



Communicable Diseases Intelligence

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TYPHOID FEVER - TASMANIA - Typhoid fever has been confirmed in a 40 year old man from Preolenna, Tasmania. He was admitted to North West General Hospital on 3 September with diarrhoea and symptoms suggestive of influenza. He had recently returned from a one month overland trip through Java with his wife and two children. None of the family had had typhoid immunisation. A positive diagnosis of S. typhi has also been made on one of the daughters (aged eight years), but to date the mother and second daughter have shown no symptoms.

VIRUS REPORTING SCHEME - 1038 reports have been received this period. Patterns indicated by these reports include an increase in influenza A infections - 72 reports received compared with 47, 34 and 15 for the previous three periods.

Reports of interest include:

- The three reports of arbovirus group B, all clinically dengue, were from patients who had visited Bali. The dengue confirmation was from a patient who had recently returned from Manila.

IMPORTED PARASITIC INFECTIONS IN TASMANIA (continued from page 6)

and a wide range of species has been recorded - emphasizing the need for clinicians and laboratories seriously to consider the possibility of these infections in patients who have travelled abroad - often years previously. In Tasmania, these parasitic infections pose mostly a diagnostic and therapeutic problem - the lack of suitable vectors and unsuitable climatic conditions not being conducive to their establishment as endemic problems. However in many parts of Mainland Australia, suitable climatic conditions and the occurrence of potential vectors might, if care is not taken, allow the establishment and re-establishment of certain of these infections.

References:

1. MJA (1979) 2:338
2. MJA (1980) 1:667
3. MJA (1980) 2:160
4. MJA (1978) 2:286
5. Aust. Microbiologist (1980) 1:No.4

MALARIA SURVEILLANCE - AUSTRALIA

(Based on information supplied by Prof. R.H. Black, Central Registry of Malaria Cases, Commonwealth Institute of Health, Sydney.)

There has been a world-wide resurgence of malaria in recent years, and the World Health Organisation (WHO) has expressed concern at the prospect of this disease spreading into areas where it was previously endemic and the receptivity remains high. The situation is being exacerbated by the emergence of drug resistance in the malaria parasite and the development of insecticide resistance in the vector mosquitoes. Both these factors are of particular concern to Australia since our immediate neighbours, particularly Papua New Guinea, West Irian and the Solomon Islands, are countries where malaria is hyperendemic.

Australia is in the maintenance phase of malaria eradication, and the country north of the 19°S parallel remains receptive to malaria. The malaria surveillance program in Australia is carried out by the Departments of Health in the receptive areas (Queensland, Northern Territory and Western Australia), and is coordinated by the Commonwealth Department of Health through the Central Registry of Malaria Cases at the Commonwealth Institute of Health (formerly School of Public Health and Tropical Medicine).

In 1978, 320 cases of malaria were imported into Australia from abroad, 200 of them from Papua New Guinea. There were also three reports of induced malaria, two congenital, and one from intravenous heroin administration.

Plasmodium vivax was the most common infection (240 reports), followed by P. falciparum (75), P. malariae (4) and P. ovale (1). There were two mixed infections with P. vivax and P. falciparum, and one that was not identified. Of the 200 cases originating from Papua New Guinea, 66 were infections with P. falciparum, and 35% of these were strains resistant to chloroquine.

In 1979, 472 imported cases of malaria were diagnosed, of which 274 originated from Papua New Guinea. Already from 1 January to 7 August 1980, 368 malaria cases have been detected. In the first half of 1980, 120 of the 297 cases entered in the Central Register were in Vietnamese refugees most of whom had been in transit in Indonesia. The majority of these cases were infections with P. vivax (112 reports), with five cases of P. falciparum and two reports of mixed P. vivax and P. falciparum infection. All refugees are accommodated outside the malaria receptive area as a precautionary measure.

There have been no indigenous cases of malaria in Australia since 1973 when there was a small outbreak in Torres Strait. However, there were introduced cases of P. vivax at Bamaga, on the Cape York Peninsula in February 1977, and P. falciparum malaria was recognised on Moa Island in the Torres Strait early this year. (CDI 80/2).

