ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 83/6
Issue date: 25 March 1983

Contents:

VIRUS REPORTING SCHEME - A total of 1082 reports were received this period.

- Antibodies against Weil-Felix OX-K, but not against OX-19 and OX-2 strains of *Proteus vulgaris*, were reported by the Prince of Wales Hospital, Sydney, in an eight year old girl who had returned from Indonesia two weeks previously. This agglutination pattern is suggestive of *Rickettsia tsutsugamushi* (scrub-typhus) infection, which is a mite-borne illness endemic to southern and eastern Asia, northern Australia and the western Pacific Islands. The multiple, serologically distinct strains of *R. tsutsugamushi* invalidate routine complement fixation, but the indirect immunofluorescence test is being evaluated using the three strains Karp, Kato and Gilliam which exhibit greater cross-reactivity.
- Fairfield Hospital, Melbourne, reported the detection of measles antigen by immunofluorescence following co-cultivation with monkey embryo kidney cells of brain biopsy material from a 20 year old male with encephalitis. Invasion of the CNS may be a common feature of measles infection, but one out of 1-2000 patients develop clinical signs of encephalitis. The usual pathogenesis is a microglial demyelination, which is believed to result from an autoimmune hypersensitivity following viral invasion.

(continued from page 5)

The outbreaks described were recognized through surveillance of routine reports of salmonella infections from diagnostic laboratories. Through early detection and investigation, the sources were identified and the outbreaks contained. This success was partly due to the unusual serotypes in the countries where the infections occurred. The Microbiological Diagnostic Unit, Melbourne, through Commonwealth funding, recently has expanded the national salmonella surveillance scheme to non-human isolates. Quarterly reports will be published in the *CDI* in conjunction with the human salmonellosis reports.

References

1. *J. Food. Protect.* (1977) *40*:718
2. *WER* (1973) *49*:378
3. *WER* (1982) *57*:329
4. In: Survey of the Microbiological Status of Foods, Phase 1 and 2. National Health & Medical Research Council (1981) : 110

The Bulletin is compiled and distributed by the Environmental Health Branch, Department of Health, P.O. Box 100, Woden, A.C.T. 2606, Australia, and is available on request.

CHOLERA SURVEILLANCE AND UPDATE; ROCKHAMPTON (JANUARY 1983)

(Contributed by A.T.C. Bourke, Y.M. Cossins, T.J. Lunney, E.R. Griggs and B.R.W. Gray, State Department of Health, Brisbane; and Department of Health, Rockhampton City Council, Queensland).

On 12 January 1983, a young severe diabetic developed diarrhoea. A toxigenic strain of Vibrio cholerae, biotype El Tor, serotype Inaba, was isolated from his faeces (reported initially in CDI 83/2 and 83/3). Household and other close contact were screened but no infections were detected. Initially it was thought that the patient may have contracted cholera from relatives in the Kenilworth area who had visited Rockhampton prior to the onset of illness. One or more of the relatives could have picked up a mild clinical or asymptomatic infection from one of the tributaries of the Mary River, which in the previous year had been found on numerous occasions to contain the O1 cholera vibrio. However, subsequent studies failed to substantiate this hypothesis. In the meantime, the investigation of what were considered to be two unlikely food sources of the infection led to the discovery that lettuce, cultivated in a lettuce market garden in North Rockhampton, were being spray-irrigated with creek water which contained the O1 cholera vibrio. Laboratory examination of the irrigation water from a stand-pipe and two out of five heads of lettuce sampled from the garden were also shown to contain this organism. Remedial action now consists of spray-irrigating the lettuce crop with creek water 30 minutes after it has been hand-chlorinated in a holding tank installed recently on the property. Since the patient consumed lettuce cultivated in that garden, it is postulated that, because of his debilitated condition, he contracted his infection from lettuce in which was entrapped creek water containing a small number of O1 cholera vibrios.

VIVAX MALARIA WITH AN EXCEPTIONALLY LONG INCUBATION PERIOD

(Based on California Morbidity (1983) No. 7).

On 24-25 August 1979, a 40 year old white male from Pasedena, California, experienced chills and sweats while on vacation in a National Park. The next day he visited a doctor because of high fever and was treated with tetracycline. The symptoms persisted, so he returned to a hospital where routine admission peripheral blood smear showed many trophozoites and dividing schizonts of Plasmodium vivax. He recovered uneventfully after treatment with chloroquine and primaquine.

