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Communicable Diseases Intelligence

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Editor Dr I.F. Cook

Contents:

VIRUS REPORTING SCHEME: A total of 1 710 reports were processed for this period.

Fifteen cases of Q fever were reported, 14 from New South Wales and one from Western Australia. No occupational data was available for the reported cases. However none of the 15 patients was involved in the Q fever vaccine field trial conducted in South Australia.

Cytomegalovirus was isolated from :-

- the tracheal aspirate of a 56 year old male who developed pneumonia, septicaemia and a urinary tract infection following a recent renal transplantation,
- the urine of a 23 year old female who developed acute pneumonia subsequent to a severe burn injury to 80% of her body,
- the bronchial washing of a 38 year old male AIDS patient with persistent cough and cryptococcal meningitis.

Rubella specific IgM antibody was detected in the serum of a 35 year old male who presented with a 2 day history of skin rash. The rubella immune status of his wife, who was in the first trimester of pregnancy, is being determined.

Adenovirus type 3 was isolated from the faeces of a 6 month old male with pneumonia complicating a Clostridium difficile infection.

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Herpes simplex virus type 1 was isolated from
. the genital lesions of a 20 year old female who had been sexually assaulted,

. the lips lesions of a 70 year old male with severe herpetic infection of the mucous membranes of the oropharynx.

Herpes simplex virus type II specific IgM antibody was detected in the serum of a 30 year old asymptomatic female whose infant had intermittent febrile convulsions and spenomegaly.

POTENTIAL QUARANTINE CASE - AUSTRALIA
(Contributed by Dr N. McK. Bennett - Fairfield Hospital - Melbourne).

A 53 year old man travelling by air from Nigeria notified airport authorities on his arrival in Perth that he was feeling unwell, and when he disembarked at Sydney airport on 12 June 1987, he was medically assessed, ordered into quarantine and admitted to the Infectious Diseases Unit in Prince Henry Hospital (Sydney) for observation.

Medical history revealed that, after travelling through Nigeria, Zimbabwe and Zaire, the patient became ill with initial symptoms of fever, myalgia and weakness on 9 June 1987, whilst visiting Zambia. As these symptoms were similar to those of malaria which he has experienced in the past, he commenced self-medication of chloroquine followed by amodiaquine the next day. He did not respond to self-medication and 2 days after the onset of fever, he developed a sore throat.

As VHF could not be excluded the patient was ordered into quarantine. On admission to Prince Henry Hospital, a blood film showed scanty malarial parasites (P. falciparum) and treatment with Fansider and quinine was initiated. Despite treatment, fever and severe, painful pharyngitis persisted. An aeromedical evacuation of the patient to the National High Security Quarantine Unit (NHSQU) at Fairfield Hospital, Melbourne, was effected on 14 June 1987 since it was considered that complete isolation in quarantine was required until Lassa Fever had been excluded.

On arrival at Fairfield Hospital, the patient was found:
. to be febrile, with a temperature of 39.4°C,
. to have a faint macular rash on his trunk with scanty 6-8mm diameter lesions over the abdomen and lower anterior thorax.
. to have pharyngeal inflammation with tonsillar enlargement and
  - exudate formation on the left tonsil,
  - two shallow ulcers on the top of the left tonsillar pillar.

No adenopathy was detected and the remaining examination was unremarkable apart from a "weepy" rash in the inguinal skin folds.

Laboratory investigations including throat swabs, liver function tests and VHF serology were unremarkable, except for a blood glocose level of 22 mmol/1, reflecting poorly controlled Type II diabetes mellitus.

Treatment was initiated with oral penicillin V, and oral quinine which was continued. The patient was afebrile and constitutionally better within 24 hours of the commencement of penicillin therapy.

Quarantine was continued until the 8th day of his illness when he was asymptomatic. Repeated VHF serological tests were negative for:

- Congo Crimea virus
- Rift Valley virus
- Ebola virus
- Lassa Fever virus
- Marburg Virus.