NON-A/NON-B HEPATITIS

(Based on WER (1980) 55:249, and abstracts from the Second Australian Symposium on Viral Hepatitis, Melbourne, March 1980.)

Rapid development in our knowledge of hepatitis A (HAV) and

hepatitis B (HBV) virus infections and their associated virus-specific antigens and antibodies, coupled with the availability of specific serological tests for the detection of Epstein-Barr virus (EBV) and cytomegalovirus (CMV) infection, have allowed the detection of non-A/non-B viral hepatitis (NANBH) as a diagnosis of exclusion.

Although the incidence of post-transfusion HBV has been significantly reduced in recent years by the institution of routine screening tests for hepatitis B antigen (HBsAg) in donor blood, the incidence of post-transfusion NANBH has not been similarly diminished. In Europe up to 80%, and in the USA more than 90%, of post-transfusion hepatitis is NANBH. Statistics for hospital populations with hepatitis are 64% HBV, 19% HAV and 17% NANBH.

In a study at Fairfield Hospital, Melbourne, it was found that of 1006 patients with acute icteric hepatitis admitted in the two year period to the end of June 1979, 50% were found to have HBV, 29% HAV, and 21% NANBH. Epidemiologically, NANBH closely resembled hepatitis B in that more than 80% of the cases occurred in adolescents and young adults, the majority of whom were intravenous drug users. The disease was rare in children. The prodromal symptoms of the age group 15-19 years resembled those of HBV, with a low incidence of fever and a common occurrence of arthralgia, but differed in that rashes were rare. The icteric phase of the illness was almost identical to that of HAV. NANBH was not found in homosexuals, nor related to tattooing, and was not seen in dialysis units.

A second study by Y. Cossart in Sydney involved the follow-up of 214 patients admitted to two cardiac surgery units. Four patients developed post-transfusion hepatitis, which was assumed to be NANBH by diagnosis of exclusion.

Although the bulk of NANBH cases can be linked to the prior transfusion of blood or blood products, a significant number of cases do not appear to be transfusion associated. These "sporadic" cases of NANBH can account for up to 15-25% of all adult viral hepatitis in some areas. NANBH occurs frequently in clinical or institutional settings favouring the percutaneous transmission of etiological agent/s, including haemodialysis, heart or renal transplantation, illicit drug use and close personal contact.

As with HBV, chronic infection may follow an acute phase of infection with NANBH, and studies suggest that as many as 70% of the NANBH patients who are drug addicts, and up to 50% of NANBH patients who have been transfused, develop histopathological sequelae. Studies involving the efficacy of immune serum globulin in the prevention of post-transfusion hepatitis have given contradicting results, although an overall reduction of icterus is evident.

Human NANBH has been transmitted experimentally to chimpanzees, where incubation periods to the first significant elevation in transaminase activity range from approximately two weeks to 14 weeks. These animal studies have conclusively demonstrated the lack of serological identity

between the presumed viral agent/s of NANBH and HAV, HBV, CMV or EBV. Like humans, chimpanzees also appear to develop chronic NANBH, with the plasma from one infected chimpanzee remaining infectious for 16 months.

Clinical, epidemiological and experimental evidence support the existence of two or more agents of NANBH. Clinical evidence derives from the observation that multiple bouts of acute NANBH can occur in single patients. Epidemiologically, "short-incubation" and "long-incubation" forms of NANBH have been described, and cross-challenge studies in chimpanzees have provided experimental evidence for the existence of two distinct NANBH agents. Electron microscopic evidence for the existence of two distinct "strains" of NANBH agents has come from several laboratories. One "strain", which breeds true upon passage in chimpanzees, induces the formation of unique cytoplasmic structures in the hepatocytes of infected animals. The other "strain" is associated with changes in hepatocyte nuclear morphology with particulate intranuclear structures, 20-30mm in diameter. However, challenge experiments with these strains have shown no superinfection capabilities, indicating a high degree of identity.

