Based on CDWR Vol. 14-17, 30 April 1988)

NOVA SCOTIA:

Q fever was not recognised in Nova Scotia until 1979 when, during the study of atypical pneumonia, cases of *Coxiella burnetii* infection were diagnosed, and every year since then, cases have occurred (Figure 1). The peak was reached in 1985; 57 cases were diagnosed that year. Between 1979 and 1987, a total of 170 cases, 115 male and 55 female, occurred. The mean age of these cases was 39.6 $\pm$ 16.4 years with a range from 12-89 years. Eight of the patients have been over 70 years of age. One hundred and forty cases or 82% of the total have been hospitalised with atypical pneumonia. There has been one death in a patient with an underlying cardiomyopathy who developed an aspiration pneumonia and *Escherichia coli* bacteraemia. At the time of admission this patient was in shock with renal failure and rhabdomyolysis.

There seems to be unique features to the epidemiology of Q fever in Nova Scotia in that exposure to infected parturient cats has resulted in a number of outbreaks in this province [1,2] Indeed, from 1979 to 1987 there have been 24 separate cat-related incidents of Q fever in the province.

Figure 1: Q fever in Nova Scotia, 1979-1987



Eight cases of Q fever endocarditis, 6 in Nova Scotia and 1 each in Prince Edward Island and New Brunswick, have also been diagnosed.
These data indicate that Q fever is common in Nova Scotia and other studies are indicated to determine the prevalence of this infection.

ONTARIO:

After many years with no reports of Q fever in Ontario, 22 human cases were recorded in 1982, and between that year and 1986, a total of 89 cases were reported in this province. However, since 1982 the annual number of cases reported has gradually been declining, from 22 in 1982 to 12 in 1986. The high incidence that occurred in 1982 was related to a number of cases associated with a cattle barn and an outbreak among research staff and animal handlers in the animal research area of a Toronto hospital. In the latter outbreak, serological surveys revealed that 97% (36/37) of the sheep, 68% (28/41) of the animal attendants, and 18% (60/331) of the staff members were seropositive for Q fever.

The most commonly reported symptom was 'fever of unknown origin', 52% (46/89), followed by weakness or fatigue, 25% (22/89); upper respiratory tract infection, 22% (20/89); headache, 21% (19/89); myalgia, 11% (10/89); nausea, 9% (8/89); abdominal pain, 8% (7/89); and vomiting, 4% (4/89). All 89 cases exhibited at least one of these symptoms. A significant proportion (33%) required hospitalisation.

The majority of cases occurred in men, with the incidence increasing from 21 to 60 years of age. Cases occurring in women were substantially fewer except in those older than 60 years, which may be due to a greater likelihood of occupational exposure to infected animals.

The highest incidence occurred during the spring and early summer, i.e., April to June, with a subsequent peak in December. This may be related to seasonal parturition of sheep, goats and cows, or possibly livestock marketing practices.

Serology was done on all 89 cases; paired sera were available for 50. Thirty-one exhibited a 4-fold or greater increase in titre using the complement fixation test (CFT); this is indicative of recent infection. A standing or single CFT titre of 1:256 or greater was obtained in 20 of 39 cases. Such a finding is considered presumptive evidence of infection at some undetermined time.

The extent of direct and indirect animal contact was also investigated for the 89 cases. Fifty-two percent (46/89) indicated that they had had no animal contact; these patients were mainly urban homemakers, children, pensioners, or unemployed persons. Where contact had occurred, 74% (32/43) had had direct animal contact, with sheep (12) and goats (10) ranking first and second. Direct animal contact was also related to ingestion of unpasteurised dairy products such as bovine milk (5%), caprine milk (5%) and cheese (5%).

Of the 45 cases indicating occupation, 76% (34/45) had close contact with animals. Livestock owners (18) and animal research workers (8) were the occupational groups most commonly affected.

Although the trend for Q fever is decreasing, there is still concern because of the severity of the symptoms, the relatively high rate of hospitalisation (29/89 or 33%), and the number of occupational cases associated with research animals and livestock (27/89 or 30%).

