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Editor *Dr Robert Hall*

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 812 reports were processed during this period.

Seven cases of Q fever (6 males, 1 gender not stated) were reported during this period. Ages ranged from 15 to 50 years. No occupational exposure data were provided.

Eighty reports of Ross River virus were received. The cumulative total for the year so far is 1251 cases.

Echovirus type 30 activity is still being observed. Twenty-four cases were reported during this period, 13 from New South Wales. The total for the year so far is 152 (VIC, 56; WA, 54; NSW, 38; SA, 4).

NOTICE:

COMMUNICABLE DISEASE SURVEILLANCE IN AUSTRALIA WORKSHOP, 29-30 JUNE 1989

National Centre for Epidemiology and Population Health, The Australian National University

The National Centre is convening this workshop to discuss desirable principles for future communicable disease surveillance in Australia, examine the need for outbreak investigation facilities and formulate recommendations about future developments in communicable disease surveillance and outbreak investigation.

- The Bulletin is compiled and distributed by the Communicable Diseases Section, Communicable Diseases and Social Health Branch, Telephone: (062) 89 1555, Department of Community Services and Health.
- Contributions are solicited, and do not preclude later publication elsewhere.

- Figures given may be subject to revision.

Speakers will include Dr A.C.I. Adams, Chief Medical Adviser, Department of Community Services and Health, Dr Norman Noah from the Public Health Laboratory Service, Colindale, United Kingdom and Dr Stanley Music from the Centers for Disease Control, Atlanta USA. Invited participants will include representatives from a number of professional bodies and representatives of State and Commonwealth government departments which have responsibility for communicable disease control.

The workshop is open to persons with particular interests in this area. The registration fee is $50.00 and application papers can be obtained from:

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TUBERCULOSIS AND HUMAN IMMUNODEFICIENCY VIRUS INFECTION: RECOMMENDATIONS OF THE U.S. ADVISORY COMMITTEE FOR THE ELIMINATION OF TUBERCULOSIS (ACET)

(Based on MMWR 1989;38:236-8,243-50)

Introduction

Tuberculosis (TB) and other mycobacterioses are well-recognised complications of immunosuppression. In the 1980s, the epidemic of human immunodeficiency virus (HIV) infection and its resulting immunosuppression in large numbers of persons have increased the incidence of mycobacterial diseases [1]. Disseminated *Mycobacterium avium* complex (MAC) disease has become an important medical problem; MAC is the most common mycobacterial species isolated from persons with acquired immune deficiency syndrome (AIDS). Of particular public health concern, however, is the increasing number of persons with disease caused by *M. tuberculosis* [2-5]. HIV infection appears to be an important risk factor for TB. Moreover, TB is one of the few respiratory diseases occurring in HIV-infected persons that is transmissible, curable, and preventable. The U.S. Advisory Committee for Elimination of Tuberculosis (ACET) is concerned that the further spread of HIV infection among populations with a high prevalence of tuberculous infection may result in dramatic increases in TB unless appropriate control measures outlined in this statement are successfully implemented.

Epidemiology

The contribution of HIV-related TB morbidity to total national TB morbidity is not precisely known, but HIV infection appears to have had a substantial impact in some areas [6,7]. Matching reported TB cases with the AIDS case registries in 43 U.S. states and 11 localities reveals that 4% of AIDS cases appear on the TB registries [CDC, unpublished data]. In Florida, 10% of AIDS patients had histories of TB [8]; in New York City, 5% of adult and adolescent AIDS patients [9]; in Connecticut, 5%

[10]; and at a university hospital in New Jersey, 21% [4]. In San Juan, Puerto Rico, 11% of autopsied AIDS patients had TB [11], and at a New York City hospital, 4% of autopsied AIDS patients had previously undiagnosed TB [12].

Some data on HIV seroprevalence among TB patients have also been accumulated. In San Francisco, 29% of non-Asian adult TB patients 18-65 years of age were infected with HIV [13]. In Seattle, a combined 23% of black and white adult TB patients 20-50 years of age were HIV-infected [14].

Evidence for an association between HIV infection and TB comes from several studies. Of 279 HIV-infected methadone-maintenance patients in New York City, 12 had histories of TB; none of the 240 patients not infected with HIV had histories of TB [15]. In another cohort of methadone-maintenance clients with documented positive tuberculin skin test reactions, 14% of HIV-infected persons and none of the HIV-negative clients developed TB during a 2-year period [16]. In Kinshasa, Zaire, a study of 500 decedents who were serologically tested at postmortem showed that 16% of HIV-infected persons and 2% of HIV-negative persons had TB diagnosed ante mortem by smear [17].

An association between TB and AIDS is particularly striking among groups with a high prevalence of both tuberculous and HIV infections, e.g., intravenous-drug users (IVDUs) [4,18] and Haitians [2,5]. However, HIV-related TB is not restricted to IVDUs and Haitians [2,5,19]. It has been reported in homosexual and bisexual men and sexual contacts of bisexual men and in one person with transfusion-associated AIDS [19,20]. Demographically, minority populations in some areas have been at particular risk of HIV-associated TB. Detailed demographic information obtained from registry matching in New York City, Florida, and Newark, New Jersey, revealed that blacks and Hispanics accounted for 80%, 90%, and 100%, respectively, of the TB/AIDS cases [4,8,9].

The finding that TB often precedes other opportunistic diseases constituting the national surveillance definition of AIDS [2,3] was confirmed in two large studies in Florida and New York City [8,9]. In Florida, 62 (57%) of the 109 AIDS patients with histories of TB developed TB over 1 month before the diagnosis of AIDS [8]. In New York City, TB was diagnosed a median of 2 months before the AIDS diagnosis among 258 persons with both diagnoses for whom such information was available [9]. These findings suggest that latent, subclinical tuberculous infection may often progress to clinical TB early in the course of HIV-induced immunosuppression and that AIDS patients known to have developed TB may represent only a small proportion of total HIV-associated TB morbidity. Additional evidence for this possibility was gathered in Miami, where 22 (31%) of 71 consecutively tested TB patients had HIV infection, but only two met the pre-1987 case definition for AIDS [21]. Similar serosurveys have not been reported from other areas, but TB clinics are included in HIV serosurveys being implemented in 30 metropolitan areas. The information will help determine the nationwide impact of the HIV epidemic on the incidence of TB.

