ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 87/12
Issue date: 22 June 1987

Editor Dr I.F. Cook

Contents:

VIRUS REPORTING SCHEME: A total of 939 reports were processed for this period.

Eight cases of Q fever were reported from Queensland. Occupational exposure data were available for only five of the cases:-

- 3 male meatworkers from Beenleigh, 2 aged 34 years and one aged 22 years respectively,
- a 48 year old male farmer from Innisfail, and
- an adult male station hand from Hughendon.

Herpes simplex type 2 was isolated from the cervical swab of a 15 week pregnant, 16 year old female who presented with recurrent genital lesions.

Poliovirus type 2 was isolated from the faeces of a 4 month old male who died of Sudden Infant Death Syndrome (SIDS).

Cytomegalovirus was isolated from:-

- the breast milk of a 32 year old female whose infant (age unknown) has been diagnosed as having a cytomegalovirus infection. It is not known whether the infant has acquired the infection congenitally or post-natally via breast milk.
- the bronchial alveolar lavage of a 24 year old female who experienced severe airflow restriction following a recent heart and lung transplantation operation.

Adenovirus (typing pending) was recovered from the faeces of a 9 year old female with severe progressive weight loss following bone marrow transplantation.

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

HIV INFECTION TRANSMITTED FROM AN ORGAN DONOR SCREENED FOR HIV ANTIBODY - NORTH CAROLINA (USA)
(Based on MMWR Vol 36/ No. 20/29 May 1987)

In August 1986, a cadaveric organ donor was found positive for antibody to the human immunodeficiency virus (HIV) by both enzyme immunosorbent assay (ELISA) and Western blot methods after some of the donated organs had been transplanted. A blood sample, which was taken after the donor had received a large number of blood transfusions, had been negative for HIV antibody. Two days later, when the organs were removed, more blood samples were collected. These were forwarded with the donated organs to the various transplantation centres. At one of these centres, one of the later samples was found to be seropositive.

Three persons received organs from this donor:
. two of them were subsequently found to be seropositive for HIV antibody
. the third, who had received the donor's heart did not survive the transplant procedure.

This is the first report of HIV transmission by organ transplantation from a donor screened for HIV antibody. A summary of the investigation of the donor and the two surviving recipients follows.

DONOR: A 30 year old male who was involved in a motor vehicle accident was comatose when admitted to a North Carolina hospital. He was hypotensive because of bleeding from multiple head and neck lacerations. On admission, blood samples where collected for type - and cross-matching, and blood transfusions were started within one hour. The patient's bleeding persisted despite surgery to improve homeostasis. Approximately 11 hours after admission, he had received a total of 56 units of blood and blood components:
- 1 unit of whole blood,
- 28 units of packed red blood cells,
- 7 units of plasma, and
- 20 units of platelets.

At this time, another blood sample was collected and tested for HIV antibody. The specimen was negative by ELISA (Abbot Laboratories, North Chicago, Illinois; optical density ratio, sample/control = .103/.131). The patient's condition did not improve, and he was declared brain-dead 2 days after HIV antibody testing. Family members consented to organ donation and denied any knowledge of the patient having a risk factor for HIV infection.

The donated kidneys, heart, and liver were removed and transported to other medical centres for transplantation. Samples of the donor's blood, which were collected when the organs were removed, were sent with each organ. As part of one centre's routine procedure, one of these blood samples was

tested for HIV antibody and was found positive by ELISA (Genetic Systems, Seattle, Washington; optical density ratio = .95/<.30) and was subsequently found positive by Western blot assay. The transplantation teams were notified of the test result, but the heart, liver, and one kidney had already been transplanted.

Personnel from the hospital where the organs had been removed were contacted. They located both the serum sample collected on admission and the serum sample previously found negative for HIV antibody:

. the serum collected at the time of admission, before any transfusions were administered, was:
  1. highly reactive on the Abbott ELISAs performed at
     - the hospital (optical density ratios = .766/.126 and .556/.126)
     - the North Carolina State Laboratory of Public Health (optical density ratios = .842/.108 and .698/.137)
  2. also positive by Western blot assay at the State Laboratory.

  When testing was repeated, the serum collected after the blood transfusion was again seronegative by ELISA at the hospital and by both ELISA and Western blot methods at the State Laboratory.

