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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME: A total of 1 751 reports were processed for this period.

Twenty cases of Q fever were reported, 13 from Queensland, 6 from New South Wales and 1 from Victoria. Occupational exposure data were only available for 8 of the Queensland cases:-

. 7 male meatworkers (2 from Rockhampton aged 22 and 32 years respectively, 2 from Beaudesert aged 32 and 44 years respectively, 1 from Wondai aged 34 years, 1 from Townsville aged 51 years and 1 from Inverell aged 19 years) and

. one 42 year old male farmer from Cairns.

None of the twenty patients were involved in the Q fever vaccine field trial conducted in South Australia.

Cytomegalovirus was isolated from the ulcerated cervix of a 21 year old female who was 38 weeks pregnant. The patient presented with profuse contact bleeding of the cervix and had a concurrent genital herpes simplex virus infection.

High haemagglutination-inhibiting (HI) antibody titres (5120) to Murray Valley Encephalitis (MVE) virus were detected in a 40 year old female who presented with palpitations and prostration following a recent visit to Argentina where she was infected with Dengue type 2. Subsequent serologic evidence of specific IgM to dengue type 2 suggests that the observed high titre of HI antibodies to MVE resulted from cross-reactivity within group B arboviruses rather than indicating a recent infection with MVE virus in Australia.

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Figures given may be subject to revision.

AIDS SURVEILLANCE - AUSTRALIA

To 12 November 1986, 344 cases of AIDS fulfilling the criteria of case definition have been reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification, by age group, and by risk category are shown below:-

TABLE 1: AIDS patients by State or Territory of notification

STATE/TERRITORY	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
NSW	235	7	242	108	6	114
VIC	52	-	52	23	-	23
QLD	23	2	25	16	2	18
WA	14	2	16	6	-	6
SA	4	-	4	-	-	-
NT	2	-	2	1	-	1
TAS	1	-	1	1	-	1
ACT	2	-	2	1	-	1
	333	11	344	156	8	164

TABLE 2: AIDS patients by age group

AGE (YEARS)	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
0-9	4	-	4	4	-	4
10-19	3	1	4	3	1	4
20-29	67	2	69	28	1	29
30-39	140	-	140	62	-	62
40-49	85	2	87	41	2	43
50-59	26	3	29	15	2	17
60+	8	3	11	3	2	5
	333	11	344	156	8	164

TABLE 3: AIDS patients by risk category

RISK GROUP	CASES	DEATHS
Homo-/Bi-sexual	302	133
IV Drug abuser	1	-
Homo-/Bi-sexual IV drug abuser	9	3
Blood transfusion recipient	24	21
Person with haemophilia	4	4
Heterosexual transmission	2	2
None of the above	2	1
	344	164

HIV SURVEILLANCE - VICTORIA

(Contributed by B. Monheit, Health Department Victoria, M. Waters, Fairfield Hospital; Ed Waldman, MDU, Melbourne University and C. Lewis, Royal Melbourne Hospital)

Testing for antibodies to HIV (Human Immunodeficiency Virus) became routinely available to the 4.1 million people of Victoria in April 1985. In addition to the Blood Bank Laboratories, three laboratories, jointly funded by the Commonwealth and State governments, have been approved to carry out all tests for HIV antibodies in the State of Victoria.

All tests for antibodies to HIV, performed on equipment approved by the National HIV Reference Laboratory, currently use HIV antibody testing kits purchased from Abbott and Electro-Nucleonic Inc. (ENI). All tests which are positive on one of the screening tests are forwarded to Fairfield Hospital (State Reference Laboratory) for confirmation by Radioimmunoprecipitation assay and/or Western Blot technique. Viral culture from individuals with equivocal results has also commenced on a small scale at Fairfield Hospital.

A specific request form (shown in CDI 85/13) developed for doctors who referred their patients for HIV antibody testing, together with the co-operation between the laboratories has enabled all test results to be pooled and analysed.

During the first six months of 1986, a total of 13 390 tests were performed on 11 867 persons in Victoria (excluding those tested by the blood banks). Two hundred and fifteen persons were found to have confirmed positive tests:

- 182 (85%) were from men who engaged in homo-/bi-sexual activities
- 6 (2.8%) were from persons who abused intravenous drugs
- 15 (7.0%) were recipients of blood or blood products
- 2 were coronial cases
- 1 was a female prostitute
- 9 (4.2%) were not specified

The number of HIV antibody positive cases among homo-/bi-sexual men and IV drugs abusers, detected for the past 4 quarters, are shown below:

	Homo-/bi-sexual men		IV drug users	
	*Number of tests	Number of new positive persons	*Number of tests	Number of new positive persons
July/Sept 1985	1 476	146	562	1
Oct/Dec 1985	1 047	96	593	3
Jan/Mar 1986	936	92	632	3
Apr/Jun 1986	940	90	603	3

* Approximately 15% of persons tested had more than one test performed during the study period.

Data analysis indicates that HIV infection is principally confined to persons in recognised high risk groups, in particular homo-/bi-sexual men.

SURVEILLANCE OF HAEMOPHILIA-ASSOCIATED ACQUIRED IMMUNODEFICIENCY SYNDROME

(Based on MMWR vol. 35/No. 43, 31 October 1986)

As of 15 September 1986, a total of 238 cases (231 males and 7 females) of haemophilia-associated acquired immunodeficiency syndrome (AIDS) have been reported to the Centers for Disease Control (CDC) through State health departments, haemophilia treatment centres (HTC) and physicians:

- 212 (89%) had haemophilia A (coagulation factor VIII deficiency)
- 16 (7%) had haemophilia B (factor IX deficiency)
- 7 (3%) had von Willebrand's disease
- 2 had an acquired inhibitor (antibody) to factor VIII
- 1 had a factor V deficiency.