Clinical features

The diagnosis of TB usually precedes or coincides with the diagnosis of AIDS but may follow it [2,3,8-10,18]. The clinical presentation of TB in an HIV-infected person may

differ from that in persons with relatively normal cellular immunity who develop reactivation TB. Apical pulmonary disease with cavitation, a classic finding in immunologically normal persons, is less common. Patients may present with infiltrates in any lung zone, often associated with mediastinal and/or hilar lymphadenopathy [22]. Extrapulmonary disease occurs in 40%-75% of patients, often in the presence of pulmonary disease [2,4,8,9]. Lymphatic and haematogenous TB are especially common among persons with HIV infection [2,4]. Central nervous system (CNS) involvement, including brain abscesses, has been reported [23] and may be especially difficult to diagnose when it occurs in conjunction with other opportunistic CNS infections such as toxoplasmosis [24]. Other unusual clinical presentations have also been reported [1].

Diagnosis

These unusual clinical features emphasise the importance of considering a diagnosis of TB in persons with known or possible HIV infection and a diagnosis of HIV infection in persons with TB. Persons who provide care to HIV-infected persons must be informed of the frequently uncharacteristic presentation of TB in this group so that the diagnosis is not overlooked. Failure to diagnose and manage TB appropriately can result in the death of the patient and infection of contacts, including other patients and health-care personnel.

To establish the diagnosis, a variety of specimens, including respiratory secretions, bronchial washings, gastric lavage, lung tissue, pleural fluid, lymph node tissue, bone marrow, blood, urine, stool, brain biopsy, and cerebrospinal fluid, may need to be obtained for mycobacterial culture. Specimens must be examined microscopically, but the inability to demonstrate acid-fast bacilli and the absence of granuloma formation does not exclude the diagnosis of TB [4,19].

A Mantoux tuberculin skin test with 5 tuberculin units (TU) of tuberculin purified protein derivative (PPD) should be administered as a diagnostic aid, although some persons with HIV infection may have falsely negative reactions because of immunosuppression [2,3]. The severity of immunosuppression and the development of AIDS is related to the duration of HIV infection. Furthermore, the proportion of HIV-infected persons with TB who have negative tuberculin skin test reactions is related to the length of time between the diagnoses of TB and AIDS. In Florida, the proportion of TB patients with positive tuberculin skin tests progressively decreased with decreasing time between the two diagnoses. All five patients in whom TB was diagnosed 2 years or more before the diagnosis of AIDS had positive reactions when TB was diagnosed, 27 (63%) of 43 who had TB diagnosed 1-24 months before the AIDS diagnosis had positive reactions, and seven (33%) of 21 in whom TB was diagnosed simultaneously with or after AIDS had positive tuberculin reactions [CDC/Florida Department of Health and Rehabilitative Services, unpublished data]. In New York City, of 23 AIDS patients known to have developed TB and for whom information on the size of the tuberculin reaction was available, seven had no induration, one had a 1-4mm induration,

two had a 5-9mm induration, and 13 had an induration of 10mm or larger [CDC/New York City Department of Health, unpublished data]. Because HIV infection causes immunosuppression and the risk for TB is high in persons with both tuberculous and HIV infection, **as a general guideline, tuberculin reactions of over 5mm induration should be considered indicative of tuberculosis infection in an HIV-infected person.**

Treatment

Anti-TB chemotherapy as described below should be started whenever acid-fast bacilli are seen in a specimen from the respiratory tract of a person with HIV infection or from a person at increased risk for HIV infection whose HIV-antibody status is unknown and who declines to be tested. Because it is impossible to distinguish TB from MAC disease by any criterion other than culture (which often takes several weeks), and because of the individual and public health implications of TB, it is important to treat such patients with a regimen that is effective against *M. tuberculosis*. As a general rule, persons with TB and HIV infection respond well to standard anti-TB drugs [2,4,19], but data on clinical and bacteriologic response in these patients are limited. Longitudinal studies will help clarify the long-term outcome of these patients.

To achieve cure, the treatment period may need to be longer than the standard regimens used for TB patients without HIV infection. When HIV infection is known or suspected, the recommended drugs and dosages for adults are isoniazid, 300 mg/day, and rifampicin, 600 mg/day (or 450 mg for patients weighing 50 kg or more, and pyrazinamide, 20-30 mg/kg/day, during the first 2 months of therapy. Patients treated with rifampicin who are on methadone should have the methadone dosage increased to avoid withdrawal symptoms resulting from the interaction between the two drugs [25]. Ethambutol, 25 mg/kg/day, should be added to the initial treatment regimen for patients with CNS or disseminated TB or when isoniazid resistance is suspected. The continuation phase should always include at least isoniazid and rifampicin. Drug susceptibility tests should be performed routinely, and the treatment regimen should be revised accordingly if resistance to any of the drugs in the regimen is found. **Treatment should be continued for a minimum of 9 months and for at least 6 months beyond documented culture conversion as evidenced by three negative cultures.** In the absence of definitive data on benefits and risks, some experts suggest that, in persons with concomitant tuberculous and HIV infections, isoniazid therapy should be continued for the person's lifetime [26]. If either isoniazid or rifampicin is not or cannot be included in the regimen, therapy should last a minimum of 18 months and for at least 12 months after culture conversion. After completion of therapy, patients should be followed closely, and bacteriological examinations should be repeated if signs of TB recur.

Compliance with therapy is sometimes poor. Supervised, directly administered ambulatory therapy is successful in noncompliant patients [27] and should be initiated if noncompliance is anticipated or suspected.

Monitoring for symptoms of toxicity to anti-TB drugs may be difficult in persons with AIDS, who frequently have similar symptoms due to HIV infection, other drugs, or other conditions. At least one study has reported a higher incidence of adverse reactions to anti-TB drugs in AIDS patients [28].