Recipient 1: The donated kidney was transplanted in an adult male with end-stage renal disease. The recipient is married and denied risk factors for HIV infection. He was negative for HIV antibody 3 days after transplantation. A blood specimen collected 10 weeks after transplantation was positive for HIV antibody by ELISA, and a specimen collected 1 week later was positive by both ELISA, and Western blot assay. The recipient experienced fever 8 days after receiving the renal allograft. A renal biopsy showed acute rejection which improved with additional immunosuppressive therapy. To date, the recipient has not developed any opportunistic illness and continues to feel well.

Recipient 2: The donated liver was transplanted in an adult male with sclerosis of the bilary ducts and progressive liver failure. He is married and denied risk factors for HIV infection. He was tested 4 days after transplantation and was negative for HIV antibody. Twelve weeks after the procedure, he was positive for HIV antibody by ELISA, and a specimen collected 4 weeks later was positive by both the conventional ELISA and an ELISA using recombinant viral proteins (ENVACORE, Abbott Laboratories). Four months after transplantation, the recipient developed

fever and malaise. A liver biopsy showed moderate allograft rejection. The recipient's condition improved with an adjustment in immunosuppressive therapy, and he returned home the following month.

MMWR Editorial Note:

Previous reports have linked kidney-transplant recipients who have subsequently become seropositive for HIV antibody with donors who were later found to have risk factors for HIV infection[1-4]. However, this is the first report of transplantation - associated HIV transmission from a cadaveric organ donor screened for HIV antibody. This donor appears to have been false-negative for HIV antibody by ELISA as a result of the large number of transfusions he received before serum was collected for testing.

The United States Public Health Service recommended in May 1985 that potential organ donors be screened for HIV antibody[5]. In January 1986, the Centers for Disease Control conducted an anonymous survey of representatives from 44 transplantation programs attending a meeting of the Southeastern Organ Procurement Foundation. All of the 26 representatives who responded reported that their centres screened donors for HIV antibody. Three of these representatives (12%) also reported identifying at least one potential organ donor who was positive for HIV antibody by ELISA and Western blot methods.

Organs from donors who are HIV-seropositve should not be used for transplantation except in very unusual circumstances. If an urgent need requires considering transplantation of an organ from a seropositive donor, the potential recipient or the appropriate family members should be informed of the risks of acquiring HIV infection. Such transplantation should not take place without the consent of either the potential recipient or the appropriate family members. When donors have been transfused before their organs are removed, testing for HIV antibody should be conducted on serum collected at the time of admission rather than on serum obtained after multiple transfusions. If donor serum collected at the time of admission is not available from other sources, a pre-transfusion sample may be available from the blood bank since many blood banks hold specimens collected for compatibility testing for at least 7 days.

REFERENCES

1. Lancet (1985) 2:672
2. Lancet (1985) 2:1361-2
3. Lancet (1986) 2:398
4. Ann Intern Med (1987) 106:244-5
5. MMWR (1985) 34:294

AIDS UPDATE - INTERNATIONAL
(Based on WER No 23, 5 June 1987)

Global data - AIDS cases reported to WHO, by country, as of 3 June 1987.

Country/Area	Date of report	Number of cases
Algeria	15.05.86	3
Angola	26.09.86	6
Anguilla	30.06.86	–
Antigua and Barbuda	31.12.86	2
Argentina	31.03.87	78
Australia	20.05.87	481
Austria	31.03.87	72
Bahamas	31.12.86	85
Bangladesh	14.04.87	–
Barbados	31.12.86	15
Belgium	31.03.87	230
Belize	31.12.86	1
Benin	13.11.86	2
Bermuda	31.12.86	48
Bhutan	14.04.87	–
Bolivia	30.06.86	1
Botswana	09.05.87	12
Brazil	30.04.87	1 695
British Virgin Islands	31.12.86	–
Bulgaria	31.03.87	1
Burkina Faso	13.11.86	–
Burma	14.04.87	–
Burundi	31.03.87	128
Cameroon	05.03.87	25
Canada	27.04.87	1 000
Cape Verde	30.04.87	4
Cayman Islands	31.12.86	1
Central African Republic	13.11.86	202
Chad	13.11.86	1
Chile	31.12.86	22
China	02.04.87	2
China (Province of Taiwan)	26.01.86	1
Colombia	31.12.86	30
Comoros	13.11.86	–
Congo	13.11.86	250
Costa Rica	31.12.86	16
Côte d'Ivoire	13.11.86	118
Cuba	30.06.86	1
Cyprus	08.10.86	1
Czechoslovakia	31.03.87	7
Democratic People's Republic of Korea	09.05.87	–
Denmark	31.03.87	150
Dominica	31.12.85	–
Dominican Republic	08.12.86	127
Eastern Mediterranean Region	07.04.87	18
Ecuador	30.06.86	7
El Salvador	31.12.86	6
Ethiopia	28.04.87	–
Finland	31.03.87	19
France		
Metropolitan	31.03.87	1 617
Overseas:		
French Guiana	31.12.86	58
French Polynesia	01.04.87	1
Guadeloupe	31.12.86	40
Martinique	31.12.86	16
Gabon	13.11.86	–
Gambia	16.03.87	14
German Democratic Republic	31.03.87	3
Germany, Federal Republic of	30.04.87	1 036
Ghana	25.05.87	145
Greece	31.03.87	41
Grenada	31.12.86	3
Guatemala	31.12.86	15
Guinea	13.11.86	–
Guinea Bissau	13.11.86	–
Guyana	31.12.86	–
Haiti	31.03.87	851
Honduras	31.12.86	13
Hong Kong	31.12.86	4
Hungary – Hongrie	31.03.87	3