Virus-like particles of approximately 27mm diameter which induce NANBH have been seen in both the serum and acute phase liver of chimpanzees. Other investigators have reported the recovery of 60mm particles with a 40mm core, as well as particles morphologically similar to HBV from NANBH infected animals. These, and other non-corroborated results have shown that the virology of NANBH is still in its infancy. The serological response to NANBH is also an enigma. Test procedures including agar gel diffusion, counterimmunoelectrophoresis, radio-immune assay and immunofluorescence have all proved unreliable. This current difficulty in defining specific confirmable antigen and antibody systems related to NANBH may be a consequence of (1) low antigen (virus) titre in blood or other tissues, (2) low potency or avidity reagent antibodies, (3) the formation of antigen-antibody complexes in vivo or in vitro.

SALMONELLA SURVEILLANCE - WESTERN AUSTRALIA 1979

(Based on reports from J.B. Iveson, Public Health Enteric Diseases Unit, State Health Laboratory Services, Western Australia.)

Although a summary of the human salmonella infections in Australia for 1979 and the first quarter of 1980 was reported in the previous bulletin (CDI 80/17), the detailed surveillance undertaken in Western Australia deserves further mention.

During 1979, a total of 8857 salmonella isolations comprising 96 serotypes were recorded from either human, foodstuffs, animal, or environmental sources throughout Western Australia. A total of 1627 salmonella infections in humans were diagnosed, and of these 58 individuals were infected with more than one serotype. A total of 67 serotypes was implicated in human infections, although two serotypes, S. muenchen and S. typhimurium were responsible for 1145 (67%) of cases.

In cases where information was supplied, the incidence of salmonella

infection was equal for both sexes, although 38% were children aged less than five years.

Sixty-five per cent of cases were diagnosed in the summer months. This increase was mainly due to the occurrence of a large scale foodborne outbreak of S. muenchen (CDI 79/4). The outbreak was traced to the sale of contaminated chicken, and during the period of investigation, 185 individuals (90% of them children under 6 years) were hospitalised. Intensive follow-up investigation of employees engaged in poultry processing, retail handling, and family contacts revealed a further 123 cases. Only eleven cases of S. muenchen infection had occurred in Perth in each of the two previous years, although the serotype is commonly associated with cases of gastroenteritis in the more remote northern area of the State, where it is also prevalent in wild marsupials and reptiles.

S. agona, a serotype first detected among Asian immigrants during 1977, was responsible for a small salmonella outbreak in a Perth maternity hospital. Other "exotic" serotypes isolated from humans were probably contracted in Asian countries, particularly the popular Bali Island tourist resort.

S. muenchen and S. typhimurium also predominated in foodstuffs, domestic animals, effluent and sewage (although S. muenchen was rarely isolated from abattoir effluents that were not processing poultry).

A more detailed report of the surveillance program for Western Australia is available from the Editor.

IMPORTED PARASITIC INFECTIONS IN TASMANIA

(Contributed by J.M. Goldsmid, Department of Pathology, University of Tasmania, Hobart. Based on the paper read at A.S.M.-N.Z.M.S. Combined Scientific Meeting, Dunedin, 1980.)

International travel throughout the world has increased dramatically over the last few years. In 1966, 139 million visitor arrivals were recorded world-wide, while by 1976, this number had increased to 221 million. Australia has also experienced a spate of overseas travel, recording a rise in international visitor arrivals of 66018 in 1957 to 556625, of whom 33.4% were from tropical countries, in 1977. The number of Australians departing for overseas has also increased remarkably - from 413917 in 1971 to 973799 in 1976. Tropical destinations comprised of 50.2% and 32% of these itinerants respectively.

Of all the Australian States, Tasmania is, from an international travel stand-point, probably the most isolated. It has been estimated that only 10% of all foreign visitors to Australia include a visit to Tasmania in their itinerary. However, despite this comparative paucity of international travellers when compared to mainland Australia, Tasmania has recorded a significant number of "exotic" parasitic infections over the last few years.

The infections recorded, together with their country of origin are given overleaf.