Contact investigation

Persons with pulmonary TB, including those with AIDS or HIV infection, are potentially infectious until a satisfactory clinical and bacteriological response to therapy is achieved. All cases must be reported immediately to the local health department so that standard procedures for TB contact investigation can be followed [29].

In one investigation carried out by the New York City Department of Health, prevalence of tuberculin positivity (21%) among contacts of pulmonary TB patients who also had, or later developed, AIDS was not substantially different from that among contacts of comparable pulmonary TB patients with no diagnosis of AIDS (30%) [CDC/New York City Department of Health, unpublished data]. It is not known how much of this high cumulative prevalence of infection represents transmission by these index patients and how much represents prior background prevalence, but these data indicate that TB patients with HIV infection must be considered potential transmitters of *M. tuberculosis*.

Infection control

Published recommendations for preventing transmission of HIV infection and tuberculous infection to health-care workers should be followed [30-34]. Because health-care workers' risk of exposure to blood during tuberculin skin testing or injecting medication is low, wearing gloves during these procedures to prevent HIV transmission is not routinely recommended. However, used needles should not be recapped and should be disposed of according to published guidelines [31]. Recommendations for glove use during drawing of blood (e.g. for liver-function testing) have been published [31]; whether to use gloves routinely during venepuncture requires consideration of several factors.

TB should be considered in the differential diagnosis of persons with HIV infection and unexplained pulmonary symptoms, and appropriate precautions should be followed. These precautions, termed AFB isolation, are most important during and immediately after procedures that may induce coughing, such as bronchoscopy, sputum collection, aerosol induction of sputum, and administration of aerosolised medications, such as pentamidine. In clinical situations where airborne exposure of staff or other patients is likely, such procedures should be carried out in rooms or booths with negative air pressure in relation to adjacent rooms or hallways and with air exhausted directly to the outside and away from intake sources. The number of air exchanges per hour in the room or booth should be sufficient to remove infectious organisms during the time between patients. Ultraviolet lights are also useful in killing airborne tubercle bacilli [33,34]. Special care should be taken to prevent inhalation of tubercle bacilli by HIV-infected persons.

Home health-care workers, hospice volunteers, paramedics, and others who care for persons with AIDS in areas where tuberculosis infection is also prevalent should be aware of the symptoms of TB, the airborne nature of its transmission, and the appropriate precautions for their particular setting. Workers who have regular contact with TB patients should participate in a TB screening program [29,33]. Consultation on methods to reduce transmission of TB is available from state and local health department TB-control programs.

Examining persons with TB or tuberculosis infection for HIV infection

All persons with TB or tuberculous infection need to be assessed for HIV infection because the medical management of TB and tuberculous infection must be altered in the presence of HIV infection. TB patients who are infected with HIV may also develop *Pneumocystis carinii* pneumonia, cytomegalovirus pneumonitis, and other pulmonary manifestations of HIV infection as their immunosuppression progresses. Assessing these patients' response to anti-TB therapy and evaluating new infiltrates may be especially difficult. Because of the differential diagnosis and medical management of pulmonary infiltrates varies greatly between normal and immunosuppressed persons, knowledge of patients' HIV status is crucial for appropriate medical management. Providing these persons with the benefits of HIV education and counselling and providing the opportunity for HIV testing may enhance HIV prevention and control efforts. All persons with TB or tuberculous infection can benefit from receiving information about reducing their risk of acquiring or transmitting HIV infection. TB patients who are infected with HIV will also benefit by being monitored for early diagnosis of opportunistic infections and other manifestations of HIV infection. Previously published guidelines for counselling and testing and notification of sex partners and those who share needles with HIV-infected persons should be followed [35].

All patients diagnosed with TB should be offered couselling and HIV-antibody testing. Particular emphasis should be placed on offering counselling and HIV-antibody testing to persons with extrapulmonary TB and persons with TB in the age group in which most HIV infections have been found. Although these are probably some geographical areas and population groups in which most persons with TB are not likely to have HIV infection, data on the prevalence of HIV infection among TB patients in the United States are too limited to be useful in defining such populations. Furthermore, even if such data were available, there is no assurance that these populations will remain free of HIV infection in the future. Monitoring the prevalence of HIV infection among persons with TB is one method for detecting the spread of HIV infection into new areas and population groups and of assuring the appropriate management of TB in the HIV-infected patient.

While the occurrence of clinical TB may be an indication of immunosuppression related to HIV infection, the presence of a positive tuberculin skin test in a person without clinical manifestations of disease does not imply a higher likelihood of

HIV infection. Nevertheless, **behaviours* that are associated with an increased risk of prevalence or HIV infection should be routinely sought in persons with positive tuberculin skin test reactions. If HIV infection is considered a possibility, counselling and HIV-antibody testing should be strongly encouraged.** Because HIV infection is one of the strongest known risk factors for the progression of latent tuberculin skin infection to TB, **the presence of HIV infection in a person with a positive tuberculin skin test is an indication for preventive therapy regardless of that person's age.** Preventive therapy should be started only after excluding active pulmonary or extrapulmonary TB.

Persons with positive skin test reactions and factors that put them at high risk for HIV infection who decline to be tested for HIV antibody should also be considered at increased risk for developing TB. At this time, isoniazid preventive therapy should be consider for such persons on an individual basis. However, as more data become available on the prevalence of HIV infection among various population groups in different geographical areas, more definitive recommendations may be issued. Such persons should be followed closely; the patients' ability and willingness to participate in the follow-up are factors that influence the decision to provide isoniazid preventive therapy.

Some HIV-infected persons and persons who decline testing but are at high risk for HIV infection might be considered at increased risk of developing TB even if their tuberculin skin tests are negative. Thus, preventive therapy might be considered for those persons with clinical or laboratory evidence of severe immunosuppression who are from developing countries where the prevalence of tuberculous infection is very high, who have a history of close contact with an infected person, who previously have had a positive tuberculin skin test reaction, or who have a radiographic abnormality consistent with past TB.