Country/Area	Date of report	Number of cases
Iceland	31.03.87	4
India	09.05.87	9
Indonesia	21.04.87	1
Ireland	31.03.87	19
Israel	31.03.87	38
Italy	31.03.87	664
Jamaica	11.05.87	16
Japan	27.04.87	38
Kenya	11.03.87	286
Lesotho	13.11.86	1
Liberia	04.02.87	1
Luxembourg	31.03.87	7
Madagascar	13.11.86	–
Malawi	13.11.86	13
Malaysia	01.04.87	1
Maldives	14.04.87	1
Malta	31.03.87	5
Mauritania	13.11.86	–
Mauritius	13.11.86	–
Mexico	31.03.87	407
Montserrat	31.12.85	–
Mozambique	31.12.86	1
Nepal	31.12.86	–
Netherlands	31.03.87	260
New Zealand	15.05.87	39
Nicaragua	31.12.86	–
Nigeria	14.03.87	–
Norway	31.03.87	45
Panama	30.09.86	12
Paraguay	31.12.86	1
Peru	30.06.86	9
Philippines	31.12.86	3
Poland	31.03.87	2
Portugal	31.03.87	54
Republic of Korea	01.04.87	1
Romania	31.03.87	2
Rwanda	30.11.86	705
Saint Christopher and Nevis	31.12.85	1
Saint Lucia	30.06.86	3
Saint Vincent and the Grenadines	30.06.86	3
Sao Tomé and Principe	01.12.86	–
Senegal	13.11.86	–
Seychelles	13.11.86	–
Singapore	01.04.87	1
South Africa	25.05.87	70
Spain	31.03.87	357
Sri Lanka	14.04.87	2
Suriname	30.06.86	2
Swaziland	10.04.87	6
Sweden	08.05.87	110
Switzerland	31.03.87	227
Thailand	27.04.87	6
Togo	13.11.86	–
Trinidad and Tobago	31.12.86	134
Tunisia	14.05.86	2
Turkey	07.05.87	19
Turks and Caicos Islands	31.12.86	2
Uganda	28.02.87	1 138
USSR	31.03.87	32
United Kingdom	30.04.87	750
United Republic of Tanzania	18.04.87	1 130
United States of America	25.05.87	35 980
Uruguay	31.12.86	8
Vanuatu	31.12.86	–
Venezuela	08.12.86	69
Yugoslavia	31.03.87	10
Zambia	13.11.86	250
Zimbabwe	21.01.87	57
Total		51 751

RUBELLA RE-INFECTION IN PREGNANCY - A CASE-REPORT OF CONGENITAL RUBELLA

(Contributed by Eric Uren, Virology Department, Royal Children's Hospital, Melbourne Victoria)

Congenital rubella, demonstrated serologically by rubella specific IgM and IgG, was diagnosed in a 3 month old male who presented with bilateral congenital cataracts, patent ductus arteriosis and developmental delay.

Serological evaluation of maternal serum, at the time of the child's presentation, showed positive rubella IgG titre but negative IgM. Medical history reported that the mother had assumed that she was immune to rubella, since she had received the vaccine six years previously and had been shown to have a positive rubella titre two years prior to this pregnancy. However, maternal serum obtained prior to, and during, pregnancy was not available for re-testing.