DISSEMINATED GONORRHOEA CAUSED BY PENICILLINASE - PRODUCING NEISSERIA GONORRHOEAE - WISCONSIN, PENNSYLVANIA

(Based on MMWR Vol 36/No 11, 27 March 1987)

During the period August-September 1986, the Centers for Disease Control (CDC) received four reports of disseminated gonococcal infection caused by penicillinase-producing Neisseria gonorrhoeae (PPNG).

Case 1: A 28 year old female was admitted to a hospital in Racine on 4 August 1986, with a one-week history of arthritis of the left knee with effusion. Synovial and cervical cultures were both positive for PPNG. She was treated for 2 days with intravenous penicillin. When the culture results became known, her therapy was changed to ceftriaxone, 500mg once daily. Despite the change in therapy, the knee remained swollen. Even though the dosage of ceftriaxone was increased to 1g every 12 hours, the knee had to be surgically drained on 14 August. The patient recovered rapidly and was discharged one week later.

Case 2: is the above patient's only recent sexual partner. When examined on 8 August he had had urethritis for one week and a swollen, painful left wrist for 2 weeks. Nine days earlier, he had been treated for the wrist symptoms with a non-steroidal, anti-inflammatory agent. Upon examination, the patient had purulent urethritis and a tender, slightly swollen wrist. Urethral culture was positive for PPNG. Synovial fluid from the wrist was not cultured. The patient was treated intramuscularly with 2g of spectinomycin and recovered completely.

Case 3: A 20 year old female presented to an emergency room in a Philadelphia hospital with a one week history of

wrist pain and pain in the right knee, left ankle, and the dorsum of the left hand for 3 days. On physical examination, she was febrile, had tenosynovitis of the extensor tendons of the left hand, and had effusion of the right knee and ankle.

Arthrocentesis of the knee yielded purulent fluid which grew PPNG. A cervical culture was also positive for PPNG. Initially the patient was treated intravenously with penicillin; therapy was changed to cefotaxime when culture results became available and she recovered completely.

Case 4: A 52 year old female presented at a Philadelphia emergency room with a 2 day history of pain in the right wrist and third finger of the left hand. She was febrile, and the right wrist and proximal inter-phalangeal joint of the left third finger were swollen and tender. Arthrocentesis of the wrist yielded purulent fluid that grew PPNG. The patient was treated intravenously with penicillin. Therapy was changed to intravenous ceftriaxone when culture results became available. She recovered completely.

Antibiotic - susceptibility testing, auxotype, protein I serovar determination, and plasmid analysis of isolates from all patients were performed at the Centers for Disease Control (Atlanta):

- all isolates were resistant to penicillin (minimum inhibitory concentration [MIC] 1-8μg/ml)
- all demonstrated moderate chromosomally mediated resistance to:
  - tetracycline (MIC range; 0.5-4.0μg/ml), and
  - cefoxitin (MIC range: 0.5-4.0μg/ml),
- all were sensitive to spectinomycin and ceftriaxone,
- all isolates were auxotype/serovar class Pro-/IA-6, and
- all contained the 2.6 mega Dalton (mDal) cryptic plasmid, the 3.2 mDal β-lactamase plasmid, and the 24.5mDal conjugative plasmid.

Despite the similarity of the isolates, suggestive of a clonal origin, no linkage could be demonstrated between the two Philadelphia patients or between either Philadelphia patient and the Wisconsin patients.

MMWR Editorial Note:

Disseminated gonococcal infection, a serious complication of gonorrhoea, is estimated to occur in 0.5%-1.0% of all gonococcal infections. Tenosynovitis and septic arthritis are the two most common clinical syndromes[1].

Published reviews have reported that disseminated gonococcal infection is predominantly caused by organisms which are extremely susceptible to antibiotics and are more likely to be nutritionally fastidious, requiring arginine, hypoxanthine, and uracil for growth ($A^-H^-U^-$ auxotype)[2-4]. This may have led to the mistaken impression that antibiotic-resistant strains of N. Gonorrhoeae do not cause disseminated gonococcal infection.

