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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 1585 reports were received this period.

. Influenza infections - Influenza A virus (127) and influenza B virus (143) reports continued to predominate the respiratory infections. Of these reports, 13 strains of influenza A virus (H_3N_2 subtype) and one strain of influenza B virus were isolated by the State Health Laboratory, Brisbane, from a total of 31 swabs taken from pupils at a boy's boarding school; and six strains of influenza A virus were isolated by Fairfield Hospital, Melbourne, from children at a reception centre. Although many of the patients with influenza A virus infection admitted to Fairfield Hospital in July presented with severe, but uncomplicated disease, an increasing number of elderly patients had bacterial pneumonia during the second half of the month.

. Gastrointestinal infections - Two outbreaks of gastroenteritis were reported by the Institute of Clinical Pathology and Medical Research, Sydney; rotaviruses (7), adenoviruses (2) and small virus-like particles (2) were identified in 11 of 13 faecal specimens referred from the Royal Prince Alfred Hospital after a ward outbreak of neonatal gastroenteritis, and small virus-like particles were identified in a single stool specimen that was submitted following an outbreak of gastroenteritis in a fitness camp in Lismore.

. Twenty echovirus type 11 cases were reported by the State Health Laboratory Services, Perth, of which 13 were in infants aged less than one year. Fever (45%) and aseptic meningitis (30%) were the commonest presentations. A similar increase of echovirus type 11 infections occurred in 1979. The outbreak was recognized initially in Western Australia during March-May (70 reports), but spread rapidly to other States with reported peaks of infection in August in South Australia (38), November in Victoria (62) and December in New South Wales (59) and Queensland (49). Of the 670 total reports, 46% were in infants less than one year of age. An increase in echovirus type 11 infections is also being reported in the UK, with aseptic meningitis a common presentation, and some infections occurring in neonates (CDR (1982) 82/32).

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LEPROSY SURVEILLANCE AND UPDATE

A recent review estimated that there were 12-15 million cases of leprosy worldwide[1]. Leprosy has been included among the six diseases selected for the United Nations Development Program/World Bank/World Health Organisation's special program for research and training in tropical diseases.

In Australia leprosy may occur in immigrants from endemic areas, in Australians and Europeans who have spent varying time in endemic areas and Australian Aboriginals from endemic areas in northern Australia. A total of 2661 persons are registered as leprosy patients in Australia, with a distribution by State of:- Northern Territory (892 cases between 1951-1981); Western Australia (1,376); Queensland (211 with 107 in Aboriginals); Victoria (102 since 1951); New South Wales (64); South Australia (14); Tasmania (2) and Australian Capital Territory (0). Historically these statistics have been cumulative annual totals, and although each State and Territory representative has endeavoured to cull the data for patients who have died, left the State or in whom the disease has been arrested, a significant proportion of the registered cases will be quiescent and of no immediate epidemiological or operational importance. Table 1 lists the numbers of new cases of leprosy since 1970.

TABLE 1 Leprosy notifications in Australia (1970-1981)

Year	NSW	ACT	VIC	SA	TAS	WA	QLD	NT	Total
1970	2	0	0	0	0	28	2	13	45
1971	6	0	3	1	0	13	7	12	42
1972	4	0	6	1	0	10	1	17	39
1973	4	0	7	1	1	4	3	13	35
1974	4	0	3	0	0	17	5	16	45
1975	4	0	6	2	1	13	4	6	35
1976	2	0	10	2	0	21	3	6	40
1977	6	0	5	0	0	16	6	16	49
1978	6	0	3	3	0	15	8	16	51
1979	8	0	7	5	0	12	10	18	60
1980	10	0	8	1	0	9	5	7	40
1981	12	0	4	2	0	9	7	17	51

M leprae is an obligate intracellular organism with a special affinity for skin, nerve and muscle tissue. Its slow generation time of approximately 12 days results in an incubation period ranging from two to ten years or longer.