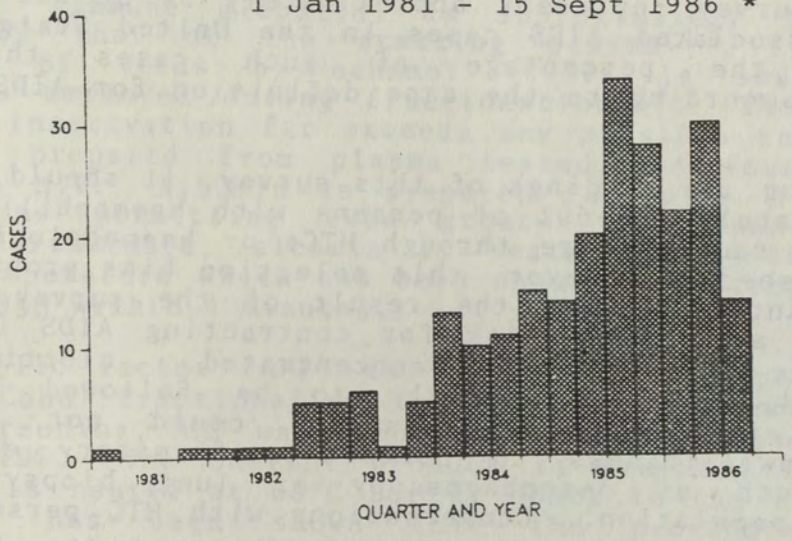
Thirteen patients were known to have had other risk factors for AIDS in addition to a haemotologic disease. The 238 patients resided in 38 states; almost half lived in California, New York, Pensylvania, New Jersey, or Missouri. The total number of cases represents a cumulative incidence of 1.6 cases of AIDS/100 haemophiliacs in the United States(1).

The first AIDS patient with underlying coagulation disorders was diagnosed as having Pneumocystic carinii pneumonia in 1981. Later it was recognised that this patient had AIDS. Since then, the number of haemophilia-associated AIDS cases has increased each year. The reported number of cases among haemophiliacs does not appear to be increasing at an exponential rate (Figure 1); however, in 1985; 92% of persons with haemophilia A and 52% of those with haemophilia B in a U.S. haemophilia cohort has antibodies to human immunodeficiency virus (HIV), suggesting exposure to the virus or to virus particles(2). HIV seropositivity in this cohort was associated with declining T helper lymphocyte numbers and with declining T helper-to-T suppressor cell ratios. Because of these high rates of seroprevalence and immunology findings, concern had been expressed that the recent incidence of haemophilia-associated AIDS may be misleadingly low because of a decline in reporting.

To determine the completeness of reporting, the Division of Host Factors (DHF), Center for Infectious Diseases, CDC, and the National Haemophilia Foundation (NHF) surveyed all United States HTCs, local NHF chapters, and physicians known to have patients with haemophilia(3). On 14 May 1986, each HTC/physician was sent a list of persons with haemophilia-associated AIDS according to DHF records as of 1 May 1986. Since patients' names are not used at DHF, cases

were identified only by the patient's date of birth, the date of diagnosis, and the nature of the AIDS diagnosis. The HTCs/physicians were asked to add to this list any other known cases - confirmed or suspected - among persons with haemophilia. DHF personnel telephoned all HTCs/physicians who had not responded by 1 August 1986.

FIGURE 1: Cases of Haemophilia-associated AIDS, by quater of diagnosis - U.S.A. 1 Jan 1981 - 15 Sept 1986 *



*Recently diagnosed cases may not be included because of a lag time in reporting

A total of 240 HTCs/physicians and 34 NHF chapters were sent letters, and written responses were received from 61 (25%) HTCs/physicians. Information was obtained by telephone from 209 of the 213 addresses who had not responded; four NHF chapters could not be reached. In addition, DHF personnel contacted the state health departments of three States that had no reported cases and no HTCs or physicians listed in the NHF directory. From these efforts, eight previously unreported cases of AIDS among persons with haemophilia were identified:

- 2 patients were from California (diagnosis of AIDS 12/84 and 7/85)
- 2 patients were from Oregon (diagnosis of AIDS 3/86 and 7/86)
- 1 each from Colorado (diagnosis of AIDS 3/85)
  Missouri (diagnosis of AIDS 5/85)
  New York (diagnosis of AIDS 4/85) and
  Virginia (diagnosis of AIDS 1/86).

In four instances, the physicians assumed that the cases had been reported to the appropriate state health departments. In the other instances, two cases involved physicians who did not realise their legal responsibility to report cases of AIDS to the state; one case involved a post mortem diagnosis of opportunistic infection, of which the physician had been unaware; and one case involved an acquired inhibitor to factor VIII, which the physician did not realise constitute a case of haemophilia-associated AIDS.

MMWR Editorial Comment:

National surveillance for AIDS cases among persons with haemophilia is maintained through the receipt of standard AIDS case report forms submitted by state health departments to CDC and through reports (without names) sent directly to DHF by physicians and nurses who care for patients with haemophilia. In the latter case, information is immediately shared with the state health department. The 8 unreported cases identified in the CDC-NHF survey represent approximately 3% of all reported haemophilia-associated AIDS cases in the United States. This approximates the percentage of such cases that were reclassified according to the case definition for AIDS revised in 1985[4].

In interpreting the findings of this survey, it should be noted that approximately 50%-60% of persons with haemophilia in the United States receive care through HTCs or haematologists (CDC data, unpublished). However, this selection bias probably does not significantly distort the result of the survey, because haemophiliacs at greater risk for contracting AIDS (ie those who require extensive concentrated clotting-factor replacement[5], are most likely to be followed by these health care providers. The survey could not determine willingness/unwillingness to perform confirmatory diagnostic procedures such as oesophagoscopy or lung biopsy in the haemophiliac population. Conversations with HTC personnel and physicians, however, suggest that confirmatory procedures are usually done. Finally, this approach to validation of the surveillance system assumes that physicians who do not initially choose to report AIDS cases (eg for reasons of confidentiality) would do so when contacted personally. This may not be the case. Never the less, the survey described here and other studies[6, 7] suggest that surveillance of AIDS (as currently defined) - particularly of haemophiliacs - is relatively complete.