Because the incidence of human cases related to contact with either bovines or their products (7/89 or 8%) during the past 5 years (1982-86) is relatively low, a *'Coxiella-free'* herd-testing program in Ontario would not appear to be warranted. The species at greatest risk of spreading the disease may be sheep and goats. However, to date, no attempt has been made to relate animal contacts to a species-specific population base nor have there been any records of animal tests related to reported human cases.

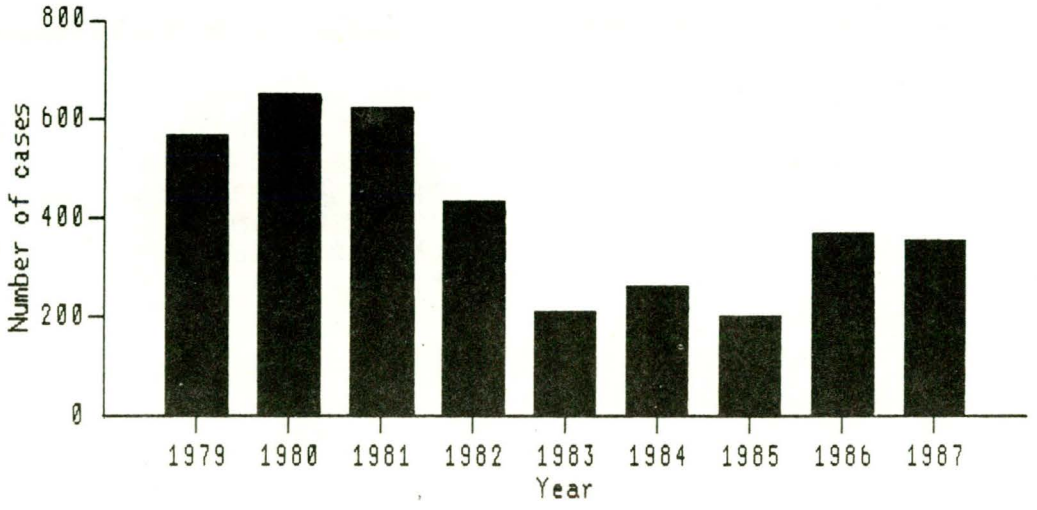
CDI Editorial Comment:

From 1979 to 1987, 3358 cases of Q fever were reported to the CDI virus reporting scheme. Figure 2 shows the distribution of cases by year.

Q fever is a zoonoses in Australia and remains an occupational exposure disease. Abbatoir workers, farmers, veterinarians, animal laboratory handlers (particularly those working with pregnant sheep, cattle and goats) and laboratory workers preparing Q fever diagnostic antigen, are at greatest risk.

Coxiella burnetii the rickettsia that causes Q fever is relatively common in cattle, sheep and goats in which it produces a mild or subclinical infection. The placental tissues of these animals may contain up to 10^9 infectious particles per gram of tissue. Infected placental tissues and

Figure 2: Q fever in Australia, 1979-1987



birth fluids are therefore a considerable health hazard. *C burnetii* may also be excreted through milk and faeces. Inhalation of contaminated dust and of droplets from infected animal tissues is the main source of human infection [3]. Recently, successful trials using a purified formalin-inactivated *C burnetii* phase 1 vaccine of the Henzerling strain, have been carried out in South Australian abbatoir workers. The vaccine was found to be safe and highly effective with minimal reactogenicity in non-immune subjects [4]. This vaccine is now pending approval by the Australian Drug Evaluation Committee.

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HEPATITIS B IMMUNOGLOBULINS AND HIV ANTIBODIES

(Based on CDWR Vol. 14-24, 18 June 1988)

In December 1987, a 3-week-old Ontario girl scheduled to be placed in foster care was tested for HIV antibody because her mother belonged to a high-risk group for AIDS. She was found to be HIV antibody reactive by both ELISA and Western blot tests. A repeat specimen was also reactive. The mother had been seronegative on 2 occasions, during the pregnancy (August 1987) and after the birth of the child (December 1987).