The organism has not been grown successfully in vitro, although the disease has been transmitted to animals. Laboratory animal models include the cultivation of M. leprae in the foot-pad of mice to detect viable bacilli in biopsy specimens of patients receiving chemotherapy,[2] the use of neonatally thymectomized Lewis rats for detecting live organisms among large numbers of dead M. leprae[3] and the inoculation of thymic nude mice for disseminated leprosy research[4]. In 1971 it was reported that a proportion of normal nine-banded armadillos (Dasypus novemcinctus) from southern USA developed lepromatous leprosy on inoculation with M. leprae[5]. A leprosy-like disease was later recognized amongst 15% of the indigenous armadillo population[6]. Naturally acquired leprosy has also been reported in a chimpanzee and in a mangabey monkey[7]. The eight-banded armadillos (Dasypus sabanicola) of South America and Korean chipmunks (Eutamias sibricus asiaticus) have also been infected successfully by intravenous or subcutaneous inoculation[7]. Since each 4.5 Kg armadillo can produce approximately 2.5×10^{12} bacilli

in the liver and spleen three years after infection, ample quantities of bacteria are now becoming available for research, much of which is directed at production of a vaccine[8].

Leprosy is not highly communicable, with only about 5% of spouses living with an untreated patient developing the disease. Although scientific proof for the main portal of entry for M. leprae is lacking, the accepted skin-to-skin contact transmission has been challenged in favour of the respiratory route. The number of bacilli shed from the intact skin of lepromatous patients is very small,[9] whereas the nasal discharge from these patients contains approximately 1×10^8 acid-fast organisms/ml[10]. These may survive for seven days or more in dried nasal secretions in the shade or dark[11]. Breast milk has also been suggested as a means of transmission,[12] and although viable bacilli have been recovered from arthropods 72 hours after feeding on lepromatous patients,[13] natural vector transmission has not been demonstrated. Ulcers secondary to sensory loss from peripheral neuropathy are not infectious.

The classification of a case of leprosy is important since it assists in determining the pattern of clinical response, the duration of treatment and the likelihood of reactions such as erythema nodosum. The five group system of Ridley and Jopling[14] is the widely accepted classification where the progression from the most limited form to generalized disease is as follows; - full tuberculoid (TT) - borderline tuberculoid (BT) - borderline (BB) - borderline lepromatous (BL) - full lepromatous (LL). Borderline cases usually develop toward the tuberculoid or lepromatous poles. The final outcome depends almost entirely on the cell-mediated immune response of the host. Tuberculoid leprosy occurs when there is a well-expressed cellular immunity, and presents as well-defined, non-sweating, anaesthetic skin lesions with little involvement and damage of peripheral nerves. The lepromin skin test is positive. The condition is not contagious and carries a good prognosis if adequately treated. Lepromatous leprosy is a systemic disease with bacteraemia and multiple organ involvement, and occurs when the cellular immunity is poorly expressed or absent. Skin and peripheral nerve lesions are widespread and less well-defined. The nasal mucosa is infiltrated and numerous bacilli are shed in nasal discharge. There is a strong humoral immunity, but the lepromin skin test is negative. Borderline leprosy, probably the commonest presentation, corresponds to differing grades of cellular immunity. Peripheral neuropathy is both widespread and severe. The lepromin test is usually negative.

Circulating antibodies play no role in the defence against infection, but antigen-antibody-complement reactions may occur with erythema nodosum leprosum, iridocyclitis, orchitis and arthritis complicating the disease[15].

At present there is no definitive diagnostic serological test for leprosy, although considerable progress has been made in the development of a specific soluble skin test for use in the recognition of subclinical infection[16]. More complex lymphocyte transformation, leucoyte migration inhibition and serological tests have also been developed. Early diagnosis depends on general awareness[17,18]. In endemic areas many patients are aware of the diagnosis themselves before they see a doctor. A high index of suspicion must be considered when dealing with patients who have resided in the tropics or subtropics presenting with chronic, painless skin lesions, evidence of peripheral nerve damage or rheumatic complaints. Diagnosis can be confirmed in lepromatous cases from

examination of skin smears or nasal scrapings for acid-fast bacilli. In tuberculoid leprosy bacilli are rarely found, and definitive diagnosis depends on a characteristic histological picture. Lepromin skin tests are not diagnostic of leprosy.