Examining HIV-infected persons (and persons at risk for HIV infection) for the presence of TB and tuberculous infection

HIV-infected persons, with or without AIDS or other HIV-related disease, should be given a Mantoux skin test with 5 TU tuberculin, PPD. Although false-negative results may occur in these persons because of HIV-induced immuno-suppression, positive tuberculin reactions are clinically meaningful. If the skin test reaction shows induration of 5mm

* Based on seroprevalence studies, behaviours that place a person at risk for HIV infection include
1) IV-drug use; and
2) male homosexual contact.
Other factors that increase the risk for HIV infection in adults include having received blood or clotting factor concentrate between 1978 and 1985 and having had sexual relations at any time since 1978 with:
1) a person known to be infected with HIV or to have AIDS;
2) a man who has had sexual contact with another man;
3) prostitutes;
4) IVDUs; or
5) persons born in countries where most transmission of HIV is thought to occur through heterosexual sexual contact.
Risk factors for HIV infection in infants and children include:
1) parents, especially the mother, with HIV infection or any of the adult risk factors; and
2) receipt of blood or clotting factor concentrates between 1978 and 1985.

or larger a chest radiograph should be obtained, and the patient should be examined for evidence of extrapulmonary TB. If abnormalities are noted, additional diagnostic studies for TB should be undertaken. Persons with clinical AIDS or other HIV-related disease should receive a chest radiograph and be examined for evidence of extrapulmonary TB, regardless of the skin test reaction.

Some population groups may have a substantially higher prevalence of HIV infection than the total population (e.g., clients in drug-treatment programs and inmates of correctional institutions). Health-care providers should routinely provide tuberculin skin testing for persons in these settings even if counselling and HIV-antibody testing are not routinely offered or such testing is refused.

Preventive therapy for tuberculous infection

Because preventive therapy with isoniazid reduced the incidence of TB in a variety of populations with tuberculous infection, any person, regardless of age, who is HIV-infected and who has a positive tuberculin skin test reaction (induration of 5mm or more) should be offered isoniazid preventive therapy unless it is medically contraindicated. The recommended duration is a minimum of 12 months, but, analogous to considerations for the treatment of TB in AIDS patients [26], some experts have suggested prolongation of isoniazid preventive therapy beyond 12 months. Although it is not known whether isoniazid prevents TB in HIV-infected persons as effectively as in other groups, the usually positive response to standard chemotherapy in HIV-infected persons with TB suggests that isoniazid preventive therapy would also be effective.

Because of the particular by high risk for TB in persons with both HIV and tuberculous infection, ensuring completion of at least 12 months of preventive therapy is crucial.

Prevention and control of TB in drug-treatment programs for IVDUs

IVDUs require special consideration because they are at high risk for tuberculous as well as HIV infection. Tuberculin skin test surveys among heroin addicts in New York City showed that the prevalence of tuberculous infection in this population was considerably higher than in the city-wide population, even after adjustment for age, race, and economic status [36]. Even before the HIV epidemic, opiate-dependent patients in New York City had a higher prevalence of TB than did nondependent patients [37].

HIV infection among IVDUs is responsible for much of the HIV-associated increase in TB in New York City and New Jersey [4,9]. Matching TB and AIDS registries in New York City revealed that 57% of the patients with both TB and AIDS were IVDUs [9].

Isoniazid preventive therapy for tuberculin-positive IVDUs provides an opportunity to prevent many TB cases, especially in the setting of drug-treatment programs, where compliance issues can be addressed. Federal regulations require tuberculin skin testing of IVDUs before admission to a treatment program [38].

The recommended technique is the intradermal (Mantoux) test with 5 TU tuberculin PPD. Given the substantial risk for TB in this group and the potential for its prevention, drug-treatment programs should perform a skin test and record the diameter of induration on each new enrollee and on others already enrolled who have not been previously tested. Persons with a tuberculin skin test with induration of 5mm or more should be further evaluated for clinical TB and, if disease is present, treated according to current guidelines. Counselling and HIV-antibody testing should be carried out for all consenting persons with induration of 5mm or more on their tuberculin skin test, all persons with a past or present history of IV-drug use, and their sex partners [35].

If there is no clinical, radiographic, or laboratory evidence of TB, isoniazid preventive therapy should be recommended for all HIV-infected persons regardless of age with a tuberculin reaction with induration of 5mm or more. **Isoniazid preventive therapy should also be recommended for all other IVDUs with a tuberculin reaction of greater than 10mm induration regardless of age.** The rationale for this recommendation is based on epidemiological studies of HIV seroprevalence among IVDUs. Although in some geographical areas the seroprevalence of HIV is still low in IVDUs, this should not be considered a stable situation. Studies of previously collected blood samples from IVDUs indicate the potential for very rapid spread of the virus within the group. The prevalence of HIV infection among IVDUs in Manhattan, Edinburgh (Scotland), and Italy had increased to 40% 3-4 years after the virus was first introduced into the group [39]. Consequently, TB and HIV prevention programs are urgently needed for IVDUs, even in areas where the current HIV seroprevalence is very low. To ensure compliance, isoniazid therapy should preferably be fully supervised and administered (daily or on a twice-weekly basis) by the drug-treatment program staff, if possible at the same time the person is seen for treatment of IV-drug use. Patients who discontinue treatment before completing at least 6 months of uninterrupted preventive therapy should be restarted on preventive therapy after reenrollment into the treatment program. Drug-treatment programs should work closely with health department TB programs in their jurisdictions for assistance in carrying out these screening and prevention recommendations.

BCG vaccination of HIV-infected persons

The benefits and risks of BCG vaccination of HIV-infected persons remain largely undocumented. However, disseminated *M. bovis* (BCG) disease was reported in one person with AIDS and Kaposi's sarcoma who was given a BCG vaccination, presumably to 'stimulate' his immune system [40]. The U.S. ACET agrees with the recommendation of the World Health Organization that **BCG should not be administered to persons with HIV infection in countries where the risk of infection is low,** such as in the United States [41].