There was serious concern about the source of the child's infection, and after a lengthy investigation it was determined that the infant had received hepatitis B immunoglobulin (HBIG) 24 hours after birth. The lot of the HBIG that had most likely been administered (Cutter 16B06) was located and found to be strongly reactive for HIV antibodies by Western blot. A serum

sample collected from the infant immediately after birth was tested and proved to be HIV seronegative by both ELISA and Western blot. The HIV antibodies in this infant are now presumed to have been passively acquired. They have declined significantly with time, and as of 28 March, 15 weeks after HBIG injection, the child is negative by ELISA, although traces of antibodies to p24, 33, 41 and 63 still persist by Western blot.

Since this case was recognised, 3 more infants have been found to be seropositive for HIV. Their mothers also belonged to a known high-risk group. Two of the 3 infants have HIV seronegative mothers. Serum from the mother of the third child is not available for testing. All 3 babies had received HBIG from the same lot (16B06) shortly after birth.

In another episode, a health-care worker involved in a needle-stick incident was found to be HIV antibody reactive. Upon inquiry, it was determined that HBIG had been administered 5 days before collection of the blood specimen. A serum sample taken at the time of the accident and prior to the administration of HBIG was tested and found to be HIV seronegative. The lot number of HBIG administered is not known, but 3 out of the 5 lots available in the hospital at that time were found to be HIV seropositive (16B06, 16B08B and (16C01B), one indeterminate (16C03), and one negative (16C04DB). The HIV antibodies in this individual are declining with time as evidenced by decreasing optical density ELISA values and a decrease in the intensity of the bands visible on the Western blot.

MMWR Editorial Comment:

It is important to note that in the 5 cases discussed above, none of the recipients of HBIG were actually at risk of HIV infection. Nevertheless, there is still cause for concern, because a great deal of unnecessary mental anguish would result if an individual were found to be seropositive for HIV, implying the presence of HIV infection, when in fact no such infection had actually occurred. Such was the case with the health-care worker and 4 infants discussed above.

HIV seropositivity, due to passive immunisation, can be present up to 6 months after the actual administration of HBIG containing HIV antibodies. Thus, in an individual who tests positive for HIV antibodies and for whom there appears to be no known risk factors for HIV infection, the physician should ask if this individual received HBIG at any time during the previous 6 months. If a history of recent HBIG administration is present, then the individual should be retested for HIV antibodies in 3 months to determine whether or not the titre has decreased. Again it should be emphasised that the presence of HIV antibodies in specimen of HBIG does not represent a risk of HIV infection.

Statement

The Canadian National Advisory Committee on Immunisation (NACI) in its 'Statement on Safety of Immune Globulin Preparations' indicated that human immunoglobulin preparations are among the safest biological products available. Plasma found positive for hepatitis B surface antigen[1] or for antibody to the human immunodeficiency virus (HIV)[2] is excluded from donor pools.

This exclusion policy and the use of the Cohn-Oncley cold ethanol procedure to extract immunoglobulins ensure that preparations carry no risk of transmitting these viruses. This fractionation procedure has been shown to remove HIV by physical partition and chemical inactivation[2]. Finally, epidemiological studies have failed to find any evidence of HIV transmission following administration of immunoglobulin preparations.

However, certain lots of various immunoglobulin products including hepatitis B immunoglobulin and varicella-zoster immunoglobulin were prepared before implementation of new regulations in 1985 which required exclusion of anti-HIV positive plasmas from donor pools. Administration of lots containing anti-HIV antibody may give risk to transiently positive anti-HIV tests due to passive transfer of antibody[3] which may be detectable for up to 6 months. **These products, however, carry no risk of transmission of HIV.**

Transmission of non-A non-B hepatitis by administration of intravenous immunoglobulin (IVIG) to agammaglobulinemic patients has been reported in 3 instances[3]. All cases were detected during clinical trails using experimental preparations of IVIG. No case of non-A non-B hepatitis has been reported in North America following administration of presently licensed products.