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INACTIVATION OF HUMAN IMMUNODEFICIENCY VIRUS DURING PROCESSING OF PLASMA PRODUCTS

(based on CDWR Vol 12-32, 9 August 1986)

Because of its continued concern about the transmission of human immunodeficiency virus (HIV) through the administration

of infected plasma fractions, Health and Welfare Canada have financially assisted the Plasma Products Division of Connaught Laboratories Ltd, Willowdale, Ontario, to undertake studies to assess the effectiveness of current processing methods in removing exogenous HIV.

Connaught uses the Cohn ethanol fractionation procedures to prepare albumin and immune globulin from plasma collected by the Canadian Red Cross. It has been demonstrated that under the conditions of Cohn fractionation, HIV is partitioned almost exclusively in the precipitates and the virus content of fraction II (immune globulin) is approximately 12-15 $\log_{10}$ $TCID_{50}$ below that of the starting plasma[1]. The actual destruction of virus by ethanol itself is small at the temperatures employed during fractionation[2]. The extent of such viral inactivation far exceeds any possible contamination of product prepared from plasma tested and found free of antibody to HIV. Albumin is prepared following 2 additional precipitations permitting even greater assurance of virus removal. Furthermore, albumin is heated at 58-60°C for 10 hours, a temperature which has been shown to destroy more than 9 $\log_{10}$ $TCID_{50}$ within 4 minutes.

Antihaemophilic factor (AHF) is prepared by cryoprecipitation prior to Cohn fractionation. Before the introduction of anti-HIV screening, AHF was reported to transmit the causative agent of AIDS. The current product is prepared from tested plasma and is heated at 68°C for 72 hours in the freeze-dried state. It has been shown that the procedure destroys approximately 45 $\log_{10}$ $TCID_{50}$ of HIV, a quantity of virus, substantially greater than that reasonably present in any pool of plasma prepared from antibody-screened donors. The same procedure is used in the preparation of Factor IX complex concentrates and in the studies described here, the heat treatment destroyed 9 $\log_{50}$ $TCID_{50}$ in less than 72 hours.

Intravenous immunoglobulin is prepared from fraction II, and involves a diafiltration at acid pH. This procedure destroyed an additional 1-2 $\log_{10}$ $TCID_{50}$ at 4°C and 4 $\log_{10}$ $TCID_{50}$ at 20^0C, providing additional evidence of product safety. These studies and those of others[3,4,5] support the following conclusions of a recent WHO advisory panel on the safety of plasma and plasma derivatives:-

- Albumin preparations are safe.
- Immune globulin preparations are safe (intramuscular and intravenous).
- Antihaemophilic factor preparations provide sufficient HIV inactivation to exceed any contamiination expected from plasma screened for antibody to HIV.
- Factor IX complex preparations are safe.

These studies were performed at Bionetics Research Inc., Kensington, Maryland. Starting virus titres ranged from 6-9 $\log_{10}$ $TCID_{50}$. Residual virus was assayed by reverse transcriptase measurements following virus culture in H9 lymphocytes for up to 28 days.

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IMMUNISATION OF CHILDREN INFECTED WITH HUMAN IMMUNODEFICIENCY VIRUS (HIV)

(based on MMWR Vol. 35 No 38, 26 September 1986)

Recommendation of the Immunisation Practices Advisory Committee (ACIP)

INTRODUCTION

This document was prepared by the United States Immunisation Practices Advisory Committee and is intended to summarise available information and to assist health-care providers in developing policies for the immunisation of children infected with human immunodeficiency virus (HIV), the virus that causes acquired immunodeficiency syndrome (AIDS). These policies may vary depending upon the prevalence of HIV infection and the incidence of vaccine-preventable diseases in the community, individual assessment of a child's health status, and the risks and benefits of immunisation in a particular situation. This discussion considers the risks and benefits of immunisation for children residing in the United States based on the risks of vaccine-preventable diseases and the prevalence of HIV infection and is intended for use by health-care providers in that country. Accordingly the report notes that the recommendations may not pertain to other countries with different risks of vaccine-preventable diseases and prevalence of HIV infection among children. Since these recommendations are based upon current information and knowledge, periodic reassessment and revision will be required as more data concerning risk and benefits associated with immunisation of infected children become known and as the prevalences of specific vaccine-preventable diseases and HIV infection change.

HIV INFECTION AMONG CHILDREN

In the period 1 June, 1981-2 September, 1986, physicians and health departments in the United States reported 24 430 cases of AIDS to the Center for Disease Control. Three hundred and forty-five (1%) of the case-patients were children under 13 years of age who met the AIDS case definition; 75% of these paediatric cases were reported from New York, Florida, New Jersey, and California. Children with less severe manifestations of HIV infection (AIDS-related complex, or ARC) or with asymptomatic infections are not now reported to CDC, and no seroprevalence studies have been conducted among children. Thus, the number of less severely affected children and the number of infected but presently asymptomatic children are uncertain. In one recently published case series, 14(48%) of 29 symptomatic HIV-infected children met the CDC criteria for AIDS[1].

Fifty percent of children reported to CDC were diagnosed as having AIDS during the first year of life; 82%, by 3 years of age. Sixty-five percent of paediatric AIDS cases reported to CDC were fatal[2]. Short-term fatality rates are lower for children with less severe disease (ARC) who have not developed opportunistic infections; however, the ultimate prognosis of these children and of asymptomatic infected children is unknown.

MECHANISMS OF TRANSMISSION OF HIV AMONG CHILDREN

Two risk factors are predominately associated with HIV infection in children: a) being born to a mother who has HIV infection, and b) receiving blood or clotting factors containing HIV. Most cases-patients (79%) are children whose mothers probably are infected with the virus. The major risk factors for infection of these women are intravenous (IV) drug abuse and sexual contact with men at risk of HIV infection (primarily through drug abuse or bisexual contacts); women of Haitian or central African origin are also at a higher risk of acquiring HIV infection, and a small percentage of infected women have a history of being transfused with blood[3]. Approximately 15% of paediatric AIDS case-patients have received transfusions of blood or blood products, and 4% have haemophilia and have been treated with clotting-factor concentrates. Information about risk factors is incomplete for 3% of children with AIDS.