Imported Parasitic Infections Recorded in Tasmania

Parasite species	No. cases	Country of origin
<u>Entamoeba histolytica</u>	4	Asia; S.E. Asia; India
<u>Ent. coli</u>	2	India
<u>Ent. hartmanni</u>	3	India; Bangladesh
<u>Endolimax nana</u>	3	Viet Nam; India; Bangladesh
<u>Iodamoeba bütschlii</u>	1	Viet Nam
<u>Giardia lamblia</u>	4	Asia; India
<u>Plasmodium vivax</u>	7	P.N.G.; India; Solomon Is.
<u>Schistosoma mansoni</u>	2	Zimbabwe; Kenya
<u>S. haematobium</u>	1	Zimbabwe
<u>Hymenolepis nana</u>	1	Bangladesh
Hookworm	3*	Viet Nam; N. Africa
<u>Trichuris trichiura</u>	3	Asia; Maldive Is.; Bangladesh
<u>Ascaris lumbricoides</u>	5	P.N.G.; Maldive Is.; Zimbabwe; S.E. Asia; Bangladesh.
<u>Strongyloides stercoralis</u>	3	Timor; P.N.G.
<u>Loa loa</u>	1	W. Africa
Tropical eosinophilia (? <u>Wuchereria bancrofti</u>)	1	Maldive Is.

*Both Ancylostoma duodenale and Necator americanus identified on Marada-Mori culture.

A number of points emerge from this investigation.

1. Although many of the people were infected with commensal species or carried light and subclinical loads, some cases were clinically severe (e.g. severe hookworm anaemia with an egg output of 21600 eggs/g faeces⁽¹⁾; a bilharzial case with symptoms suggestive of Katayama syndrome⁽¹⁾; amoebic liver abscess⁽¹⁾ and a relatively severe case of tropical pulmonary eosinophilia⁽²⁾).
2. Some of the infections were of long standing (e.g. amoebiasis acquired 10 years previously; strongyloidiasis of 35 years duration⁽³⁾ and hookworm of 7 years duration).
3. In the series above, of 27 infected patients, 21 (77.8%) were Australian residents who had acquired their infections while abroad and only six (22.2%) of the infections were brought to Tasmania by visitors or immigrants from the tropics.

This latter point is of interest as in Tasmania, parasitic infections in humans, with the exceptions of Giardia lamblia, Enterobius vermicularis and in the past hydatid, are uncommon, and thus in this State at least, parasitic infections can be classed as "diseases of affluence" found in those financially able to travel abroad, rather than the "diseases of poverty" that they are usually regarded as. It is noteworthy however, that the situation in regard to the ratio of Australian residents:Non Australian residents infected, might be quite different in States receiving large numbers of S.E. Asian refugees^(4,5).

To conclude, imported exotic parasitic infections are not rare in Tasmania,

(continued on page 1.)

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

1

REPORTING PERIOD - 21-8-80 - 3-9-80 BULLETIN NUMBER

80/18

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.....	3		1			5	5	2	16
0101 ADENOVIRUS TYPE 1.....						6		2	8
0102 ADENOVIRUS TYPE 2.....	1		1	5		2		1	10
0103 ADENOVIRUS TYPE 3.....								1	1
0105 ADENOVIRUS TYPE 5.....						1		1	2
0106 ADENOVIRUS TYPE 6.....						1			1
0107 ADENOVIRUS TYPE 7.....				1		1			2
0108 ADENOVIRUS TYPE 8.....								1	1
0110 ADENOVIRUS TYPE 10.....								1	1
0119 ADENOVIRUS TYPE 19.....								9	9
0128 ADENOVIRUS TYPE 28.....						1			1
0199 ADENOVIRUS TYPING PENDING.....		1	1		5				7
0201 INFLUENZA A VIRUS.....	1		4			17	5	11	38
0202 INFLUENZA A VIRUS SUBTYPE H3N2.....	1			9	9		15		34
0203 INFLUENZA B VIRUS.....	4		1			4	4		13
0301 PARAINFLUENZA VIRUS TYPE 1.....						2		2	4
0302 PARAINFLUENZA VIRUS TYPE 2.....						1	1		2
0303 PARAINFLUENZA VIRUS TYPE 3.....		7		1	5	7	4	3	27
0400 RESPIRATORY SYNCYTIAL VIRUS (RS) ...	27	22	4	11	34	72	9	5	184
0500 RHINOVIRUS (ALL TYPES).....	2			1	3		4	1	11
0600 MYCOPLASMA PNEUMONIAE.....	1	1	2	1		1	2		8
0700 ORNITHOSIS-PSITTACOSIS.....	1								1
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....							1		1
0809 COXSACKIEVIRUS A9.....			1			3			4
0816 COXSACKIEVIRUS A16.....							1		1
0901 COXSACKIEVIRUS B1.....						1			1
0902 COXSACKIEVIRUS B2.....									1
1009 ECHOVIRUS TYPE 9.....						1			1
1011 ECHOVIRUS TYPE 11.....				1					1
1022 ECHOVIRUS TYPE 22.....				1					1
1030 ECHOVIRUS TYPE 30.....				6					6
1099 ECHOVIRUS TYPING PENDING.....			1						1