CDI Editorial Comment

HBIG administered in Australia is manufactured by the Commonwealth Serum Laboratories. Since May 1985, all plasma for use in the production of immunoglobulins has been screened for HIV antibody (as individual donations and as pooled plasma). There are no remaining stocks of immunoglobulins produced before the introduction of screening.

Imported therapeutic goods for administration to humans must be screened for HIV antibody and hepatitis B surface antigen (HBsAg). Evidence must be submitted that both individual donors and the final bulk product have been tested for HIV antibody and HBsAg and found negative.

REFERENCES

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THE AUSTRALIAN NATIONAL WORKSHOP ON AIDS EPIDEMIOLOGY

The National Workshop on AIDS Epidemiology was conducted in July 1988 by the Australian Department of Community Services and Health with the concurrence of the Intergovernmental Committee on AIDS (IGCA) and the collaboration of the National Health and Medical Research Council, Special Unit on AIDS Epidemiology and Clinical Research. The workshop reviewed the optimal national HIV data requirements, the current methods of their collection, including associated problems encountered throughout Australia, and has suggested ways and means for their improvement.

The Workshop has recommended that:-

1. the level of funding provided to individual States for the treatment of HIV infections under the Medicare agreement be dependant upon the number of live Category A AIDS cases notified to the NHMRC Special Unit by mid November of the previous year and not on the number of Group IV HIV infections.

2. future formulae for the disbursement of Commonwealth AIDS funds to the States and Territories should include a proviso that a certain proportion of those funds be spent on providing staff to perform data collection and collation functions.

3. second blood samples be taken from those patients confirmed as HIV antibody positive after supplemental testings, so that retesting could be done, if necessary, in order to rule out errors in identification.

4. HIV prevalence data on ethnic populations be obtained through testing of clients at special settings like STD clinics and prisons and not specifically through ethnicity directed research projects.

5. a) a national HIV surveillance system based on data compiled from tests performed by authorised laboratories be instituted.

   b) individual States and Territories would be responsible for devising their own methods of identification to overcome the problems of double counting and ensuring individual confidentiality.

   c) States and Territories would provide aggregated data on individuals (not tests) to the NHMRC Special Unit at regular intervals. The data provided would include the following variables for each individual:

      1. Gender
      2. Age
      3. Postcode of residence
      4. Risk activity/Reason for test
      5. Result of test

      These data would be provided on both positive and negative individuals.

   d) a suitable format for the regular returns of the above aggregated data be designed and negotiated between the States and Territories and the NHMRC Unit on AIDS Epidemiology & Clinical Research.

6. a full public debate on a specific proposal for HIV testing, indicating the specific group, justifications and approach, should be conducted prior to any decision whether to institute studies involving anonymous testing or not.

AIDS UPDATE - CANADA

(Based on an update report from the Federal Centre for AIDS, Ottawa, Canada, 2 August 1988)

To 2 August 1988, 1896 cases (1862 adults and 34 paediatric) of AIDS which meet the surveillance case definition for AIDS (revised 1 September 1987) have been reported to the Federal Centre for AIDS, Ottawa. The distribution of those patients by Province of notification (Table 1), by age group (Table 2), by risk category (Table 3) and by primary diagnosis (Table 4) are shown below:

Table 1: AIDS cases by Province of notification

PROVINCE	CASES	(%)	DEATHS
British Columbia	382	(20.1)	193
Alberta	110	(5.8)	67
Saskatchewan	23	(1.2)	16
Manitoba	26	(1.4)	14
Ontario	737	(38.9)	449
Quebec	568	(30.0)	284
New Brunswick	8	(0.4)	6
Nova Scotia	30	(1.6)	12
Prince Edward Island	2	(0.1)	1
Newfoundland	8	(0.4)	6
North West Territories	1	(0.1)	0
Yukon	1	(0.1)	0
TOTAL	1896	(100.0)	1048

Table 2: AIDS cases by sex and age groups

AGE (YEARS)	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
0-14	14	20	34	10	11	21
15-19	4	0	4	4	0	4
20-29	348	32	380	181	16	197
30-39	826	32	858	439	21	460
40-49	409	9	418	231	6	237
50+	175	25	200	113	16	129
Unknown	2	0	2	0	0	0
TOTAL	1778	118	1896	978	70	1048