The patient had lived his entire life in the USA except for a brief business trip to India in early September 1978, where he stayed one day in New Delhi and six days in Bangalore. He did not take any antimalaria medications before, during or after his visit. He had a brief bout of traveller's diarrhoea (untreated) in India but denied any other illness from the time he returned to the USA by direct flight in September 1978 until the onset of his malaria in August 1979 - almost one full year later.

This case is instructive because of the extraordinary long incubation period after infection in India. Vivax infections acquired in different parts of the world show striking differences in the duration of the incubation period and the length of latency between the primary attack and relapses. These differences have been broadly correlated with climatic zones. Prolonged latency has been observed in areas where year

round malaria transmission does not occur. Because of winter in temperate regions or the dry season in some tropical areas, anopheline mosquitoes are not available for transmission. The long latency may be considered a selective adaptation of the parasite to the local seasonal pattern of anophelism. In type I strains (e.g. Chesson), the incubation period for the first clinical attack is short, from 10-40 days (average 14 days) and relapses may begin one month or sooner after the first attack. Type II strains (e.g. St Elizabeth) also have a short incubation before the primary attack, but have a latency of five months or more between the first attack and first relapse. Type III strains (e.g. Hibernans) have a prolonged incubation period of 6-12 months before the primary attack, succeeded by a series of relapses at short intervals, and by a second long latency followed by relapses. Relapses of vivax malaria after three years are unusual with any of the three types.

The first clinical attack of vivax malaria can also be delayed when prophylactic antimalarials, such as chloroquine, are taken by travellers to endemic areas to suppress the primary attack. The first symptomatic attack of malaria may then occur six months or more after infection, long after leaving the endemic area.

Editorial Comment

Antimalarial drugs act only as suppressive agents, since none are effective against the sporozoite stage. The current range of drugs for suppression is quite limited, and recommendations for chemoprophylaxis for non-immune travellers visiting malarious areas for relatively short periods were detailed in CDI 82/12. However, resistance patterns are continually changing.[1,2] Strains of P. falciparum have shown foci of resistance to chloroquine (in various countries of America, Asia, Africa and South West Pacific - see CDI 82/12), pyrimethamine (in Africa) and combinations of pyrimethamine with sulphadoxine (Fansidar; in Indonesia, Thailand, Papua New Guinea; Brazil and East Africa) and pyrimethamine with sulphone (Maloprim; in East Africa). Foci of resistance of P. vivax or P. malariae in areas of Colombia, Venezuela, Pakistan, Malaysia, Taiwan and Vietnam are still restricted to proguanil and pyrimethamine and one geographical area of the South West Pacific to primaquine.

In Australia during 1982, an interim total of 549 imported cases and four introduced cases of malaria have been recorded in the Central Register of Malaria Cases (personal communication, P.M. Moodie, Commonwealth Institute of Health, Sydney), of which 434 infections were due to P. vivax. Comparable figures for 1981 and 1980 were 497 (378 due to P. vivax) and 628 (529 due to P. vivax) respectively. Although the proportion of P. falciparum cases has remained fairly constant, which may reflect better education of travellers for the need of chemoprophylaxis, continual publicity together with the distribution of concise acceptable information including forms of protection other than chemoprophylaxis is essential.

No resistance to 4 aminoquinolines (e.g. chloroquine) has been seen in P. vivax and P. malariae in the field. However, therapy and chemoprophylaxis of P. vivax (and P. ovale) infections is not without problems, as the parasite may persist in the liver (hypnozoites) for as long as four years after chloroquine suppression is discontinued. Relapses due to

reactivation may be prevented by the use of primaquine. Nevertheless, the use of primaquine for terminal prophylaxis is not indicated for all travellers, and should be evaluated on an individual basis i.e. the intensity and duration of the individual's exposure to P. vivax and P. ovale and whether the traveller is returning to the receptive areas of Australia (i.e. north of 19°S latitude). The use of primaquine chemoprophylactically (i.e. starting on the day of departure from an endemic area) is controversial, since infection with these relapsing species is rarely life-threatening, and the drug has been associated with severe side-effects such as haemolytic anaemia in G6PD-deficient persons.