Dapsone (4,4'-diaminodiphenylsulphone) is the most widely used, cheapest and effective drug against leprosy. Acedapsone, a di-acetyl derivative, has been the mainstay of control in isolated Aboriginal communities since it may be given intramuscularly only once every 77 days. Of the alternative drugs, rifampicin is more expensive and has important side effects[19], and clofazimine is also expensive and causes skin pigmentation (although this has been no barrier to its use in Aborigines). About three to four months after dapsone treatment is started, leprosy bacilli from nasal washings or from serial skin biopsy specimens of lepromatous patients fail to multiply in normal mice, indicating 99.9% kill[20,21]. A similar kill with clofazimine takes about four months, whereas rifampicin is more bactericidal, achieving 99.9% kill in three to four days[22], so that patients with lepromatous disease may very rapidly be rendered no significant public health risk.

Although resistance to dapsone was suspected in the 1950's, the first clinical and experimental proof of such resistance was not reported until 1964[24]. Low dosage dapsone therapy and irregular treatment appear to predispose leprosy patients to subsequent development of dapsone resistance. In surveys conducted prior to 1976, the prevalence of secondary dapsone resistance was estimated to be 2.5/100 patients at risk in Malaysia, 3.7/100 in Israel, 10/100 in Costa Rica and about 19/100 in Ethiopia[25,26]. More recent surveys have estimated prevalences of 6.4/100 and 4.1/100 in two regions of South India and 3.5-4.0/100 in the Shanghai Municipality, China. Reports of secondary dapsone resistance have also been reported from Australia (about 3/100)[17].

When LL and BL patients relapse with secondary dapsone resistance, they can infect their contacts with dapsone-resistant M. leprae and those who subsequently develop clinical leprosy will suffer from primary dapsone resistance. Primary dapsone resistance has been identified in Cebu, Philippines (2/62 patients), Chingleput, South India (7/40) and Bamako, Mali (12/30). Cases have also been reported from Gudiyatham Taluk and Jakarta, Indonesia[25]. Because of this prevalence of primary and secondary dapsone resistance, it is now necessary to give combined therapy to all new leprosy patients, both lepromatous and tuberculoid. Rifampicin-resistant leprosy has also been reported[27].

The use of BCG vaccine may have modified epidemics but results of trials are conflicting[23]. In Australia it is recommended that all children in endemic areas should be given the vaccine as early as possible within the first eight months of life, as should any Mantoux-negative child contacts of infectious cases.

The production of large amounts of purified M. leprae in the armadillos has paved the way for a leprosy vaccine. The organism can be purified almost free of host tissue without damaging bacillary antigens, and studies with a vaccine consisting of irradiated, killed M. leprae in combination with live BCG have suggested a capacity to induce cell-mediated immunity in lepromin negative contacts and patients with very early indeterminate leprosy[16]. Future trials will be conducted in healthy volunteers in Europe and America[28]. Nevertheless, since the development of a vaccine will take 10-15 years because of the implications of long incubation periods and immunodeficiency in lepromatous patients, emphasis on chemotherapy research remains.

A list of references used for this article is available from the Editor.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 1