This case seems to indicate rubella re-infection during pregnancy leading to intra-uterine infection and congenital rubella. Rubella re-infection during pregnancy has been documented[1-3], but infection of the foetus with subsequent congenital abnormalities[4] is rare.

CDI Editorial Comment

In the case presented, the mother's immune status in relation to rubella, prior to and during pregnancy, cannot be ascertained. It appeared that she has seroconverted following rubella vaccination since a positive rubella titre was reported two years prior to conception. However it cannot be ascertained whether:-

- the reported positive rubella antibody titre was consistent with immune protection, and
- rubella antibody titre at the time of conception remains comparable to that detected two years previously.

Rubella re-infection had evidently taken place, but maternal rubella immunity remains doubtful and subsequent congenital abnormalities appear severe.

REFERENCES

1. MJA (1982) 12:514
2. BMJ (1981) 282:187
3. MJA (1977) 3:77
4. Lancet (1985) 1:700

POST OPERATIVE LISTERIOSIS - A CASE REPORT

(Contributed by D. Mitchell, Registrar - Infectious Diseases Unit - Woden Valley & Royal Canberra Hospitals)

Following an uncomplicated elective surgery involving resection and grafting of an abdominal aortic aneurysm, the patient, a 69 year old man, was admitted to the Intensive Care Unit for observation and post-operative ventilation.

Twelve hours following the surgical procedure, the patient's condition deteriorated with high fever, hypotension, oliguria and decreased respiratory function. Sepsis was suspected and empirical therapy with intravenous administration of cefataxime, metronidazole and gentamicin was initiated. The patient's condition failed to improve and necessitated prolonged ventilation, inotropic support and total parenteral nutrition.

Blood cultures of samples taken on the first post-operative night grew Listeria monocytogenes after five days incubation. An examination of the cerebrospinal fluid showed no evidence of meningitis. The antibiotic therapy was subsequently changed to intravenous ampicillin and gentamicin, and the patient's condition rapidly improved.

Comment

In the majority of cases, the probable source of Listeria acquisition is difficult to ascertain. However L.monocytogenes is a recognised pathogen causing septicaemia or meningitis in neonates, pregnant women or immunosuppressed patients. Since the present case had none of the above risk factors it was postulated that the patient was an asymptomatic bowel carrier and that bowel handling during surgery caused dissemination of the organism.

Third generation cephalosporins, such as cefataxime, are often used as empirical therapy in very sick septic patients, particularly for situations arising in the Intensive Care Unit setting. Listeria is however one organism that is resistant to this group of antibiotics. Once the organism was identified, the patient rapidly improved on ampicillin substitution.

A CHOLERA CASE - QUEENSLAND

(Contributed by A.T.C. Bourke*, L.R. Ashdown +, Y.M. Cossins*, G.D. Hapgood* and J. Wilson ++

* Queensland Department of Health
\+ Australian Health Laboratory (Townsville)
++ Dalrymple Shire Council)

On 6 February 1987, a previously healthy 24 year old male, caretaker on a station in Dalrymple Shire, 90km north of Charters Towers, developed acute diarrhoea. On 11 February, his condition improved sufficiently to allow him to travel to Charters Towers hospital for medical attention. The patient was not admitted but cholera was diagnosed on 17 February when Vibrio cholerae 01 biovar El Tor subtype Inaba was isolated from a faecal specimen obtained at hospital presentation. In the meantime, the patient made an uneventful recovery.

The patient gave no history of overseas travel, recent contact with sick persons or treatment for any gastrointestinal symptoms. Neither his close contacts, a 19 year old wife and a 6 month old son, nor his casual contacts, his wife's parents and relatives whom he visited during his disease incubation,

reported any gastrointestinal disturbances and all were laboratory confirmed to be free of cholera infection. Food storage and consumption histories did not implicate food as the vehicle of infection.

Subsequent epidemiologic investigation centred on water supply, consumption and disposal. The patient's residence is serviced by a septic-tank system, and water reticulated from an elevated 2 500-gallon circular corrugated iron tank which holds water supplied by a windmill-powered deep bore. The overflow, used for watering stock, is drawn off by a hose to a ground-level 25 000-gallon circular corrugated iron tank.