Table 3: AIDS by risk category

RISK GROUP	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
ADULTS						
Homo-/bisexual	1529		1529	817		817
IV drug user	11	2	13	6	2	8
Homo-/bisexual IV Drug User	45		45	28		28
Blood/blood products recipient	58	24	82	42	16	58
Heterosexual activity**	78	65	143	48	36	84
None of the above	43	7	50	27	5	32
Total	1764	98	1862	968	59	1027
PAEDIATRIC						
Perinatal transmission	12	17	29	*	*	*
Blood/blood products recipient	2	3	5	*	*	*
Total	14	20	34	10	11	21

* Death breakdown not available.

** Heterosexual activity includes (a) persons originating in or residing in countries with a high prevalence of HIV and where heterosexual transmission of HIV is common; and (b) persons reporting heterosexual activity with person(s) at risk of HIV infection.

Table 4: AIDS by primary diagnosis

PRIMARY DIAGNOSIS	CASES	DEATHS
ADULTS		
Kaposi's Sarcoma (KS)	372	190
Pneumocystis carinii Pneumonia (PCP)	1023	560
KS and PCP	45	30
Other opportunistic infection	335	200
Other malignancies	57	32
HIV Wasting Syndrome	20	9
HIV Encephalopathy	10	6
Total	1862	1027
PAEDIATRIC		
PCP	10	8
Lymphoid interstitial pneumonitis	6	3
Cytomegalovirus	6	5
Other opportunistic infection	12	5
Total	34	21

IMPORTATION OF BIOLOGICAL MATERIALS

Quarantine restrictions forbid the importation into Australia of biological materials without the authority of a Permit to Import Biological Material (Form Q41) issued by the Department of Primary Industries and Energy. Material brought into Australia not covered by a Q41 may by subject to destruction, and heavy penalties may be incurred by the importer. A Permit to Import may be obtained by lodging an application to Import Biological Materials (Form Q39) with the Department of Primary Industries and Energy, Edmund Barton Building, BARTON ACT 2600. Approval should not be anticipated.

In addition, all materials of human origin must be screened for and certified free of Hepatitis B Surface Antigen and Human Immunodeficiency Virus, prior to the material arriving in Australia. Exemption from screening requirements may be given in special circumstances. Enquiries should be directed to Human Quarantine, Communicable Diseases Section on telephone (062) 89 7793 or facsimile (062) 89 6963.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