Currently available data indicate that most paediatric HIV infections are acquired from infected women during pregnancy, during labor and delivery, or perhaps shortly after birth. The risk of perinatal transmission from an infected mother to her infant is not known, although prospective studies indicate the rate of transmission has ranged from 0% (0/3) to 65% (13/20)[4,5]. Seropositive women who had previously delivered an infected child had the highest of these transmission rates (65%) in subsequent pregnancies. In a retrospective study evaluating nine children whose mothers were later diagnosed as having AIDS, two (22%) children had antibody to HIV. Additional prospective studies are needed to define more precisely the rate of perinatal transmission of HIV.

PREVALENCE OF HIV INFECTION AMONG WOMEN OF CHILD-BEARING AGE

The prevalence of HIV infection among women of child-bearing age varies depending on the patient group and geographic area[3]. Reported confirmed seroprevalences are less than 0.01% among female blood donors in Atlanta, Georgia, and 0.06% among female U.S. military recruit applicants[3,6]. In contrast, the reported prevalence of HIV antibody among IV drug abusers has ranged from 2% to 59%, with the highest prevalence in New York City and northern New Jersey. Female sex partners of IV drug-abusing men with AIDS or with ARC had a reported seroprevalence of 40%-71%, whereas 10% of female partners of asymptomatic infected haemophiliacs were reported to be seropositive[3]. Seroprevalence among prostitutes has varied greatly (5%-40%) depending on the geographic area and has been largely attributed to a coincidental history of IV drug abuse[3]. Seroprevalence has been reported to be as high as 5% among persons born in countries in which heterosexual transmission of HIV is thought to play a major role (eg Haiti, central African countries)[7].

IMMUNOLOGICAL ABNORMALITIES ASSOCIATED WITH HIV INFECTION

Children with symptomatic HIV infection (AIDS or ARC) have immunological abnormalities similar to those of adult AIDS patients, including hypergammaglobulinaemia, decreased T4 lymphocyte count, reversed helper/suppressor T-cell ratios,

poor T-lymphocyte responses to mitogen stimulation, and altered humoral immunity. Lymphopenia (cell counts less than 1 500 cells/mm^3) is uncommon. Antibody responses of children with AIDS or ARC to diphtheria and tetanus toxoid boosters and to pneumococcal vaccine were absent or lower than those of age-matched controls, which is consistent with defective humoral immunity[8,9]. Some HIV-infected children responded adequately to immunisation; 60% of AIDS and ARC patients given measles-mumps-rubella vaccine (MMR) prior to diagnosis had protective levels of measles antibodies 5-66 months after immunisation.

Asymptomatic HIV-infected adults as a group generally have less severe abnormalities of immunologic function than adults with AIDS or ARC, and some may have normal immunological function, although individual asymptomatic adults may have severe abnormalities[10]. Immunological function of asymptomatic HIV-infected children has not yet been adequately studied but presumably would be more intact than that of symptomatic HIV-infected children. In a small prospective study, all 29 children with symptomatic HIV-infection had immunological abnormalities within 5-13 months of being found infected, compared with only two of seven (29%) children reported to have asymptomatic HIV-infection[1].

CONCERNS ABOUT IMMUNISATION OF HIV-INFECTED CHILDREN

The immunological abnormalities associated with symptomatic HIV infection have raised concerns about the immunisation of infected children. Replication of live, attenuated vaccine viruses may be enhanced in persons with immunodeficiency diseases and theoretically may produce serious adverse reactions following immunisation of symptomatic HIV-infected (AIDS and ARC) patients[11]. Concerns have been expressed on theoretical grounds that antigenic stimulation by immunisation with inactivated vaccines might lead to a deterioration of clinical status of HIV-infected children, but this effect has not been documented[12]. Since symptomatic HIV-infected patients patients have abnormal primary and secondary antibody responses, the efficacy of immunisation may be decreased[13]. The efficacy of immunisation for asymptomatic HIV-infected children is unknown, but presumably would be higher than for symptomatic HIV-infected children.

Because most HIV-infected children become infected perinatally, it is to be expected that their mothers are infected with HIV. Other family members may also be infected with HIV and may have abnormal immunological function and Prospective evaluation of 16 asymptomatic HIV-infected mothers of children diagnosed as having AIDS or ARC showed that 12 (75%) mothers developed AIDS or ARC during a 30-month follow-up period[4]. Regardless of the immune status of the recipient, poliovaccine virus is often excreted by children vaccinated with oral poliovaccine (OPV) and may be transmitted to close contacts[14]. Immune-deficient individuals (either recipients or contacts) have a higher risk of developing vaccine-associated poliomyelitis than normal individuals. There is no risk of transmitting the viruses contained in measles, mumps, rubella (MMR) vaccine to family members[15-17].

While the risks of vaccination are not known with certainty, potential risks may exist if HIV-infected children are not vaccinated. If local outbreaks of measles occur in geographic areas in which there is both a cluster of unvaccinated children and a high prevalence of HIV-infection, the risk of measles for unvaccinated, HIV-infected children may be high. Measles infection among patients with immune deficiency may be severe, protracted, and fatal.

EXPERIENCES WITH IMMUNISATION OF HIV-INFECTED PERSONS

Some children infected perinatally with HIV have received routine immunisation with OPV and MMR before their illnesses were recognised. Out-patient medical records from New York City and Miami for 213 children with symptomatic HIV infection (AIDS and ARC), presumably acquired during the perinatal period, were reviewed to determine immunisation history and possible vaccine-associated adverse reactions. One hundred and seventy-one children (80%) had received at least one dose of OPV and diphtheria and tetanus toxoids and pertussis vaccine (DTP), 95 (45%) had completed primary immunisation with OPV and DTP (three doses and four doses, respectively), and 63 (30%) had received MMR or measles vaccine. Thirty-eight (39%) of 98 children who had available records of dates of immunisation and onset of symptoms consistent with HIV infection had received at least one live-virus vaccine after symptom onset. No serious or unusual adverse reactions were noted in the medical records of these children following immunisation.