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

2

REPORTING PERIOD - 21 - 8 - 80 - 3 - 9 - 80 BULLETIN NUMBER . 80/18
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

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
1101 POLIOVIRUS TYPE 1.....		1				4			5
1102 POLIOVIRUS TYPE 2.....						1	1		2
1103 POLIOVIRUS TYPE 3.....		2				1	1		4
1104 POLIOVIRUS-VACCINAL STRAIN.....					2	2			4
1200 MUMPS VIRUS.....	3	1		2		1		1	8
1300 HERPES VIRUS GROUP-NOT TYPED.....	5	1				2	1		9
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....	8			2			27	36	73
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	3				1				3
1303 VARICELLA-ZOSTER VIRUS.....	1	1	1		1				4
1306 HERPES SIMPLEX TYPE 1.....	5		2	14		4			25
1307 HERPES SIMPLEX TYPE 2.....	35		3	21		28			87
1399 HERPES VIRUS TYPING PENDING.....			1		2	5			8
1401 COXIELLA BURNETI.....	7			11		3	19		40
1502 PICORNA VIRUS-NOT TYPED.....								5	5
1514 MOLLUSCUM CONTAGIOSUM.....						2		1	3
1521 MEASLES VIRUS.....			3				1		4
1522 RUBELLA VIRUS.....	6	1		3		1	2	1	14
1530 HEPATITIS A VIRUS.....						2		16	18
1531 HEPATITIS B VIRUS.....				23		7		6	36
1532 HEPATITIS B ANTIGEN.....			9				5		14
1541 CHLAMYDIA A - TRIC TYPE.....	10		3			3		46	62
1556 CMV - CYTOMEGALOVIRUS.....	2		3	23	4	4	6	2	44
1564 ROTAVIRUS.....	48		12		6	22		30	118
1599 ENTEROVIRUS TYPING PENDING.....		1	3		14	6			24
ROSS RIVER VIRUS							2		2
ASTROVIRUS	8								8
SMALL VIRUS (LIKE) PARTICLE	5				4	2			11
DENGUE							1		1
ARBO. GROUP B.				3					3
Total.....	187	39	56	139	90	226	117	184	1038