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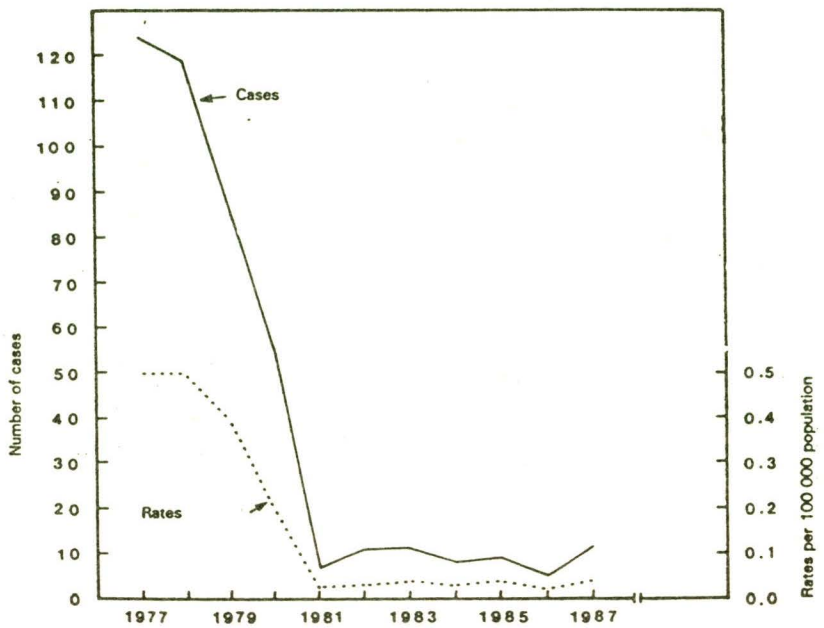
DIPHTHERIA IN CANADA: 1977-1987

(Based on Can Dis Wkly Report 1988;14:73-6)

Since national tabulation of diphtheria in Canada began in 1924, there has been a remarkable decline in both morbidity and mortality. An earlier report described the downward trend of the disease for the period 1924 to 1976 [1]. The data presented here indicate that this trend has continued (Figure 1). A very marked decline is observed from 1980 onwards. This is primarily due to a change in case definition that was introduced in the autumn of that year to exclude carriers from the case count in all provinces. Moreover, laboratory evidence suggests that the number of persons harbouring *Corynebacterium diphtheriae,* with or without symptoms, is actually decreasing [2,3]. In fact, since 1983 the intermedius biotype appears to have disappeared in Western Canada. In a highly immunised population such as Canada's, this may be due to the fact that vaccine-induced immunity, while it does not prevent carriage of the organism, almost always prevents membrane formation and coughing which decreases transmission of the organism [4]. Elimination of cutaneous diphtheria notification from 1980 onwards in the United States, is the main reason for a similar marked decline in reports of diphtheria [2].

Figure 1: Diphtheria: number of reported cases in Canada, 1977 - 1987



Demographic descriptions of cases and deaths

During the period 1977-1987 no cases were reported by the 4 Maritime provinces. A large proportion (56%) of the total cases were reported by Alberta. While the majority of the total cases (63%) were less than 15 years of age, persons over 30 accounted for 20% of all cases (Table 1). The proportion of cases under 15 years of age has remained unchanged (2/3) since 1976 [1] and is higher than that found in the United States where only half of the reported cases are under that age [3]. The majority of cases occur in autumn and winter.

Diphtheria infection in Canada is mainly associated with poor socioeconomic conditions [3]. The endemicity in North American Indians and Inuit, and indigent adults is related to this factor [2,3,5]. However, diphtheria is not restricted to these 2 groups and several episodes have been reported in city schools [6,7].

Table 1: Diphtheria: cases by age group, Canada, 1977 - 1987

Year	Total cases	Age group (years)										NS[a]
		<1	1-4	5-9	10-14	15-19	20-24	25-29	30-39	40-59	60+	
1977	124	9	33	34	14	7	4	1	4	4	3	11
1978	119	10	20	28	10	10	9	5	10	7	6	4
1979	84	4	9	29	5	9	8	5	6	6	2	1
1980	55	1	7	20	10	3	2	5	4	2	1	—
1981	7	—	1	2	1	—	2	1	—	—	—	—
1982	11	1	4	—	4	—	—	—	—	1	—	1
1983	11	—	2	2	1	1	—	—	1	—	1	3
1984	8	—	1	1	4	1	—	—	—	—	1	—
1985	9	—	—	3	1	1	1	—	2	1	—	—
1986	5	—	—	2	—	1	1	—	—	1	—	—
1987[b]	11	—	4	1	1	—	—	1	—	3	—	1
Total	**444**	**25**	**81**	**122**	**51**	**33**	**27**	**18**	**27**	**25**	**14**	**21**
Percentage of total	100	5.6	18.2	27.4	11.5	7.4	6.1	4.1	6.1	5.6	3.2	4.7
AAR[c] 1977-1987	0.16	0.62	0.51	0.63	0.24	0.13	0.10	0.07	0.06	0.04	0.04	
AAR 1981-1987 — TAM 1981-	0.04	0.04	0.12	0.09	0.09	0.03	0.02	0.01	0.01	0.02	0.01	

[a] Age not stated.
[b] Preliminary data.
[c] Average annual age-specific rates calculated using 1982 and 1984 mid-year population statistics.

Coinciding with the decline in reported cases, the number of deaths has also decreased. Five were reported during this surveillance period: 4 from British Columbia and 1 from Saskatchewan. All occurred between 1977 and 1980. Four of the 5 were males; ages ranged from 3 to 92 years. Two of 3 adult cases had underlying malignancy and 1 was a North American Indian. The case-fatality rate for the period 1977-1987 was 1%, which is lower than that reported for the previous period 1970-1977 [1]. Twenty per cent of the deaths occurred in individuals less than 10 years of age, which is similar to the proportion reported for the previous period [1]. The case-fatality rate rose with age, reaching 6% for those over 30.