REPORTING PERIOD - 5/8/82 - 18/8/82 BULLETIN NUMBER 82/17

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED	26					1	13	3	43
0101 ADENOVIRUS TYPE 1	1					1			2
0102 ADENOVIRUS TYPE 2	3					4			7
0103 ADENOVIRUS TYPE 3						1		3	4
0105 ADENOVIRUS TYPE 5	1		1			4			6
0106 ADENOVIRUS TYPE 6		1				1			2
0112 ADENOVIRUS TYPE 12						1			1
0119 ADENOVIRUS TYPE 19				3					3
0199 ADENOVIRUS TYPING PENDING			4	1	5	6			16
0201 INFLUENZA A VIRUS	9	2	2	10		4		2	29
0202 INFLUENZA A VIRUS SUBTYPE H3N2	1	1		36	44	2	14		98
0203 INFLUENZA B VIRUS	35	8	1	35	13	14	30	7	143
0301 PARAINFLUENZA VIRUS TYPE 1	1	1		1		2		1	6
0302 PARAINFLUENZA VIRUS TYPE 2								2	2
0303 PARAINFLUENZA VIRUS TYPE 3	1	2			1		1		5
0399 PARAINFLUENZA VIRUS TYPING PENDING						1	1		2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	20	19	2	42	44	16	2	12	157
0500 RHINOVIRUS (ALL TYPES)	4			4	2	1	8	5	24
0600 MYCOPLASMA PNEUMONIAE	63		2	2	1	8	6	4	86
0700 ORNITHOSIS-PSITTACOSIS	2								2
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED							2	2	4
0809 COXSACKIEVIRUS A9				1					1
0816 COXSACKIEVIRUS A16						1			1
0901 COXSACKIEVIRUS B1	1								1
0903 COXSACKIEVIRUS B3						1			1
0905 COXSACKIEVIRUS B5	1							1	2
1007 ECHOVIRUS TYPE 7		1							1
1009 ECHOVIRUS TYPE 9						1			1
1011 ECHOVIRUS TYPE 11								16	16
1013 ECHOVIRUS TYPE 13	1						1		2
1021 ECHOVIRUS TYPE 21	1								1
1101 POLIOVIRUS TYPE 1							1	1	2
1102 POLIOVIRUS TYPE 2								1	1
1103 POLIOVIRUS TYPE 3						2			2
1104 POLIOVIRUS-VACCINAL STRAIN	2								2
1200 MUMPS VIRUS	33			1		1	4	8	47
1300 HERPES VIRUS GROUP-NOT TYPED	38		2			11		1	52
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2		1			1	60	64
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5	1						2	8
1303 VARICELLA-ZOSTER VIRUS			1	1		2		1	5
1306 HERPES SIMPLEX TYPE 1	12		8	31		21	15		87
1307 HERPES SIMPLEX TYPE 2	98		7	31		18	32		186
1399 HERPES VIRUS TYPING PENDING			4	1	3	2			10
1401 COXIELLA BURNETI	33					3	9		45
1502 PICORNA VIRUS-NOT TYPED								1	1
1514 MOLLUSCUM CONTAGIOSUM						1		1	2
1521 MEASLES VIRUS	6			4			3		13
1522 RUBELLA VIRUS				5			1		6
1532 HEPATITIS B ANTIGEN	13		5	27		12	5	9	71
1535 HEPATITIS A ANTIBODY	7					7	2	6	22
1541 CHLAMYDIA A - C TRACHOMATIS	37		2			1		42	82
1556 CMV - CYTOMEGALOVIRUS	21		1	22	7	8	4	3	66
1562 REOVIRUS (ALL TYPES)						1			1
1564 ROTAVIRUS	18	11	13		9	18	5	6	80
1599 ENTEROVIRUS TYPING PENDING		1	7		4				12
ROSS RIVER VIRUS	2			14			5	1	22
SMALL VIRUS (LIKE) PARTICLE	9					1			10
DENGUE							17		17
Total	505	50	62	273	133	179	182	201	1,585

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 5/8/82 to 18/8/82 .... 82/17