Only one adverse reaction following immunisation of an HIV-infected person has been documented. A 19-year-old asymptomatic army recruit received multiple immunisations during basic training, including primary immunisation with smallpox vaccine. Two and one-half weeks later, he developed cryptococcal meningitis and was diagnosed as having AIDS. One and one-half weeks later, while being treated for meningitis, he developed lesions of disseminated vaccinia. He was treated with vaccinia immune globulin and recovered from vaccinia, but has since died of AIDS.

CDC has not received any reports of vaccine-associated poliomyelitis among HIV-infected vaccine recipients or their contacts or among other persons known to be infected with HIV. There have been no reports of serious adverse events following MMR administration from areas in which paediatric AIDS cases are occurring.

IMMUNISING CHILDREN WHO MAY BE INFECTED WITH HIV: SPECIAL CONSIDERATIONS

Children born to women who are at risk of HIV-infection or who are known to be infected with HIV should be evaluated for infection with the virus-including being tested for antibody[3,18]. For asymptomatic children presenting for immunisation, this evaluation and testing is not necessary to make decisions about immunisations. Children infected with HIV are best cared for by paediatricians knowledgeable in the management of patients with this infection. Since little information is currently available on the safety and efficacy of immunising children who may be infected with HIV, special studies of these children need to be conducted.

RECOMMENDATIONS

Children with symptomatic HIV infection

A. Live-virus and live-bacterial vaccines (e.g. MMR, OPV, BCG) should not be given to children and young adults who are immunosuppressed in association with AIDS or other clinical manifestations of HIV infection. For routine immunisations, these persons should receive inactivated poliovaccine (IPV) and should be excused for medical reasons from regulations requiring measles, rubella, and/or mumps immunisation.

B. Concerns have been raised that stimulation of the immune system by immunisation with inactivated vaccines in these individuals might cause deterioration in immunological function. However, such effects have not been noted thus far among children with AIDS or among other immunosuppressed individuals after immunisation with inactivated vaccines. The potential benefits of immunisation of these children outweigh the concerns of theoretical adverse reactions. Immunisation with DTP, IPV, and Haemophilus influenzae type b vaccines is recommended in accordance with the ACIP recommendations, although immunisation may be less effective than it would be for immunocompetent children[19-21].

C. As with other conditions that produce chronic immunosuppression, the Committee recommends annual immunisation with inactivated influenza vaccine for children over 6 months of age and one-time administration of pneumococcal vaccine for children over 2 years of age[22-24].

D. Children and young adults with AIDS or other clinical manifestations of HIV infection - as other immunosuppressed patients - may be at increased risk of having serious complications of infectious diseases, such as measles and varicella. Following significant exposure to measles or varicella, these persons should receive passive immunisation with immune globulin (IG) or varicella-zoster immune globulin (VZIG), respectively[15,25].*

Children with previously diagnosed asymptomatic HIV infection

A. A small number of children and young adults known to be infected with HIV and without overt clinical manifestations of immunosuppression have received live-virus vaccines without adverse consequences. Further information is needed, but on the basis of data now available, the Committee believes that such persons should be vaccinated with MMR in accordance with ACIP recommendations[15-17]. Vaccinated children should be followed for possible adverse reactions and for the occurrence of vaccine-preventable diseases since immunisation may be less effective than for uninfected persons.

B. Available data suggest that OPV can be administered without adverse consequences to HIV infected children who do not have overt clinical manifestations of immunosuppression. However, because family members of such children may be immunocompromised due to AIDS or HIV infection and therefore at increased risk of paralysis from contact with spread vaccine virus, it may be prudent to use IPV routinely to immunise asymptomatic children with previously diagnosed HIV infection[19].

C. Immunisation with DTP and Haemophilus influenzae type b vaccines is recommended in accordance with ACIP recommendations[20,21].

Children not known to be infected with HIV

Children and young adults not known to be infected with HIV should be immunised in accordance with ACIP recommendations.

Children residing in the household of a patient with AIDS

Children whose household members are known to be immunocompromised due to AIDS or other HIV infections should not receive OPV because vaccine viruses are excreted by the recipient of the vaccine and may be communicable to their immunosuppressed contacts. These children should receive IPV for routine immunisation[28]. Because extensive experience has shown that live, attenuated MMR vaccine viruses are not transmitted from vaccinated individuals to others, MMR may be given to a child residing in the household of a patient with AIDS[15-17].

+Such family members may have been infected by sexual contact with an HIV-infected person by parenteral exposure to infected blood (e.g. by sharing needles), or as haemophiliacs who received clotting factors, or by perinatal transmission.

*Some physicians administer full replacement doses of intravenous IG on a 2-4 week schedule to children with AIDS and other clinical manifestations of HIV infection. This therapy may provide some protection against such diseases as measles and varicella.

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DIAGNOSIS AND MANAGEMENT OF MYCOBACTERIAL INFECTIONS IN PERSONS WITH HUMAN IMMUNODEFICENCY VIRUS

(Based on MMWR (1986) 35 (28) 448-52)

The number of new tuberculosis cases reported to the U.S. Centers for Disease Control in 1985 was essentially the same as that reported in 1984[1]. In contrast, the average annual decline in morbidity during the past 32 years has been 5%. As previously reported in the CDI (No. 86/8: 8-9) the failure of tuberculosis morbidity to decline as expected in 1985 is probably related to the occurrence of tuberculosis among persons with acquired immunodeficency syndrome (AIDS) or human immunodeficiency virus (HIV) infection. Several reports have indicated that mycobacterial disease is common among AIDS patients and among persons at risk for AIDS[2-9]. The most common mycobacterial species isolated from patients with diagnosed AIDS is Mycobacterium avium complex (MAC), although in some groups in which tuberculous infection is highly prevalent, M. tuberculosis is more common[10-12]. Even among groups in which MAC is the most common mycobacterial pathogen, M. tuberculosis accounts for a substantial proportion of the mycobacteral isolates. The association between mycobacterial disease and AIDS raises several important clinical and public health issues that are discussed below.