- One week before he became ill, the caretaker had introduced one fish and approximately half a gallon of water from one of the two swimming holes in that section of Allingham Creek which traverses the station property. Allingham Creek drains into the Fletcher River which in turn flows into the Burdekin River system.
- During the 5 days prior to disease onset, the caretaker had swum in and drunk water from the two swimming holes in Allingham Creek which was, at the time, swollen and fast flowing because of recent rainfalls. In addition, he had also consumed water from three deep bores on the station, including the bore which supplies the home. He had also bathed in the ground level 25 000-gallon tank containing the fish.

The following samples were collected on 18 and 24 February for bacteriological examination:

- Water from Allingham Creek swimming holes (by then the water level had fallen and was barely perceptible - however sampling was done at 2 points 80 m apart at the more frequently used swimming hole close to the house),
- overflow water from the hose draining the elevated 2 500-gallon tank,
- water from the reticulated system ie hose attached to elevated tank, ground-level tank, pipe directly connected to pumping mechanism, house tap over kitchen sink, and tap in backyard,
- water from the ground-level 25 000-gallon tank and subsurface incrustations scraped from the side of the tank.

Vibrio cholerae 01 biovar El Tor subtype Inaba was only recovered from the water samples and from the subsurface incrustations scraped from the side of the ground-level 25 000-gallon tank. No isolates were recovered from any other samples. It was concluded that the addition of a fish and the accompanying amount of water from Allingham Creek might have seeded the tank with Vibrio cholerae 01 biovar El Tor subtype Inaba which has persisted for at least 25 days. It was then postulated that, during the patient's incubation of the disease, the cholera organism was present in Allingham Creek, and that the patient had contracted cholera through drinking water from the creek on several occasions.

This reported cholera case seemed to indicate that Allingham Creek might have contaminated the Burdekin River system. Such contamination was also implicated when an earlier cholera case was reported in March 1984, involving a 38 year old male carpenter, resident of Canberra, who acquired the infection

while on a camping trip between Charters Towers and Greenvale[1]. Since the latest case report, subsequent surveillance of the environment has to date yielded no isolates of the cholera organism. Monitoring will, however, be extended into the next summer period to determine whether contamination of the Burdekin River System occurs as a seasonal pattern.

REFERENCE

1. MJA (1986) 144:29

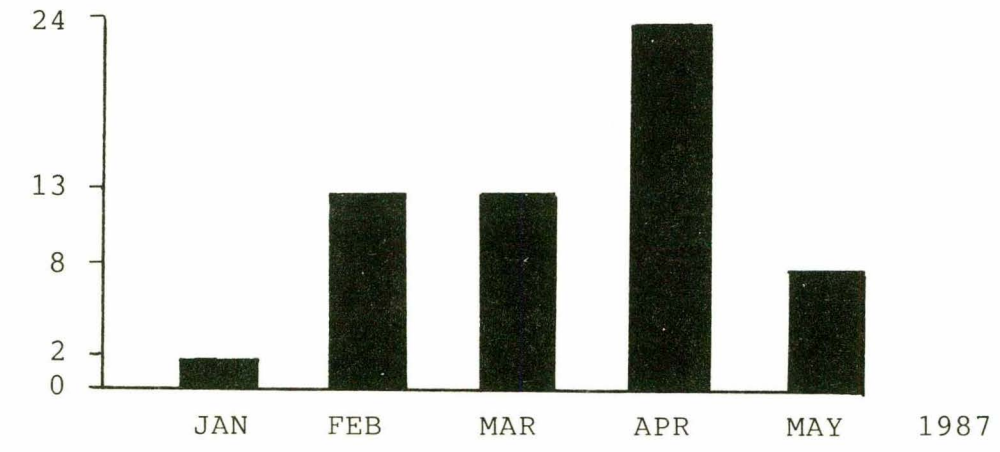
MYCOPLASMA PNEUMONIAE IN NEW SOUTH WALES - JANUARY TO MAY 1987

(Contributed by Dr A.L. Cunningham, L. Hueston and P.R. Field, Virology Department, Institute of Clinical Pathology and Medical Research, Westmead Hospital)

Sixty cases of Mycoplasma pneumoniae were notified. Diagnosis was based on:

- demonstration of a complement fixing antibody titre to M.pneumoniae 512 with a positive M.pneumoniae specific IgM.
- demonstration of a four fold or greater rise in complement fixing antibody titre to M.pneumoniae with a positive specific IgM.