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 21/8/80 to 3/9/80 80/18 3
 Viral Identifications by Clinical Information Table 1.
 Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Enceph-
 alitis; 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	6					9				
0101 ADENOVIRUS TYPE 1.....	1					1	6				
0102 ADENOVIRUS TYPE 2.....		5					2				
0103 ADENOVIRUS TYPE 3.....	1										
0105 ADENOVIRUS TYPE 5.....	1			1							
0106 ADENOVIRUS TYPE 6.....							1				
0107 ADENOVIRUS TYPE 7.....		1									
0110 ADENOVIRUS TYPE 10.....	1										
0119 ADENOVIRUS TYPE 19.....	5										
0128 ADENOVIRUS TYPE 28.....		1									
0201 INFLUENZA A VIRUS.....	1	34				1			1	1	2
0202 INFLUENZA A VIRUS SUBTYPE H3N2		21		1							1
0203 INFLUENZA B VIRUS.....		12									1
0301 PARAINFLUENZA VIRUS TYPE 1....		4									
0302 PARAINFLUENZA VIRUS TYPE 2....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....		27									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	3	175				1					
0500 RHINOVIRUS (ALL TYPES).....		10									
0600 MYCOPLASMA PNEUMONIAE.....	2	4									
0700 ORNITHOSIS-PSITTACOSIS.....	1										
0809 COXSACKIEVIRUS A9.....		1				1	2				
0816 COXSACKIEVIRUS A16.....											1
0901 COXSACKIEVIRUS B1.....							1				
0902 COXSACKIEVIRUS B2.....											
1009 ECHOVIRUS TYPE 9.....				1							
1011 ECHOVIRUS TYPE 11.....				1							
1022 ECHOVIRUS TYPE 22.....							1				
1030 ECHOVIRUS TYPE 30.....				5			1				

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

4

PERIOD : 21 / 8 / 80 to 3 / 9 / 80 80/18

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.-CONTINUED

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/ muc memb
1101 POLIOVIRUS TYPE 1.....		1					4				
1102 POLIOVIRUS TYPE 2.....		1					1				
1103 POLIOVIRUS TYPE 3.....		2					1		1		
1104 POLIOVIRUS-VACCINAL STRAIN.....							1				
1200 MUMPS VIRUS.....	3	1		1							
1300 HERPES VIRUS GROUP-NOT TYPED..		1									4
1301 HERPES SIMPLEX VIRUS NOT-TYPED	4	1		1							28
1302 EPSTEIN-BARR VIRUS (EB VIRUS) -	1										
1303 VARICELLA-ZOSTER VIRUS.....	1	1				1					1
1306 HERPES SIMPLEX TYPE 1.....		2	1								11
1307 HERPES SIMPLEX TYPE 2.....											6
1401 COXIELLA BURNETI.....	8	4									
1502 PICORNA VIRUS-NOT TYPED.....		1		1			2				
1521 MEASLES VIRUS.....						1					2
1522 RUBELLA VIRUS.....											12
1530 HEPATITIS A VIRUS.....	2						1	15			
1531 HEPATITIS B VIRUS.....	14	1						21			
1532 HEPATITIS B ANTIGEN.....								14			
1556 CMV - CYTOMEGALOVIRUS.....	9	3						1		1	
1564 ROTAVIRUS.....	6						109				
ASTROVIRUS							8				
SMALL VIRUS (LIKE) PARTICLE	4						7				
Total.....	69	32	1	12		6	158	51	2	2	69

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 21/8/80 to 3/9/80 ... 80/18
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 ...

-CONTINUED

VIRUS OR VIRAL ANTIGEN	Eye	Gen-ital	Endo/sal gland	RES	Muscle/joint	Con-genital	PUO	Fever/malaise	Other	SIDS
1556 CMV - CYTOMEGALOVIRUS.....		2		2	1	7	10		9	
1564 ROTAVIRUS.....	1									
ROSS RIVER VIRUS					2					
DENGUE (TYPE 3)								1		
ARBO. GROUP B.								3		
Total.....	5	199	4	2	8	8	22	47	16	4

DISEASE	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Salmonella infections		21	13	57	10	3	12	1	117	* 1573
Shigella infections		2	20	5	4		3		34	363
Smallpox									—	—
Syphilis	17	16	39	20	19		42	4	157	* 1537
Tetanus									—	4
Trachoma									—	1
Tuberculosis (all forms)	28	28	20	18	17			4	115	1012
Typhoid fever	3								3	11 + 3 CARRIERS
Typhus (all forms)									—	—
Vibrio parahaemolyticus infections									—	—
Yellow Fever									—	—
Yersinia enterocolitica infections									—	—

(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.)

* Corrections made to the Cumulative Total since last report

Leptospirosis + 1 case for N.S.W.

Q fever -1 case for N.S.W.

Salmonella infections + 1 case for N.T.

Syphilis + 2 for N.S.W.

+ 3 for N.T.