DIAGNOSIS OF TUBERCULOSIS IN PATIENTS LIKELY TO HAVE HIV INFECTION

Clinicians should consider the diagnosis of tuberculosis in patients with, or at risk of, HIV infection, even if the clinical presentation is unusual[4, 13, 14]. Available data indicate that extrapulmonary forms of tuberculosis, particularly lymphatic and disseminated (miliary), are seen much more frequently among patients with HIV infection than those without such infection. Pulmonary tuberculosis in patients with HIV infection cannot be distinguished from other pulmonary infections, such as Pneumocystis carinii pneumonia, on the basis of clinical and radiographic findings. Patients with tuberculosis may have infiltrates in any lung zone, often associated with mediastinal and/or hilar lymphadenopathy Cavitation is uncommon. Appropriate specimens to establish a culture-confirmed diagnosis include respiratory secretions, urine, blood, lymph node, bone marrow, liver, or other tissue or body fluid that is indicated clinically. All tissue specimens should be stained for acid-fast bacilli and cultured for mycobacteria.

In the presence of undiagnosed pulmonary infiltrates, bronchoscopy with lavage and transbronchial biopsy (if not contraindicated) may be needed to obtain material for both culture and histological examination. A tuberculin skin test should be administered, but in the absence of a reaction does not rule out the diagnosis of tuberculosis because immunosuppression associated with HIV infection may cause false-negative results.

TREATMENT OF MYCOBACTERIAL DISEASE IN PATIENTS WITH HIV INFECTION

Chemotherapy should be initiated whenever acid-fast bacilli are found in a specimen from a patient with HIV infection and clinical evidence of mycobacterial disease.

Because of the difficulty in distinguishing tuberculosis from MAC disease by any criterion other than culture, and because of the individual and public health implications of tuberculosis, it is important to treat patients with a regimen effective against tuberculosis. With some exceptions, patients with tuberculosis and HIV infection respond relatively well to standard antituberculosis drugs[15], however, their treatment should include at least three drugs initially, and treatment may need to be longer than the standard duration of 9 months[15]. The recommended regimen is:

- isoniazid (INH), 10-15 mg/kg/day up to 300 mg/day;
- rifampicin (RIF), 10-15 mg/kg/day up to 600 mg/day;
- ethambutol (EMB), 25 mg/kg/day, or pyrazinamide (PZA), 20-30 mg/kg/day.

The latter two drugs are usually given only during the first 2 months of therapy. The addition of a fourth drug may be indicated in certain situations, such as central nervous system or disseminated disease or when INH resistance is suspected. An initial drug-susceptibility test should always be performed, and the treatment regimen revised if resistance is found to any of the drugs being used. The appropriate duration of treatment for patients with tuberculosis and HIV is unknown; however, it is recommended that treatment continue for a minimum of 9 months and for at least 6 months after documented culture conversion. If INH or RIF is not included in the treatment regimen, therapy should continue for a minimum of 18 months and for at least 12 months following culture conversion. After therapy is completed patients should be followed closely, and mycobacteriological examinations should be repeated if clinically indicated.

The clinical significance and optimal therapy of MAC disease in patients with HIV infection is not well defined, and there are no definitive data on the efficacy of treatment. In the U.S., a commonly used regimen for the treatment of MAC disease substitutes rifabutin for rifampicin, combined with INH, EMB and clofazimine (rifabutin and clofazimine are experimental drugs and are available in the U.S. under investigational new

drug protocols). If M. tuberculosis is isolated from a patient receiving this four-drug regimen, treatment is switched to one of the three-drug regimens outlined above (INH, RIF and EMB or PZA). If MAC is isolated from a patient who has been started on a three-drug regimen, the clinician may continue the three-drug regimen or switch to the four-drug regimen of INH, EMB, rifabutin and clofazimine.

Although experience is very limited, patients with disease due to M. kansasii should respond to INH, RIF and EMB. Some clinicians advocate the addition of streptomycin, 1 gram twice weekly, for the first 3 months. Therapy should continue for a minimum of 15 months following culture conversion.

Monitoring the toxicity of antimycobacterial drugs may be difficult for patients who may be receiving a variety of other drugs and may have other concomitant conditions. Because hepatic and haematological abnormalities may be caused by the mycobacterial disease, AIDS, or other drugs or conditions, the presence of such abnormalities is not an absolute contraindication to the use of the treatment regimens outlined above.

INFECTION CONTROL

The infection control procedures applied to patients with HIV infection who have undiagnosed pulmonary disease should, in addition to conforming to the recommendations for preventing transmission of HIV,(17) take into account the possibility of tuberculosis. This is particularly important when diagnostic procedures, such as sputum induction or bronchoscopy, are being performed.

CONTACT INVESTIGATION FOR TUBERCULOSIS

Patients with pulmonary tuberculosis and HIV should be considered potentially infectious for tuberculosis, and standard procedures for tuberculosis contact investigation should be followed(18). Specific data on the infectiousness of tuberculosis in patients with HIV infection are not yet available.

EXAMINING HIV-INFECTED PATIENTS FOR TUBERCULOSIS AND TUBERCULOSIS INFECTION

Individuals who are known to be HIV seropositive should be given a Mantoux skin test with 5 tuberculin units of purified protein derivative as part of their clinical evaluation. Although some false-negative skin tests may be encountered in this setting as a result of immunosuppression induced by HIV infection, significant reactions are still meaningful(19). If the skin test reaction is significant, a chest radiograph should be obtained, and if abnormalities are detected, additional diagnostic procedures for tuberculosis should be undertaken. Patients with clinical AIDS or other Class IV infections (20) should receive both a tuberculin skin test and a chest radiograph because of the higher probability of false-negative tuberculin reactions in immunosuppressed patients.

EXAMINING PATIENTS WITH CLINICALLY ACTIVE TUBERCULOSIS OR LATENT TUBERCULOSIS INFECTION FOR HIV INFECTION

As part of the evaluation of patients with tuberculosis and tuberculous infection, risk factors for HIV should be identified. Voluntary testing of all persons with these risk factors is recommended[21]. In addition, testing for HIV antibody should be considered for patients of all ages who have severe or unusual manifestations of tuberculosis. The presence of HIV infection has implications regarding treatment (see above), alerts the physician to the possibility of other opportunistic infections, and allows for counselling about transmission of HIV infection[22]. Testing for HIV antibody is especially important for persons over age 35 with asymptomatic tuberculosis infection, because INH would not usually be indicated for persons in this age group unless they are also HIV seropositive.