The monthly incidence of M.pneunomiae cases is depicted in the figure below:



Six of the sixty cases were notified from the Wollongong area where an increased incidence of atypical pneumonia, although not all Legionella has been reported. All 6 cases occurred in April and May.

In the corresponding period in 1986:

- only 20 cases were notified according to the above diagnostic criteria,
- no cases were reported from the Wollongong region.

ORAL TYPHOID VACCINE

The Commonwealth Serum Laboratories is currently distributing an orally administered, live, attenuated vaccine for active immunisation against typhoid for the needle-shy traveller.

The vaccine product is:

. RECOMMENDED	for those travelling to or resident in countries where poor food and water sanitation practices occur irrespective of climate, in particular countries in Asia, Africa, Latin America and the Pacific Islands. It must be emphasised however that, regardless of immune protection, the individual should exercise care in selecting food and water for consumption in these countries.
. MARKETED	as TYPH-VAX (ORAL) The painless solution.
. PRESENTED	in a blister pack containing 3 easy-to-swallow enteric coated capsules.
. INTENDED	to provide the complete course of immunisation for all persons above 6 years of age (efficacy in children aged below 6 years is unknown at present)
. ORALLY ADMINISTERED	according to a recommended dosage of 1 capsule on each of days 1, 3 and 5 irrespective of age
. TO BE SWALLOWED	whole one hour before a meal - the capsule should not be chewed to prevent destruction of the organism by gastric acid.
. TO BE STORED	between +2 and +8°C in a dry place and protected from light - the product should not be used after the expiry date.
. NOT TO BE ADMINISTERED	concurrently with antibiotics or other drugs (eg antimalarials, sulphonamides) which are active against salmonellae. The vaccine should be administered first and at least one week should elapse between the final dose of the vaccine and such drugs.
. CONTRA-INDICATED IN:	primary and acquired immune deficiency including that from treatment with immunosuppressive and antimitotic drugs; - acute febrile illness; and - acute intestinal infection.

SUBJECT TO THE FOLLOWING

1. WARNING:
no data are currently available about the efficacy of Typh-Vax (Oral) in individuals with blood dyscrasias, leukaemia, lymphoma or any type of malignant neoplasm affecting the bone marrow or lymphatic system.
These individuals may fail to develop protection because of their disturbed immune functions.

2. ADVERSE REACTIONS:
are infrequent and generally mild. The following adverse effects were reported during trials:- constipation, abdominal cramps, diarrhoea, nausea, vomiting, anorexia and fever.

3. PREGNANCY:
NO DATA on the safety of Typh-Vax (Oral) during pregnancy is available.

4. DURATION OF PROTECTION:
conferred by the vaccine is not known at present. Repeat vaccination after 12 months appears advisable if exposure to infection is anticipated.

A MUTANT STRAIN OF SALMONELLA TYPHI
Each capsule contains not less than 10^9 viable organisms of Salmonella typhi strain Ty21a (S.typhi Gal E.mutant). This attenuated Ty21a berna strain is a mutant of S.typhi which is deficient in the enzyme UDP-4-galactose epimerase. In the presence of adequate amounts of galactose this organism, unable to effectively metabolise galactose, accumulates bacteriotoxic levels of galactose-containing metabolites and ultimately undergoes spontaneous lysis. However, in the presence of a restricted supply of galactose the organism develops the smooth lipopoly saccharide coat believed to be antigenetically relevant for eliciting an immune response. In the intestine, where galactose is normally present, the organism is however unable to survive for long and the vaccine strain cannot be detected in the stool after 3 days following oral ingestion.

EFFECTIVE IN PROMOTING IMMUNITY
The claim for vaccine efficacy was based:

INDIRECTLY
on a placebo-controlled, randomised double-blind trial of Ty21a conducted by Wahdan et al[1-2] in Alexandria, Egypt, where three doses of vaccine (1-3 x 10^9 viable vaccine organisms per dose) or placebo were given to 32 000 school children on days 1, 3 and 5 of one week. Before ingestion of liquid vaccine (reconstituted lyophilate) or placebo, each child chewed a 1g sodium bicarbonate table to neutralise gastric acid. Notable adverse reactions were not detected. During 36 months of surveillance the vaccine efficacy was reported to be 96% with a range of 77% to 99%[1].