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	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0100 ADENOVIRUS NOT TYPED		1									
0101 ADENOVIRUS TYPE 1	1	1									
0102 ADENOVIRUS TYPE 2		3					4				
0103 ADENOVIRUS TYPE 3	1	2									
0105 ADENOVIRUS TYPE 5		3					3				
0106 ADENOVIRUS TYPE 6		2									
0112 ADENOVIRUS TYPE 12		1									
0199 ADENOVIRUS TYPING PENDING		1									
0201 INFLUENZA A VIRUS	1	16						1			
0202 INFLUENZA A VIRUS SUBTYPE H3N2	1	93		2		1	1				2
0203 INFLUENZA B VIRUS	1	119	1	2		2		1	2		3
0301 PARAINFLUENZA VIRUS TYPE 1		4		1							
0302 PARAINFLUENZA VIRUS TYPE 2		2									
0303 PARAINFLUENZA VIRUS TYPE 3		6									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	4	144	1						1		
0500 RHINOVIRUS (ALL TYPES)	2	20									
0600 MYCOPLASMA PNEUMONIAE	25	52	1				1		1		
0700 ORNITHOSIS-PSITTACOSIS		1									
0809 COXSACKIEVIRUS A9		1									
0816 COXSACKIEVIRUS A16											1
0901 COXSACKIEVIRUS B1							1				
0905 COXSACKIEVIRUS B5		1				1					
1007 ECHOVIRUS TYPE 7							1				
1009 ECHOVIRUS TYPE 9	1										
1011 ECHOVIRUS TYPE 11		4		6		1					1
1013 ECHOVIRUS TYPE 13							1				
1021 ECHOVIRUS TYPE 21		1									
1101 POLIOVIRUS TYPE 1	1	1									
1102 POLIOVIRUS TYPE 2		1									
1103 POLIOVIRUS TYPE 3							2				
1104 POLIOVIRUS-VACCINAL STRAIN	1										
1200 MUMPS VIRUS	10		2	10							
1301 HERPES SIMPLEX VIRUS NOT-TYPED		3									39
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	1	1									
1303 VARICELLA-ZOSTER VIRUS	1										4
1306 HERPES SIMPLEX TYPE 1	1	12		1							35
1307 HERPES SIMPLEX TYPE 2		1									12
1401 COXIELLA BURNETI	16	2									
1502 PICORNA VIRUS-NOT TYPED				1							
1514 MOLLUSCUM CONTAGIOSUM											2
1521 MEASLES VIRUS	2	1	1	1							8
1522 RUBELLA VIRUS	2										
1532 HEPATITIS B ANTIGEN	33							37			
1535 HEPATITIS A ANTIBODY	2							18			
1556 CMV - CYTOMEGALOVIRUS	18	16						3	1	7	3
1562 REOVIRUS (ALL TYPES)							1				
1564 ROTAVIRUS		1					82				
ROSS RIVER VIRUS	3										15
SMALL VIRUS (LIKE) PARTICLE							9				
DENGUE	3										9
Total	131	517	6	24		5	106	60	5	7	135

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 3

PERIOD : 5/8/82 to 18/8/82 ... 82/17

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
0103 ADENOVIRUS TYPE 3.....	1						1			
0119 ADENOVIRUS TYPE 19.....	3									
0201 INFLUENZA A VIRUS.....	1						1	8	1	
0202 INFLUENZA A VIRUS SUBTYPE H3N2					1		3	7		1
0203 INFLUENZA B VIRUS.....					4		4	29	2	1
0302 PARAINFLUENZA VIRUS TYPE 2....								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								5		
0500 RHINOVIRUS (ALL TYPES).....							1	1		
0600 MYCOPLASMA PNEUMONIAE.....			1				2	3	1	
0700 ORNITHOSIS-PSITTACOSIS.....								1		
0903 COXSACKIEVIRUS B3.....										1
1011 ECHOVIRUS TYPE 11.....								10		1
1013 ECHOVIRUS TYPE 13.....								1		
1104 POLIOVIRUS-VACCINAL STRAIN....										1
1200 MUMPS VIRUS.....			24		1			4	1	
1301 HERPES SIMPLEX VIRUS NOT-TYPED		26								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			3				1		2	
1306 HERPES SIMPLEX TYPE 1.....	6	27						3		
1307 HERPES SIMPLEX TYPE 2.....		174								
1401 COXIELLA BURNETI.....							9	19	2	
1521 MEASLES VIRUS.....			1					1		
1522 RUBELLA VIRUS.....						3		1		
1535 HEPATITIS A ANTIBODY.....								2	1	
1541 CHLAMYDIA A - C TRACHOMATIS...	3	77								
1556 CMV - CYTOMEGALOVIRUS.....		4				6	3	2	4	1
ROSS RIVER VIRUS					4			1		
SMALL VIRUS (LIKE) PARTICLE									1	
DENGUE					4			7		
Total.....	14	308	29		14	9	25	106	15	6