PREVENTIVE THERAPY

HIV seropositivity in a person of any age with a significant tuberculin reaction is an indication for INH preventive therapy[16]. Although it is not known whether INH therapy is as efficacious in preventing tuberculosis in HIV-infected persons as in other groups, the usually good response of HIV-infected persons with tuberculosis to standard therapy suggests that INH preventive therapy would also be effective. Before instituting preventive therapy, clinically active tuberculosis should be excluded.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 10/11/86 - 23/11/86 BULLETIN NUMBER 86/24
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RANC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED			10		8	1	19	2	40
0101 ADENOVIRUS TYPE 1						4		1	5
0102 ADENOVIRUS TYPE 2	1	1		1		2			5
0103 ADENOVIRUS TYPE 3								1	1
0105 ADENOVIRUS TYPE 5	1								1
0107 ADENOVIRUS TYPE 7						1			1
0113 ADENOVIRUS TYPE 13	1								1
0119 ADENOVIRUS TYPE 19				1					1
0128 ADENOVIRUS TYPE 28	1								1
0199 ADENOVIRUS TYPING PENDING		1			7				8
0201 INFLUENZA A VIRUS	1		3	1		1	1		7
0203 INFLUENZA B VIRUS	5	2	4					1	12
0301 PARAINFLUENZA VIRUS TYPE 1					1				1
0302 PARAINFLUENZA VIRUS TYPE 2	2	1			1	1		1	6
0303 PARAINFLUENZA VIRUS TYPE 3	3			4	4	15	9	1	36
0399 PARAINFLUENZA VIRUS TYPING PENDING					2				2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	40	1	4	5	2	19	19	7	97
0500 RHINOVIRUS (ALL TYPES)				1	11	10	5	2	29
0600 MYCOPLASMA PNEUMONIAE	3			1		4	8	13	29
0700 ORNITHOSIS-PSITTACOSIS	4						1		5
0816 COXSACKIEVIRUS A16								2	2
0901 COXSACKIEVIRUS B1						1			1
0906 COXSACKIEVIRUS B6						1			1
1005 ECHOVIRUS TYPE 5				1				1	2
1008 ECHOVIRUS TYPE 8						1			1
1011 ECHOVIRUS TYPE 11				22	10	2		2	36
1014 ECHOVIRUS TYPE 14							1		1
1018 ECHOVIRUS TYPE 18						1			1
1025 ECHOVIRUS TYPE 25								1	1
1100 POLIOVIRUS NOT TYPED			1		5		2		8
1101 POLIOVIRUS TYPE 1						2		1	3
1103 POLIOVIRUS TYPE 3						3		1	4
1104 POLIOVIRUS-VACCINAL STRAIN					1				1
1200 MUMPS VIRUS								1	1
1300 HERPES VIRUS GROUP-NOT TYPED	40			5				1	46
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1					1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	9	2	11			3	15	13	53
1303 VARICELLA-ZOSTER VIRUS	2			1	1		2	2	8
1306 HERPES SIMPLEX TYPE 1	10		10	30		27	68	30	175
1307 HERPES SIMPLEX TYPE 2	30		22	61		15	176	77	381
1399 HERPES VIRUS TYPING PENDING					5				5
1401 COXIELLA BURNETI	6			1			13		20
1502 PICORNA VIRUS-NOT TYPED			2				21	2	25
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)			1						1
1521 MEASLES VIRUS		1						1	2
1522 RUBELLA VIRUS	13			12			26	9	60
1532 HEPATITIS B ANTIGEN	90	2	8	14		24	39	20	197
1535 HEPATITIS A ANTIBODY	5		1	6		5	3	12	32
1541 CHLAMYDIA A - C TRACHOMATIS	28		2			37	39	30	136
1543 CHLAMYDIA A - LGV TYPE	1			13			12		26
1556 CMV - CYTOMEGALOVIRUS	4		1	20	1	7	18	15	66
1564 ROTAVIRUS	25	2	4			17	59	5	112
1571 ENTEROVIRUS TYPE 71 (BRCR)	1				4	1			6
1599 ENTEROVIRUS TYPING PENDING		1	5		12				18
9992 ROSS RIVER VIRUS							27		27
9994 SMALL VIRUS (LIKE) PARTICLE		1							1
9997 KUNJIN VIRUS							1		1
9998 ARBO. GROUP B.								1	1
Total	326	15	89	201	75	205	584	256	1,751