Research for new antimalarial drugs is difficult and expensive, and is centred currently around the aminoalcohols (mefloquine[3], halofantrine[4]) and qinghaosu[3] and its derivatives. Malaria vaccines should open new vistas for immunoprophylaxis,[5] but apart from the numerous technical difficulties, unforeseen legal, commercial and marketing problems have arisen recently and need to be reconciled.[6]

References

1. BMJ (1982) 285:674
2. WER (1982) 57:381
3. Lancet (1982) 2:286
4. Am. J. Trop. Med. Hyg. (1982) 31:1075
5. Br. Med. Bull. (1982) 38:161
6. Science (1983) 219:466

SALMONELLA IN CHOCOLATE AND SPICES

(Based on CDWR (1983) 9:35 and CDR (1983) 83/09:3)

CHOCOLATE - In the first quarter of 1982, 77 strains of Salmonella napoli were isolated from patients in Milan, Italy. In May and June 1982, 32 cases of S. napoli were reported to the Communicable Disease Surveillance Centre (CDSC), UK. Most of the infections were in children less than 15 years of age living in the south of England. Since only 15 human isolations and no non-human isolations had been reported in the previous 30 years, a common source was evident and an epidemiological investigation was initiated.

Overall, in the period May-August 1982, 272 human isolates of S. napoli were reported; 202 were primary cases, 43 were household contacts of cases and 27 were asymptomatic. Of the 202 primary cases, 118 (58%) were children aged <15 years, and 60% of those aged 15 years and over were women. Complications included bacteraemia in 16 cases, and peritonitis, diabetic coma, septic arthritis and ischiorectal abscess each in one case. Case control studies showed an association between gastroenteritis due to S. napoli and the consumption of imported Italian chocolate, principally chocolate covered bars from one production line labelled "Rocky Junior" and "Tommy Junior" designed to be particularly attractive to children. On 23 July the Department of Health and Social Security issued a public health warning, and with the importer's active co-operation, wholesale distribution was stopped and four-fifths (2.4 million bars) of the total imported products were recalled and destroyed. The last case was reported three weeks later.

Of the 224 individual "Rocky Junior" and 146 "Tommy Junior" bars examined, S. napoli was found in 107 (48%) "Rocky" bars and 47 (32%) "Tommy" bars. It was also demonstrated that the

organism was contained in the chocolate layer of the bars and not in the base or cream filling. Quantitative estimates of the level of contamination ranged from 2-23 organisms per gm of chocolate (3 gm chocolate covering per bar), and the ingestion of a single bar was sufficient to cause illness.

The factory where the contaminated chocolates were made was clean and modern, and the production method was not unusual. It is universal practice to roast raw cocoa beans and this process should generally lead to sterilization of the beans. After roasting of the beans further heat processing may not be sufficient to destroy salmonellas. Recontamination by dust from the raw beans may introduce salmonellas, or contamination may occur through seepage of water used in the warm water jackets of storage vessels and of pipes carrying liquid chocolate. This water may be taken from untreated sources and S. napoli has been isolated from water sources in northern Italy. Perpetuation of contamination may result from re-cycling of excess chocolate. The addition of other ingredients, such as chocolate crumb, cocoa butter, cocoa powder and dried milk, may further introduce contamination to the production line.

Chocolate products have occasionally been contaminated with Salmonella, and this has resulted in extensive recalls,(1) but salmonellosis arising from consumption of chocolate is rare. In 1970-71, confectionery made with cocoa powder contaminated with S. durham infected at least 109 persons in different parts of Sweden.(2) In 1973-74, more than 200 cases of S. eastbourne in Canada and eastern U.S.A. were traced to a variety of chocolate products manufactured in Quebec; cocoa beans presumably imported from West Africa were the source of contamination.

SPICES - Between November 1981 and September 1982, 126 confirmed cases of S. oranienburg were identified in Norway.(3) The serotype was isolated from one brand of black pepper found in homes of some of the cases and in retail stores. The peppercorns were imported from the Federal Republic of Germany, and were originally harvested in Brazil.