Carriage, transmission and immunisation

While it is believed that the number of persons carrying *C. diphtheriae* is declining, it is also true that very few laboratories still routinely look for the organism [3]. This may reduce the chances of identifying both cases and carriers. Despite this, 3 investigations carried out during the past 7 years have reported relatively high carriage rates. The first one in the autumn of 1981 involved the contacts of 2 cases and 1 carrier from a family in Northern Ontario. Six children, all in frequent contact with each other and all fully

immunised, were identified as carriers of the same toxigenic *C. diphtheriae var gravis* strain [8]. During the second investigation, 4% of the asymptomatic contacts of a case in a Toronto school in 1985 were positive for the organism (2.5% carried toxigenic strains) despite high immunisation coverage [5]. In 1987, during the investigation of an outbreak of scarlet fever on an indian reservation in Alberta, non-toxigenic *C diphtheriae* was frequently isolated from throat swabs. **These 3 investigations illustrate once more the continuing presence of *C. diphtheriae* even in well immunised populations. Unimmunised or inadequately immunised individuals continue to be at risk of exposure and subsequent disease and, consequently, it is essential to maintain high levels of immunity.**

Adsorbed diphtheria toxoid vaccines were introduced in 1980 in British Columbia, Alberta, Saskatchewan, Manitoba and New Brunswick; in 1984 in Ontario, Nova Scotia, Prince Edward Island and Newfoundland; and in 1985 in Quebec. These vaccines have the following 2 advantages:

(1) a formulation which allows safe and effective routine immunisation of adults without Schick testing; and
(2) increased antigenicity which has extended the recommended period of time between booster doses from 5 to 10 years.

Both advantages have contributed to improved protection of the Canadian adult population against diphtheria. When the timing of the introduction of the adsorbed vaccines is considered with respect to the sharp decrease that occurred in the incidence of the disease prior to 1981, it is unlikely that these vaccines contributed to this decline. However, they probably have contributed to the maintenance of low rates since that time.

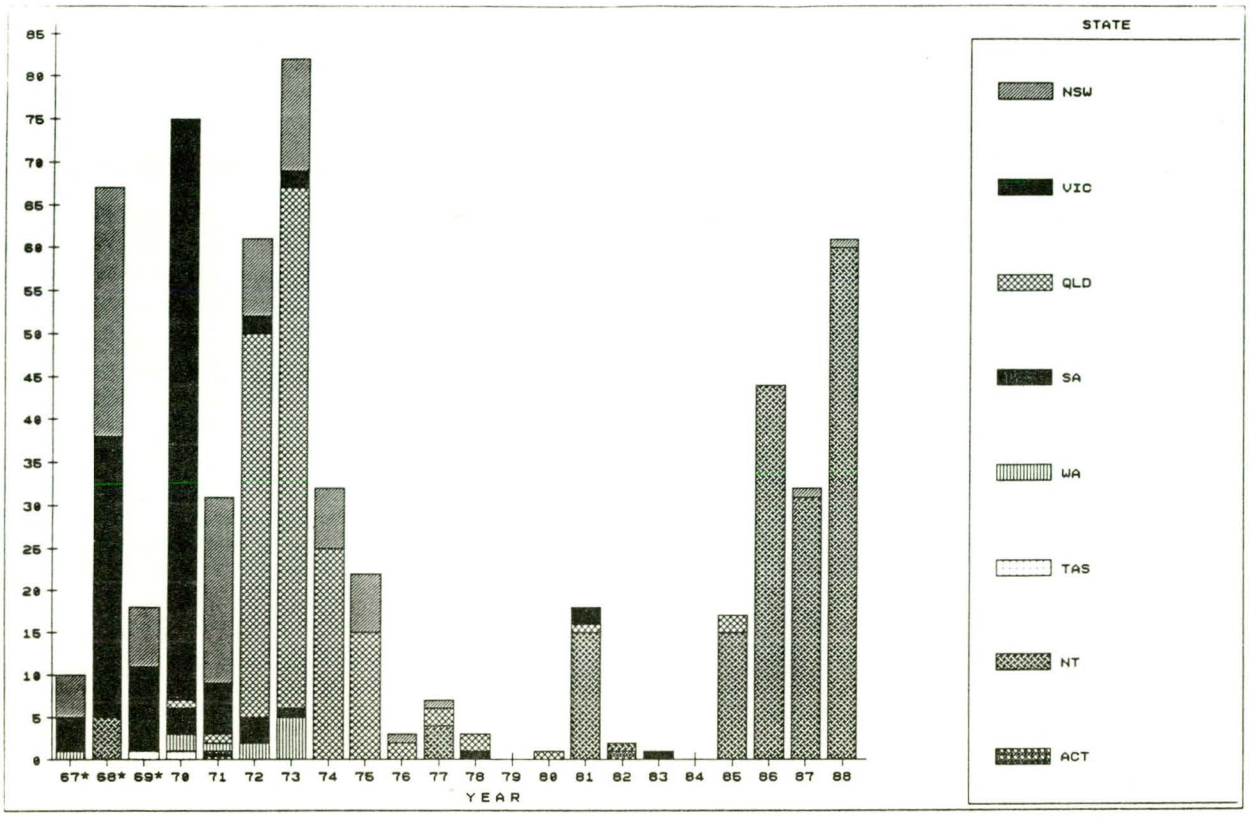
CDI Editorial Comment

Diphtheria in Australia

Diphtheria notification statistics were first available for all States and Territories in Australia in 1904. The first year in which no deaths due to diphtheria were reported was 1966; eleven deaths were notified between 1967 and 1976 [9]. Only two deaths due to diphtheria have been reported since then - one in 1977 and one in 1984 (both females)[Australian Bureau of Statistics]. Notifications of cases of diphtheria for the period 1966 to 1988 are shown in Figure 2.