DIRECTLY
on randomised, placebo-controlled field trial of efficacy conducted in Santiago, Chile[3], where three doses of vaccine (1-3 x 10^9 viable organisms per dose ie enteric-coated capsule) or placebo were administered to 24 000 school children on days 1, 3 and 5 of one week, in the classroom during the cool, non-typhoid season, mid-July to early September 1983. During 3 years of surveillance the vaccine efficacy was reported to average 67% from a range of 47% to 79%.

It is noted that the 67% protection conferred for at least 3 years[3] by three doses of Ty21a in enteric-coated capsules given within one week in Santiago, Chile is less than the impressive 96% efficacy over 3 years provided by a liquid formulation used in Egypt[2]. Although factors such as obvious differences in vaccine formulation and genetic constitution of the recipient populations may have contributed to the difference in vaccine efficacy, epidemiologic criteria such as the mean annual incidence in the placebo group in Santiago (103 x 10^5 per year), twice as high as that in Alexandria (46 x 10^5 per year), seem to indicate a significant difference between the force of infection and modes of transmission occurring in the two countries.

In addition to its apparent efficacy, Ty21a is claimed to:
1. offer greater advantage over parenteral killed whole cell vaccine in that Ty21a live oral typhoid vaccine causes no adverse reactions[1, 4, 5], despite those listed in the above product information.
2. offer immune protection equal to that conferred by the heat/phenol-inactivated parenteral vaccine, the only other widely available effective typhoid vaccine.

REFERENCES

1. Bull WHO (1980) 58:469-74
2. J Infect Dis (1982) 145:292-96
3. Lancet (1987) 1:1049-52
4. J Infect Dis (1977) 136:717-23
5. Develop Biol Stand (1983) 53: 9-14

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 1-6-87 to 14-6-87 BULLETIN NUMBER 87/12
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		1	2	1	3	2	15	4	28
0101 ADENOVIRUS TYPE 1				1		1			2
0102 ADENOVIRUS TYPE 2						2			2
0103 ADENOVIRUS TYPE 3						1		1	2
0105 ADENOVIRUS TYPE 5						2			2
0107 ADENOVIRUS TYPE 7		1							1
0112 ADENOVIRUS TYPE 12		1							1
0199 ADENOVIRUS TYPING PENDING					4				4
0301 PARAINFLUENZA VIRUS TYPE 1					1				1
0302 PARAINFLUENZA VIRUS TYPE 2					4			1	5
0303 PARAINFLUENZA VIRUS TYPE 3	1			1	8	2	1	1	14
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)		7	2		12	49	7	19	96
0500 RHINOVIRUS (ALL TYPES)			2	3	2	4	1	6	18
0600 MYCOPLASMA PNEUMONIAE		1				2	1	3	7
0903 COXSACKIEVIRUS B3			1	7		2			10
1005 ECHOVIRUS TYPE 5				1					1
1006 ECHOVIRUS TYPE 6		1							1
1011 ECHOVIRUS TYPE 11				1					1
1013 ECHOVIRUS TYPE 13			1						1
1014 ECHOVIRUS TYPE 14	1								1
1018 ECHOVIRUS TYPE 18	1								1
1025 ECHOVIRUS TYPE 25		1							1
1100 POLIOVIRUS NOT TYPED			1			1	2		4
1101 POLIOVIRUS TYPE 1								1	1
1102 POLIOVIRUS TYPE 2						1		1	2
1103 POLIOVIRUS TYPE 3						1			1
1200 MUMPS VIRUS							1	1	2
1300 HERPES VIRUS GROUP-NOT TYPED	11			3		1	1	1	17
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2							2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)					2	2	4	8	16
1303 VARICELLA-ZOSTER VIRUS				3			1	2	6
1306 HERPES SIMPLEX TYPE 1				28	1	14	29	15	87
1307 HERPES SIMPLEX TYPE 2				61		14	61	52	188
1399 HERPES VIRUS TYPING PENDING					4				4
1401 COXIELLA BURNETI							8		8
1502 PICORNA VIRUS-NOT TYPED			12				12	1	25
1521 MEASLES VIRUS				1	1			4	6
1522 RUBELLA VIRUS							2	1	3
1532 HEPATITIS B ANTIGEN			3	26		28	18	9	84
1535 HEPATITIS A ANTIBODY			1	2		12	1	3	19
1541 CHLAMYDIA A - C TRACHOMATIS						24	3	53	80
1556 CMV - CYTOMEGALOVIRUS	2	1	1	38	4	13	15	23	97
1564 ROTAVIRUS		2	9	2	10	19		3	45
1571 ENTEROVIRUS TYPE 71 (BRCR)				2					2
1599 ENTEROVIRUS TYPING PENDING		1	1		1		1		4
9992 ROSS RIVER VIRUS							31	4	35
9998 ARBO. GROUP B. ...							1		1
Total	16	19	36	181	57	197	216	217	939