PERIOD 24-8-88 TO 7-9-88

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	3	20	0	0	0	0	13	0	0	1	0	37
0101	ADENOVIRUS TYPE 1	1	1	0	1	0	0	2	0	0	0	0	5
0102	ADENOVIRUS TYPE 2	1	4	0	0	0	0	1	0	0	0	0	6
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	1	0	0	0	0	2	0	1	0	0	4
0201	INFLUENZA A VIRUS	7	81	1	0	0	1	1	0	0	0	0	91
0202	INFLUENZA A VIRUS SUBTYPE H3N2	1	7	0	0	0	0	0	0	0	0	0	8
0203	INFLUENZA B VIRUS	2	2	0	0	0	0	0	0	0	0	0	4
0206	INFLUENZA A H1N1	4	53	0	0	0	1	0	0	0	0	0	56
0299	INFLUENZA VIRUS - TYPING PENDI	0	2	0	0	0	0	0	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	1	11	0	0	0	0	0	0	0	0	0	12
0302	PARAINFLUENZA VIRUS TYPE 2	0	6	0	0	0	0	0	0	0	0	0	6
0303	PARAINFLUENZA VIRUS TYPE 3	0	20	0	0	0	0	0	0	0	0	0	20
0400	RESPIRATORY SYNCYTIAL VIRUS (R	5	73	0	0	0	0	0	0	0	0	1	79
0500	RHINOVIRUS (ALL TYPES)	1	25	0	0	0	0	0	0	0	0	0	26
0600	MYCOPLASMA PNEUMONIAE	5	38	0	0	0	0	0	0	1	0	0	44
0700	ORNITHOSIS-PSITTACOSIS	0	2	0	0	0	0	0	0	0	0	0	2
0809	COXSACKIEVIRUS A9	0	0	1	2	0	0	1	0	0	0	1	5
0904	COXSACKIEVIRUS B4	1	1	0	0	0	0	0	0	0	0	0	2
0905	COXSACKIEVIRUS B5	0	1	0	0	0	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	1	0	0	0	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	0	0	0	5	0	0	2	0	0	0	0	7
1100	POLIOVIRUS NOT TYPED	0	2	0	0	0	0	2	0	0	0	0	4
1101	POLIOVIRUS TYPE 1	0	1	0	0	0	1	1	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	0	0	0	0	0	0	1
1200	MUMPS VIRUS	0	0	1	0	1	0	0	0	0	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	7	0	0	1	0	1	0	0	0	0	18	27
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	8	3	0	0	0	2	0	2	0	1	0	16
1303	VARICELLA-ZOSTER VIRUS	5	0	1	0	0	1	0	0	0	0	5	12
1306	HERPES SIMPLEX TYPE 1	6	5	0	0	1	0	0	0	0	1	63	76
1307	HERPES SIMPLEX TYPE 2	27	1	0	0	0	0	0	0	0	1	13	42
1399	HERPES VIRUS TYPING PENDING	0	2	0	0	0	0	0	0	0	0	2	4
1502	PICORNIA VIRUS - NOT TYPED = E	0	5	0	0	0	1	8	0	0	0	1	15
1521	MEASLES VIRUS	1	0	0	0	0	0	0	0	0	0	1	2
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	0	0	0	5	5
1532	HEPATITIS B ANTIGEN	33	0	0	0	0	0	1	30	0	0	0	64
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	6	0	0	0	6
1541	CHLAMYDIA A - C. TRACHOMATIS	6	0	0	0	0	0	0	0	0	0	0	6
1556	CMV - CYTOMEGALOVIRUS	3	34	0	1	0	1	0	0	0	5	1	45
1564	ROTAVIRUS	1	0	0	0	0	0	120	0	0	0	0	121
1566	NORWALK AGENT	0	1	0	0	0	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	4	0	4	0	0	8	0	0	0	0	16
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	1	0	0	0	0	1
TOTAL		129	409	4	14	2	9	164	38	2	9	111	891

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

PERIOD 24-8-88 TO 7-9-88

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
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	0	1	1	0	2
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	0	0	0	0	0	2
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	0	0	0	0	0	0	2	0	0	3
0201	INFLUENZA A VIRUS	0	0	0	0	1	0	3	5	4	0	13
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	0	0	0	0	0	1	0	1
0203	INFLUENZA B VIRUS	0	0	0	0	1	0	0	0	0	0	1
0206	INFLUENZA A H1N1	0	0	0	0	0	0	0	2	4	0	6
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	0	0	0	0	1	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	0	1	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	2	1	0	3
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	0	0	1	1
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	2	3	3	0	8
0903	COXSACKIEVIRUS B3	0	0	0	0	0	0	0	0	1	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	0	0	0	1	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	0	0	1	1
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	3	0	0	0	0	0	0	0	0	3
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	5	0	0	0	0	0	0	1	0	6
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	1	4	0	0	0	1	0	1	0	7
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	0	2	0	2
1306	HERPES SIMPLEX TYPE 1	0	66	0	0	0	0	0	2	1	0	69
1307	HERPES SIMPLEX TYPE 2	0	253	0	0	0	0	0	0	2	0	255
1399	HERPES VIRUS TYPING PENDING	1	2	0	0	0	0	0	0	1	0	4
1401	COXIELLA BURNETI	0	0	0	0	0	0	0	0	1	0	1
1532	HEPATITIS B ANTIGEN	0	1	1	0	0	0	0	0	4	0	6
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	0	3	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	2	96	0	0	0	0	0	0	1	0	99
1556	CMV - CYTOMEGALOVIRUS	1	1	0	1	0	4	0	11	19	0	37
1564	ROTAVIRUS	0	0	0	0	0	0	0	1	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	1	0	2
9992	ROSS RIVER VIRUS	0	0	0	0	6	0	0	0	0	0	6
TOTAL		9	428	6	1	8	4	6	31	54	3	550