The majority of notifications in the last four years have been reported from the Northern Territory. These notifications refer to mainly cutaneous infections in Aborigines in Central Australia.. Cutaneous infections are an important source of maintaining infection in communities in tropical and subtropical areas, and result in higher environmental carrier rates than do respiratory tract infections [10]. When identified, these cases are treated with penicillin to render them non-infectious to others.. Non-immune Caucasians are at most risk of clinical infection as the Aboriginal community in the area appears to have a high degree of immunity since no clinical disease has been seen in Central Australia since 1977. In addition, the vaccination coverage in Aboriginal children in contact with health authorities in Central Australia is reported as approximately 88% (3 doses) [11]. The estimated vaccination coverage for the whole of Australia is 70%

Figure 2: Diphtheria notifications, Australia - 1967-1988



* Data for 1967, 1968, and 1969 are notifications for the financial years 1966/7, 1967/8, 1968/9.

Diphtheria vaccine is available as a monovalent vaccine, combined with -tetanus vaccine (CDT and ADT) or combined with tetanus and pertussis vaccines (DTP ie triple antigen vaccine).The National Health and Medical Research Council (NH&MRC) recommends that all children be vaccinated against diphtheria using the triple antigen vaccine (or CDT vaccine if there is a contraindication to the pertussis element of the vaccine) at 2, 4, 6 and 18 months of age. In addition NH&MRC also recommends that the ADT vaccine be used to boost immunity in adults.

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(* Full reference not available).

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 13. 4 December 1988 - 31 December 1988

DISEASE	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	TOTAL	CUMULATIVE TOTAL
Amoebiasis		1							1	60
Ankylostomiasis			1		2		NN		3	35
Anthrax										
Arbovirus infection	3	6	20			39	1	NN	69	779
Brucellosis			2						2	16
Campylobacter infection	159	NN	NN	143	38	NN	24	3	367	4082
Chancroid		NN		NN		NN				4
Cholera										2
Congenital rubella syndrome			NN			NN				2
Diphtheria							5		5	61
Donovanosis		NN	12	NN	3		2	NN	17	133
Giardiasis	42	NN	NN	71	13	NN	NN		126	1753
Genital herpes	42	NN	94	NN	NN	NN	7	2	145	1895
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN		4
Gonnorhoea	59		134	14	69	2	31	2	311	3475
Hepatitis A (infectious)	5	4	9	3	19				40	599
Hepatitis B (serum)	33	24	89	7	35	1	2	2	193	1688
Hepatitis - unspecified	3	1	2	1	NN	NN			7	70
Hydatid disease		1							1	15
Lassa fever										
Legionnaires disease	7	3		4		NN	1	NN	15	68
Leprosy										20
Leptospirosis	3	7		1	1	1			13	104
Lymphogranuloma venereum		NN		NN	NN	NN		NN		
Malaria	13	3		1	4			2	23	339

DISEASE	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	TOTAL	CUMULATIVE TOTAL
Marburg disease										
Measles	1	NN	7	4	1	NN	NN		13	248
Meningococcal infections	3	3	1		4	NN	1		12	124
Non-specific urethritis	154	NN		NN	NN	NN	5	NN	159	3198
Ornithosis	4	1							5	140
Pertussis (whooping cough)	2	8	NN	8	7	NN			25	153
Plague										
Poliomyelitis										
Q fever	64		12	3					79	* 424
Rabies						NN		NN		
Salmonella infections	92	10	105	24	21	8	20		280	3482
Shigella infections	9	1	15	3	4		7		39	580
Smallpox										
Syphilis	21		140	2	7		71	1	242	2157
Tetanus	1								1	5
Trachoma		NN	NN		2		NN		2	268
Tuberculosis (all forms)	36	34	10	6	11	1	2	3	103	1164
Typhoid fever	2								2	41
Typhus (all forms)										8
Vibrio parahaemolyticus infections		NN	NN			NN		NN		2
Yellow fever										
Yersinia infections	6		NN	10	1	NN		NN	17	172

NN - Not notifiable

(Note: Data collected under the National 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 reporting by medical practitioners etc.)

* ADJUSTMENT TO THE CUMULATIVE TOTAL SINCE LAST REPORT

Q Fever	-2 Queensland	Period 12
	-1 South Australia	Period 12

CUMULATIVE TOTALS FOR PERIOD 10 SHOULD READ:

Q Fever	345
Trachoma	266
Tuberculosis	1061

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 27/4/89 TO 10/5/89

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA) PMH(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	2	0	0	0	2	1	4	9
0101	ADENOVIRUS TYPE 1	0	0	3	0	1	0	0	0	4
0102	ADENOVIRUS TYPE 2	0	0	3	0	1	0	0	0	4
0103	ADENOVIRUS TYPE 3	0	0	8	0	0	0	0	0	8
0107	ADENOVIRUS TYPE 7	0	0	1	0	0	0	0	0	1
0119	ADENOVIRUS TYPE 19	0	0	0	0	2	0	0	0	2
0130	ADENOVIRUS TYPE 30	0	0	0	0	1	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	5	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	0	2	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	5	5	1	1	0	1	13
0303	PARAINFLUENZA VIRUS TYPE 3	0	1	0	5	0	0	0	0	6
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	1	0	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	5	4	5	6	0	4	6	30
0500	RHINOVIRUS (ALL TYPES)	0	2	15	10	0	0	0	1	28
0600	MYCOPLASMA PNEUMONIAE	0	0	3	1	3	0	0	0	7
0700	ORNITHOSIS-PSITTACOSIS	0	0	3	0	0	0	0	0	3
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	1	0	1
0904	COXSACKIEVIRUS B4	0	2	0	0	0	0	0	0	2
0905	COXSACKIEVIRUS B5	0	1	2	0	0	0	0	0	3
1000	ECHOVIRUS NOT TYPED	0	1	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	1	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	1	0	0	0	1	0	0	2
1024	ECHOVIRUS TYPE 24	0	0	1	0	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	2	0	0	0	0	0	0	2
1030	ECHOVIRUS TYPE 30	0	9	2	0	11	2	0	0	24
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	3	0	0	3
1102	POLIOVIRUS TYPE 2	0	0	1	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	3	0	0	1	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	2	0	0	82	1	1	0	87
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	5	10	2	0	0	2	0	19
1303	VARICELLA-ZOSTER VIRUS	3	2	2	2	2	0	0	0	8
1306	HERPES SIMPLEX TYPE 1	1	37	23	0	2	7	1	11	82
1307	HERPES SIMPLEX TYPE 2	0	37	22	0	17	11	0	5	92
1399	HERPES VIRUS TYPING PENDING	0	1	0	1	0	0	0	0	2
1401	COXIELLA BURNETI	0	0	2	0	5	0	0	0	7
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	2	0	4	6
1522	RUBELLA VIRUS	0	1	1	0	0	0	0	0	2
1530	HEPATITIS B ANTIGEN	0	3	21	0	31	6	1	14	76
154[illegible]	HEPATITIS A ANTIBODY	0	1	[illegible]	[illegible]	[illegible]	1	1	[illegible]	[illegible]
1541	CHLAMYDIA A - C. TRACHOMATIS	14	46	32	0	21	0	1	14	128
1556	CMV - CYTOMEGALOVIRUS	0	8	2	3	0	1	3	7	24
1564	ROTAVIRUS	0	14	3	0	0	1	0	0	18
1599	ENTEROVIRUS TYPING PENDING	0	0	0	5	0	2	2	0	9
9902	ROSS RIVER VIRUS	2	54	4	1	1	3	0	15	80
TOTAL		18	238	173	46	192	44	18	83	812