The only other known incident involving pepper occurred in Canada between December 1973 and April 1974, when 14 persons from the Maritime provinces were infected with S. weltevreden. The implicated black and white pepper imported from India had not been sterilized with ethylene oxide before retail packaging. In 1980, spices from India were suspected of causing gastro-enteritis from S. kingabwa in the USA, but conclusive proof was lacking. Spices frequently contain low levels of spore-forming bacteria e.g. Clostridium perfringens, Bacillus cereus and occasionally Salmonella. These organisms have the potential to cause illness if allowed to grow in food with which spices come in contact. Pepper, in contrast to many other spices, like sage, is not always cooked with the food but may be sprinkled on top and left for some time at ambient temperature.

In Australia ethylene oxide is not an approved treatment, so that spices are not sterilized prior to retail sale. Although a recent survey failed to detect Salmonella in samples of black and white pepper, cinnamon, nutmeg, dried ginger and dried chives, the levels of contamination with B. cereus, C. perfringens and E. coli (one sample) were cause for concern.(4)

(continued on page 1).

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 3/3/83 - 16/3/83 BULLETIN NUMBER 83/6

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WWH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED	1	1	2			7	4	2	17
0101 ADENOVIRUS TYPE 1				1		3			4
0102 ADENOVIRUS TYPE 2	1		1	2		6			10
0105 ADENOVIRUS TYPE 5				1		3			4
0106 ADENOVIRUS TYPE 6					1				1
0119 ADENOVIRUS TYPE 19								18	18
0199 ADENOVIRUS TYPING PENDING			2		4				6
0201 INFLUENZA A VIRUS							2	1	3
0203 INFLUENZA B VIRUS			1					1	2
0301 PARAINFLUENZA VIRUS TYPE 1						1			1
0302 PARAINFLUENZA VIRUS TYPE 2							2	1	3
0303 PARAINFLUENZA VIRUS TYPE 3						3	1		4
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)			1			2	13	3	19
0500 RHINOVIRUS (ALL TYPES)				4	8	5	5	1	23
0600 MYCOPLASMA PNEUMONIAE	24		1	4		5	6	6	46
0700 ORNITHOSIS-PSITTACOSIS				4		1			5
0899 COXSACKIEVIRUS GROUP A TYPING PENDING							3		3
0902 COXSACKIEVIRUS B2								2	2
0903 COXSACKIEVIRUS B3	1			1	2		1		5
1007 ECHOVIRUS TYPE 7								1	1
1011 ECHOVIRUS TYPE 11	7		7	8	2	1	7	4	36
1014 ECHOVIRUS TYPE 14							2		2
1017 ECHOVIRUS TYPE 17				1					1
1022 ECHOVIRUS TYPE 22				1			2		3
1025 ECHOVIRUS TYPE 25		1							1
1101 POLIOVIRUS TYPE 1	2					3	3		8
1102 POLIOVIRUS TYPE 2	1								1
1104 POLIOVIRUS-VACCINAL STRAIN			4		4	1			9
1200 MUMPS VIRUS			1	1		2	1	1	6
1300 HERPES VIRUS GROUP-NOT TYPED	37			1		7			45
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1			5		1	1	69	77
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	6					2		2	10
1303 VARICELLA-ZOSTER VIRUS	3					4		1	8
1306 HERPES SIMPLEX TYPE 1	15			28		14	9		66
1307 HERPES SIMPLEX TYPE 2	89			51		18	32	1	191
1399 HERPES VIRUS TYPING PENDING			12		4	2			18
1401 COXIELLA BURNETI	6		1				3		10
1402 OTHER RICKETTSIAE			1						1
1502 PICORNA VIRUS-NOT TYPED	4		15					3	22
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS	4			4					8
1522 RUBELLA VIRUS	1		2	1			5	1	10
1532 HEPATITIS B ANTIGEN	24		10	31		19	5	5	94
1533 HEPATITIS B ANTIBODY							1		1
1535 HEPATITIS A ANTIBODY	5		1	9		7	8	16	46
1541 CHLAMYDIA A - C TRACHOMATIS	20		5			1	9	68	103
1556 CMV - CYTOMEGALOVIRUS	9	1	5	19	8	3	3	7	55
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS			3						3
1571 ENTEROVIRUS TYPE 71 (BRCR)				2					2
1599 ENTEROVIRUS TYPING PENDING		8	6		6	2	6		28
POXVIRUS GROUP NOT TYPED				1					1
ROSS RIVER VIRUS							31	5	36
SMALL VIRUS (LIKE) PARTICLE						1			1
Total	261	11	81	181	39	124	165	220	1,082