Cases of disseminated gonococcal infection caused by PPNG are being reported more frequently[5-7]. There have also been reports of disseminated gonococcal infection caused by gonococci with chromosomally mediated resistance to penicillin[8]. Furthermore, in a recent, large prospective study, disseminated gonococcal infection isolates were no more susceptible to antibiotics than isolates from localised anogenital gonorrhoea[9]. Patients with disseminated gonococcal infection caused by resistant gonococcal strains should be hospitalised and treated with ceftriaxone (1-2g/day intravenously) until signs and symptoms resolve. Daily outpatient therapy with either ceftriaxone (250 mg intramuscularly) or an oral regimen defined either by in-vitro susceptibility tests should follow, for at least one week of antimicrobial therapy. When an infection does not respond to appropriate antimicrobial therapy, surgical drainage should be considered.

Less than 50% of synovial-fluid cultures in gonococcal arthritis are positive. Therefore, antibiotic-resistant N.gonorrhoeae should be considered in culture-negative, clinically diagnosed cases of gonococcal arthritis that do not respond to standard antimicrobial therapy.

In 1986, 16,608 PPNG infections were reported to the Centers for Disease Control (Atlanta)[10], a 90% increase from 1985. As the incidence of PPNG and other resistant strains increases, there is likely to be an increase in the incidence of disseminated gonococcal infection caused by antibiotic-resistant N.gonorrhoeae.

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ORAL POLIOMYELITIS VACCINE (OPV) - ASSOCIATED POLIOMYELITIS - AUSTRALIA - A REVIEW.

Introduction

In reviewing the deployment of OPV as a public health tool in controlling poliomyelitis in Australia, a number of issues relating to vaccine efficacy, safety and public health impact should be evaluated against the background of a virtual elimination of poliomyelitic diseases caused by wild poliovirus[1].

For practical reasons, Australia, like a number of Western developed countries, has adopted the use of OPV for at least the last two decades. The preferential use of OPV over IPV (inactivated poliovirus vaccine) is based on:

. OPV lower cost,
. OPV ease of administration,
. OPV rapid induction of both circulating humoral antibodies, and local intestinal mucosal immunity, thus
  - providing local resistance to subsequent infection with wild poliovirus and
  - reducing the number of symptomless excretors of wild poliovirus in the community.

However, disadvantages of OPV reside in:

. OPV contra-indication in immunodeficient or immunosuppressed persons
. OPV potential for rare paralytic disease in recipients and in contacts in the household and in the community.

The latter disadvantage tends to currently pre-occupy the public health advocacy, since naturally acquired poliomyelitis has now become so rare in economically developed countries that the likehood of acquiring this disease may be exceeded by the risk of rare OPV - associated paralytic infection. This situation has engendered considerable controversy over the relative merits of IPV and OPV and led the Communicable Diseases Committee (CDC) to a reiterate the national immunisation policy in relation to OPV.

Situation in Australia

The number of reactions to OPV which the Adverse Drug Reaction Advisory Committee (ADRAC) commented as being causally possible averaged 2 cases per year, for the past 3 years (Jan 1984 -March 1987). The Therapeutics Goods Compliance Branch of the Commonwealth Department of Health had also indicated that during the same period the number of OPV doses used averaged 2 million doses per year. Hence the overall frequency of OPV-associated reactions, as reported by ADRAC, would average 1 case per million doses used.

It should be noted however that the above incidence reflects the rate of reactions to OPV and not the number of cases of paralytic poliomyelitis. In fact, the rate of OPV-associated poliomyelitis is very rare. During the period between August 1984 and March 1987, only one case of flaccid paralysis was recorded by ADRAC in June 1982, a 12 month old male who had received Sabin OPV.

The rate of OPV-associated poliomyelitis is apparently much less than in the United States, with a reported overall frequency of one case per 2.6 million doses distributed (2). However, this probably reflects the limitations of ADRAC reporting scheme.