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1.

PERIOD 27/4/89 TO 10/5/89

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

		1	2	3	4	7	8	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	1	0	0	4	0	0	0	6
0101	ADENOVIRUS TYPE 1	0	2	0	0	1	0	0	0	3
0102	ADENOVIRUS TYPE 2	0	3	0	0	0	0	0	0	3
0103	ADENOVIRUS TYPE 3	0	6	0	0	1	0	0	0	7
0130	ADENOVIRUS TYPE 30	0	0	0	0	1	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	2	0	0	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	0	2	0	0	0	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	11	0	1	0	0	0	0	12
0303	PARAINFLUENZA VIRUS TYPE 3	0	4	0	0	0	0	0	1	5
0399	PARAINFLUENZA VIRUS TYPING PEN	0	1	0	0	0	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	1	28	0	0	0	0	0	0	29
0500	RHINOVIRUS (ALL TYPES)	0	24	0	0	0	0	0	0	24
0600	MYCOPLASMA PNEUMONIAE	0	7	0	0	0	0	0	0	7
0700	ORNITHOSIS-PSITTACOSIS	0	3	0	0	0	0	0	0	3
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	1	1
0904	COXSACKIEVIRUS B4	0	1	0	0	1	0	0	0	2
0905	COXSACKIEVIRUS B5	1	1	0	0	1	0	0	0	3
1000	ECHOVIRUS NOT TYPED	0	0	0	0	0	0	0	1	1
1004	ECHOVIRUS TYPE 4	0	0	0	1	0	0	0	0	1
1024	ECHOVIRUS TYPE 24	0	0	0	1	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	2	0	0	0	0	0	0	2
1030	ECHOVIRUS TYPE 30	5	3	0	15	0	0	0	0	23
1100	POLIOVIRUS NOT TYPED	0	0	0	0	2	0	0	0	2
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	18	0	0	0	0	0	0	17	35
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	2	2	0	0	1	1	0	0	6
1303	VARICELLA-ZOSTER VIRUS	2	0	0	0	0	0	0	6	8
1306	HERPES SIMPLEX TYPE 1	1	3	0	0	0	0	0	61	65
1307	HERPES SIMPLEX TYPE 2	2	0	0	0	0	0	0	33	35
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	2	2
1401	COXIELLA BURNETI	1	0	0	0	0	1	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	1	0	0	5	0	0	0	6
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	2	2
1532	HEPATITIS B ANTIGEN	21	0	0	0	0	39	0	0	60
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	3	0	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	8	1	0	0	0	0	0	0	9
1556	CMV - CYTOMEGALOVIRUS	0	10	0	0	0	1	1	1	13
1564	ROTAVIRUS	0	0	0	0	17	0	0	0	17
1599	ENTEROVIRUS TYPING PENDING	0	2	1	1	3	0	0	1	8
9992	ROSS RIVER VIRUS	25	1	0	0	0	0	0	3	29
TOTAL		88	123	1	19	37	45	1	129	443

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2.

PERIOD 27/4/89 TO 10/5/89

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE 68 - 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	0	0	0	3
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	1	0	0	0	1
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	0	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	0	0	0	0	0	0	0	1	0	1
0119	ADENOVIRUS TYPE 19	2	0	0	0	0	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	1	0	0	0	0	0	0	0	0	2	3
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	0	0	0	1	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	0	0	1	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	0	1	0	1
0500	RHINOVIRUS (ALL TYPES)	0	0	1	0	0	0	0	0	1	2	4
1009	ECHOVIRUS TYPE 9	1	0	0	0	0	0	1	0	0	0	2
1030	ECHOVIRUS TYPE 30	0	0	0	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	0	1	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	2	49	0	0	0	0	0	0	1	0	52
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	10	1	2	0	0	0	0	0	13
1306	HERPES SIMPLEX TYPE 1	0	16	0	0	0	0	0	1	0	0	17
1307	HERPES SIMPLEX TYPE 2	0	57	0	0	0	0	0	0	0	0	57
1401	COXIELLA BURNETI	0	0	0	0	0	0	0	2	3	0	5
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	16	0	16
1541	CHLAMYDIA A - C. TRACHOMATIS	5	114	0	0	0	0	0	0	0	0	119
1556	CMV - CYTOMEGALOVIRUS	0	0	1	0	2	1	0	3	4	0	11
1564	ROTAVIRUS	0	0	0	0	0	0	0	1	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	1	1
9992	ROSS RIVER VIRUS	0	0	0	0	36	0	3	12	0	0	51
TOTAL		15	239	12	1	40	1	5	22	28	6	369