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 24-8-88 TO 7-9-88

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

		019	065	110	111	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	0	1	2	4	6	6	1	19	39
0101	ADENOVIRUS TYPE 1	2	1	0	0	2	0	0	0	5
0102	ADENOVIRUS TYPE 2	3	1	2	0	2	0	0	0	8
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	0	0	0	2
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	0	1	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	1	1	0	3	2	0	7
0201	INFLUENZA A VIRUS	23	3	32	23	0	23	0	0	104
0202	INFLUENZA A VIRUS SUBTYPE H3N2	2	0	2	0	1	0	0	4	9
0203	INFLUENZA B VIRUS	0	0	2	0	1	2	0	0	5
0206	INFLUENZA A H1N1	37	0	6	1	1	0	0	19	64
0299	INFLUENZA VIRUS - TYPING PENDI	0	0	0	2	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	1	0	1	1	1	0	0	8	12
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	3	1	1	0	0	1	7
0303	PARAINFLUENZA VIRUS TYPE 3	0	3	13	0	0	0	0	5	21
0400	RESPIRATORY SYNCYTIAL VIRUS (R	12	12	18	21	8	1	3	7	82
0500	RHINOVIRUS (ALL TYPES)	7	2	7	4	0	2	0	5	27
0600	MYCOPLASMA PNEUMONIAE	8	3	30	3	8	0	0	0	52
0700	ORNITHOSIS-PSITTACOSIS	2	0	0	0	0	0	0	0	2
0809	COXSACKIEVIRUS A9	2	0	0	0	3	0	0	0	5
0903	COXSACKIEVIRUS B3	1	0	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	1	0	0	0	0	1	0	0	2
0905	COXSACKIEVIRUS B5	0	0	0	0	0	1	0	0	1
1009	ECHOVIRUS TYPE 9	0	1	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	6	1	0	0	0	0	0	0	7
1100	POLIOVIRUS NOT TYPED	0	0	1	1	0	3	0	0	5
1101	POLIOVIRUS TYPE 1	0	1	1	0	1	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	0	0	0	1	0	1	0	2
1200	MUMPS VIRUS	0	1	0	0	1	1	0	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	26	4	0	0	30
1301	HERPES SIMPLEX VIRUS - NOT TYP	5	1	0	0	0	0	0	0	6
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	13	0	0	2	8	0	0	23
1303	VARICELLA-ZOSTER VIRUS	0	4	1	0	7	2	0	0	14
1306	HERPES SIMPLEX TYPE 1	43	13	20	0	42	27	0	0	145
1307	HERPES SIMPLEX TYPE 2	54	31	9	0	144	57	0	2	297
1399	HERPES VIRUS TYPING PENDING	4	0	0	4	0	0	0	0	8
1401	COXIELLA BURNETI	0	0	0	0	0	1	0	0	1
1502	PICORNIA VIRUS - NOT TYPED = E	0	2	0	0	0	7	0	6	15
1521	MEASLES VIRUS	0	0	1	0	0	1	0	0	2
1522	RUBELLA VIRUS	1	0	3	0	0	1	0	0	5
1532	HEPATITIS B ANTIGEN	0	8	10	0	28	11	0	13	70
1535	HEPATITIS A ANTIBODY	0	3	5	0	0	1	0	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	3	39	31	0	12	1	0	19	105
1556	CMV - CYTOMEGALOVIRUS	27	4	7	3	8	13	1	19	82
1564	ROTAVIRUS	13	13	19	8	15	41	7	6	122
1566	NORWALK AGENT	0	0	1	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	1	0	0	2	0	13	2	0	18
9992	ROSS RIVER VIRUS	0	1	0	0	1	4	0	0	6
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	1	0	1
TOTAL		262	163	229	79	322	235	18	133	1441