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0100 ADENOVIRUS NOT TYPED.....		1									
0101 ADENOVIRUS TYPE 1.....	1	3									
0102 ADENOVIRUS TYPE 2.....		1				1	2				1
0107 ADENOVIRUS TYPE 7.....							1				
0128 ADENOVIRUS TYPE 28.....							1				
0201 INFLUENZA A VIRUS.....	2	2									
0203 INFLUENZA B VIRUS.....	2	5									
0301 PARAINFLUENZA VIRUS TYPE 1....		1					1				
0302 PARAINFLUENZA VIRUS TYPE 2....		6									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	30				1	1				1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	3	93									
0500 RHINOVIRUS (ALL TYPES).....		18									
0600 MYCOPLASMA PNEUMONIAE.....		20	1	1							
0700 ORNITHOSIS-PSITTACOSIS.....	1	3									
0816 COXSACKIEVIRUS A16.....		1									1
0901 COXSACKIEVIRUS B1.....									1		
0906 COXSACKIEVIRUS B6.....											1
1005 ECHOVIRUS TYPE 5.....				2							
1011 ECHOVIRUS TYPE 11.....		6		24			3				2
1014 ECHOVIRUS TYPE 14.....							1				
1018 ECHOVIRUS TYPE 18.....				1							
1025 ECHOVIRUS TYPE 25.....							1				
1101 POLIOVIRUS TYPE 1.....	1						2				
1103 POLIOVIRUS TYPE 3.....	1						1				
1104 POLIOVIRUS-VACCINAL STRAIN....		1									
1300 HERPES VIRUS GROUP-NOT TYPED..	1										
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	14	3					1	3			3
1303 VARICELLA-ZOSTER VIRUS.....		1						1	1		5
1306 HERPES SIMPLEX TYPE 1.....	2	4	1	1				1		2	111
1307 HERPES SIMPLEX TYPE 2.....	5										133
1401 COXIELLA BURNETI.....	5	2									1
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....		1									2
1522 RUBELLA VIRUS.....	3	3	1								44
1532 HEPATITIS B ANTIGEN.....	54							121			
1535 HEPATITIS A ANTIBODY.....	9							22			
1541 CHLAMYDIA A - C.TRACHOMATIS...	18										
1543 CHLAMYDIA A - LGV TYPE.....	18									2	
1556 CMV - CYTOMEGALOVIRUS.....	6	17								6	
1564 ROTAVIRUS.....	1						111				
1571 ENTEROVIRUS TYPE 71 (BRCR)....	1	1		2							1
1599 ENTEROVIRUS TYPING PENDING....				1							
9992 ROSS RIVER VIRUS.....	5	1									6
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
9997 KUNJIN VIRUS.....											1
9998 ARBO. GROUP B.									1		
Total.....	154	224	3	32		2	127	148	3	10	314

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Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....			1							
0103 ADENOVIRUS TYPE 3.....	1									
0105 ADENOVIRUS TYPE 5.....	1									
0113 ADENOVIRUS TYPE 13.....							1			
0119 ADENOVIRUS TYPE 19.....	1									
0201 INFLUENZA A VIRUS.....								4		
0203 INFLUENZA B VIRUS.....							2	2	1	
0303 PARAINFLUENZA VIRUS TYPE 3....							1	2	1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....							1	3		
0600 MYCOPLASMA PNEUMONIAE.....								8	4	
0700 ORNITHOSIS-PSITTACOSIS.....					1			1		
1008 ECHOVIRUS TYPE 8.....										1
1011 ECHOVIRUS TYPE 11.....								4		
1103 POLIOVIRUS TYPE 3.....						1				1
1300 HERPES VIRUS GROUP-NOT TYPED..							1			
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			6	11			3	8	6	
1303 VARICELLA-ZOSTER VIRUS.....								2		
1306 HERPES SIMPLEX TYPE 1.....	8	44					1	1		
1307 HERPES SIMPLEX TYPE 2.....		244								
1401 COXIELLA BURNETI.....							1	12	1	
1502 PICORNA VIRUS-NOT TYPED.....			1					1		
1521 MEASLES VIRUS.....							1			
1522 RUBELLA VIRUS.....			4	1	13			8	7	
1532 HEPATITIS B ANTIGEN.....									22	
1535 HEPATITIS A ANTIBODY.....									1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	6	112								
1543 CHLAMYDIA A - LGV TYPE.....		6								
1556 CMV - CYTOMEGALOVIRUS.....		6	1		2	7	4	4	13	
1571 ENTEROVIRUS TYPE 71 (BRCR)....							1			
9992 ROSS RIVER VIRUS.....			1		11			10		
9997 KUNJIN VIRUS.....					1					
9998 ARBO. GROUP B.								1		
Total.....	17	413	14	12	28	8	17	71	56	2

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 7 - 14 June 1986 to 11 July 1986 Bulletin..86/24....

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis	3		1	1					5	29
Ankylostomiasis			1	1			NN		2	15
Anthrax									-	-
Arbovirus infection	15	1	77		1				94	1 059
Brucellosis									-	11
Campylobacter infections	86	4	NN	94	7	NN		NN	191	1 305
Chancroid				NN	2				2	7
Cholera									-	-
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria							3		3	23
Donovanosis			2	NN	10		4		16	79
Giardiasis	31		NN	64	10	NN	NN	NN	105	690
Genital herpes	75		15	16	NN	NN	2	NN	108	722
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	-
Gonorrhoea	91		83	40	115	1	42	9	381	2 693
Hepatitis A (infectious)	24	3	21	63	54		2		167	* 1 036
Hepatitis B (serum)	39	20	35	7	30	1	4	4	140	* 1 023
Hepatitis – unspecified	5		4	3	NN	NN	1		13	89
Hydatid disease									-	6
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	2	2	NN	2		NN		NN	6	43
Leprosy			1						1	9
Leptospirosis	1		5		1				7	111
Lymphogranuloma venereum				NN	NN	NN		NN	-	2
Marburg disease			NN			NN		NN	-	-
Malaria	16	6	29	3	3			2	59	386
									-	-
Meningococcal infections	3	2		1	1	NN			7	22

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	282		2	30	NN	NN	NN	NN	314	2 570
Ornithosis				4					4	25
Pertussis (whooping cough)	6	1	NN	1	6	NN		NN	14	395
Plague									-	-
Poliomyelitis									-	-
Q. fever	8		14	6					28	* 168
Rabies				NN		NN		NN	-	-
Salmonella infections	64	10	32	37	13	2	23	1	182	1 611
Shigella infections	14	2	12	5	14		19		66	511
Smallpox									-	-
Syphilis	33	1	27	11	11		77	1	161	1 274
Tetanus									-	4
Trachoma		NN				NN	NN		-	35
Tuberculosis (all forms)	38	42	18		3	3	2	1	107	* 563
Typhoid fever		1							1	22
Typhus (all forms)			4						4	12
Vibrio parahaemolyticus infections			NN			NN		NN	-	5
Yellow fever									-	-
Yersinia infections	4		NN	1		NN		NN	5	51

NN - Not Notifiable

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

* Adjustment to the Cumulative Total since last report:

Hepatitis A	+3	South Australia
Hepatitis B	+3	South Australia
Q fever	+3	South Australia
Tuberculosis	+1	South Australia
Typhoid fever	+ 5	New South Wales
Typhus (all forms)	-5	New South Wales