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 1-6-87 to 14-6-87 BULLETIN NO 87/12

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.....		1					1				
0102 ADENOVIRUS TYPE 2.....		1		1							
0105 ADENOVIRUS TYPE 5.....		2				1					
0107 ADENOVIRUS TYPE 7.....		1									
0112 ADENOVIRUS TYPE 12.....		1					1				
0301 PARAINFLUENZA VIRUS TYPE 1....		1									
0302 PARAINFLUENZA VIRUS TYPE 2....		5									
0303 PARAINFLUENZA VIRUS TYPE 3....		14									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	100									
0500 RHINOVIRUS (ALL TYPES).....		3					2				
0600 MYCOPLASMA PNEUMONIAE.....	1	5									
0903 COXSACKIEVIRUS B3.....	1	4		2							
1005 ECHOVIRUS TYPE 5.....				1							
1006 ECHOVIRUS TYPE 6.....		1									
1011 ECHOVIRUS TYPE 11.....				1							
1013 ECHOVIRUS TYPE 13.....				1							
1014 ECHOVIRUS TYPE 14.....		2									
1018 ECHOVIRUS TYPE 18.....											1
1025 ECHOVIRUS TYPE 25.....			1								
1200 MUMPS VIRUS.....		1									
1301 HERPES SIMPLEX VIRUS NOT-TYPED											2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	5	1									
1303 VARICELLA-ZOSTER VIRUS.....											6
1306 HERPES SIMPLEX TYPE 1.....		5									46
1307 HERPES SIMPLEX TYPE 2.....											77
1401 COXIELLA BURNETI.....	1	4									1
1521 MEASLES VIRUS.....	1										5
1522 RUBELLA VIRUS.....	1										
1532 HEPATITIS B ANTIGEN.....	31							40	1		
1535 HEPATITIS A ANTIBODY.....	3							10			
1541 CHLAMYDIA A - C.TRACHOMATIS...											5
1556 CMV - CYTOMEGALOVIRUS.....	15	16					3	1	1	4	2
1564 ROTAVIRUS.....							44				
1571 ENTEROVIRUS TYPE 71 (BRCR)....				1		1					1
9992 ROSS RIVER VIRUS.....	7	2									4
Total.....	67	170	1	7		2	51	51	2	4	152

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 1-6-87 to 14-6-87 BULLETIN NO 87/12
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	Gen-ital	Endo/sal gland	RES	Muscle/joint	Con-genit-al	PUO	Fever/mal-aise	Other	SIDS
0103 ADENOVIRUS TYPE 3.....	1								1	
0600 MYCOPLASMA PNEUMONIAE.....								1		
0903 COXSACKIEVIRUS B3.....								5		
1018 ECHOVIRUS TYPE 18.....							1			
1025 ECHOVIRUS TYPE 25.....				1						
1101 POLIOVIRUS TYPE 1.....							1			
1102 POLIOVIRUS TYPE 2.....										2
1103 POLIOVIRUS TYPE 3.....									1	
1200 MUMPS VIRUS.....								2		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			4		1			5	1	
1306 HERPES SIMPLEX TYPE 1.....	4	29							3	
1307 HERPES SIMPLEX TYPE 2.....	1	109	1							
1401 COXIELLA BURNETI.....					1			5		
1532 HEPATITIS B ANTIGEN.....									12	
1535 HEPATITIS A ANTIBODY.....									6	
1541 CHLAMYDIA A - C.TRACHOMATIS...		75								
1556 CMV - CYTOMEGALOVIRUS.....		3	3			2		17	33	1
1564 ROTAVIRUS.....									1	
9992 ROSS RIVER VIRUS.....					21			8	2	
9998 ARBO. GROUP B.					1					
Total.....	6	216	8	1	24	2	2	43	60	3