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 3/3/83 to 16/3/83 .... 83/6

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
0101 ADENOVIRUS TYPE 1.....		4									
0102 ADENOVIRUS TYPE 2.....		4					3				
0105 ADENOVIRUS TYPE 5.....							4				
0106 ADENOVIRUS TYPE 6.....		1									
0201 INFLUENZA A VIRUS.....		2									
0203 INFLUENZA B VIRUS.....									2		
0301 PARAINFLUENZA VIRUS TYPE 1....		1									
0302 PARAINFLUENZA VIRUS TYPE 2....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....		5									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	19									
0500 RHINOVIRUS (ALL TYPES).....		22									
0600 MYCOPLASMA PNEUMONIAE.....	7	29		1					1		
0700 ORNITHOSIS-PSITTACOSIS.....		3									
0902 COXSACKIEVIRUS B2.....				1							
0903 COXSACKIEVIRUS B3.....	1	1		2					1		
1007 ECHOVIRUS TYPE 7.....						1					
1011 ECHOVIRUS TYPE 11.....	1	3		18		1	4				1
1014 ECHOVIRUS TYPE 14.....	1						1				
1017 ECHOVIRUS TYPE 17.....											1
1022 ECHOVIRUS TYPE 22.....		1					2				
1025 ECHOVIRUS TYPE 25.....				1							
1101 POLIOVIRUS TYPE 1.....	2	1					5				
1102 POLIOVIRUS TYPE 2.....	1										
1104 POLIOVIRUS-VACCINAL STRAIN....	1						8				
1200 MUMPS VIRUS.....	1					1					
1300 HERPES VIRUS GROUP-NOT TYPED..											1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	2	1	1							2	33
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	4		1					1			
1303 VARICELLA-ZOSTER VIRUS.....						1					6
1306 HERPES SIMPLEX TYPE 1.....	1	3	1							3	30
1307 HERPES SIMPLEX TYPE 2.....	2	1		1							7
1401 COXIELLA BURNETI.....	3										
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....	1	1	3	1							3
1522 RUBELLA VIRUS.....	1										6
1532 HEPATITIS B ANTIGEN.....	54							35			
1533 HEPATITIS B ANTIBODY.....								1			
1535 HEPATITIS A ANTIBODY.....	8							34			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1						1				
1556 CMV - CYTOMEGALOVIRUS.....	5	9			2	1		5		8	1
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....							3				
1571 ENTEROVIRUS TYPE 71 (BRCR)....				2							
1599 ENTEROVIRUS TYPING PENDING....				1							
9992 ROSS RIVER VIRUS.....	6	2									13
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	104	115	6	28	2	5	33	76	4	13	103

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 3/3/83 to 16/3/83 ... 83/6
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
0102 ADENOVIRUS TYPE 2.....						1	1	1		
0105 ADENOVIRUS TYPE 5.....								1		
0119 ADENOVIRUS TYPE 19.....	1	17								
0201 INFLUENZA A VIRUS.....								3		
0302 PARAINFLUENZA VIRUS TYPE 2....								1		
0500 RHINOVIRUS (ALL TYPES).....								1		1
0600 MYCOPLASMA PNEUMONIAE.....							3	8	3	
0700 ORNITHOSIS-PSITTACOSIS.....							1	1		
0902 COXSACKIEVIRUS B2.....							1			
0903 COXSACKIEVIRUS B3.....					1					
1011 ECHOVIRUS TYPE 11.....					1		3	7		
1200 MUMPS VIRUS.....			4					1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	40								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			2					1	1	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	2	27					2	1		
1307 HERPES SIMPLEX TYPE 2.....		180								
1401 COXIELLA BURNETI.....							1	4	2	
1402 OTHER RICKETTSIAE.....							1			
1522 RUBELLA VIRUS.....								6	2	
1532 HEPATITIS B ANTIGEN.....									5	
1535 HEPATITIS A ANTIBODY.....								1	3	
1541 CHLAMYDIA A - C.TRACHOMATIS...	2	98								
1556 CMV - CYTOMEGALOVIRUS.....		9				5	4	3	7	1
9992 ROSS RIVER VIRUS.....				1	23			9	1	
Total.....	6	371	6	1	25	6	17	49	25	2