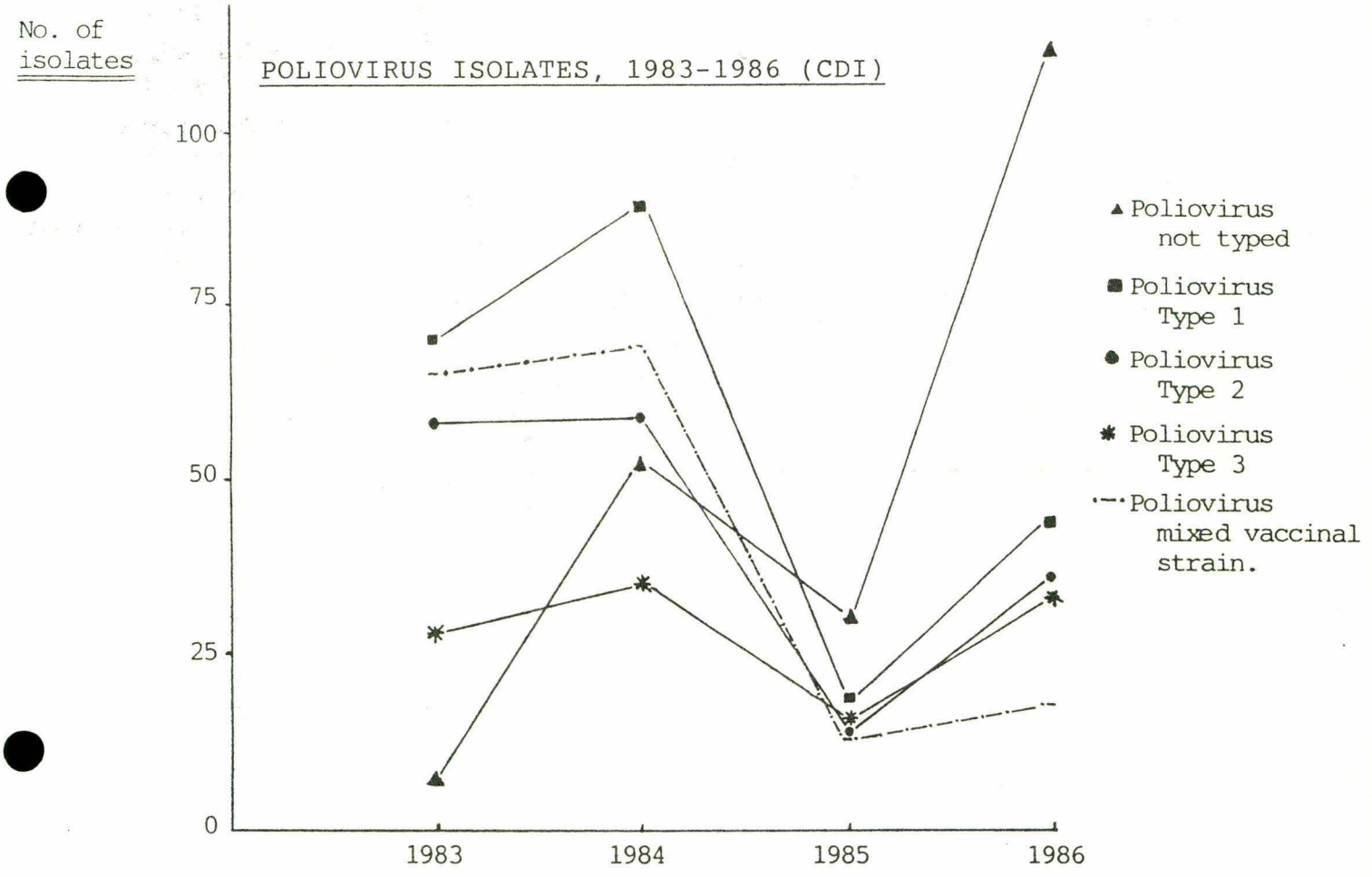
The CDI (Communicable Diseases Intelligence) bulletin, as part of its routine fortnightly survey of ten major laboratories across Australia, has recorded the following number of poliovirus isolates:

. 1983 - 293 isolates reported

. 1984 - 304 isolates reported
. 1985 - 67 isolates reported
. 1986 - 242 isolates reported

The distribution of different strains of poliovirus isolates reported during the period January 1983 - December 1986 is depicted in figure 1. (NB. Isolates were recovered from cases with significant morbidity which necessitated laboratory investigation)

FIGURE 1:

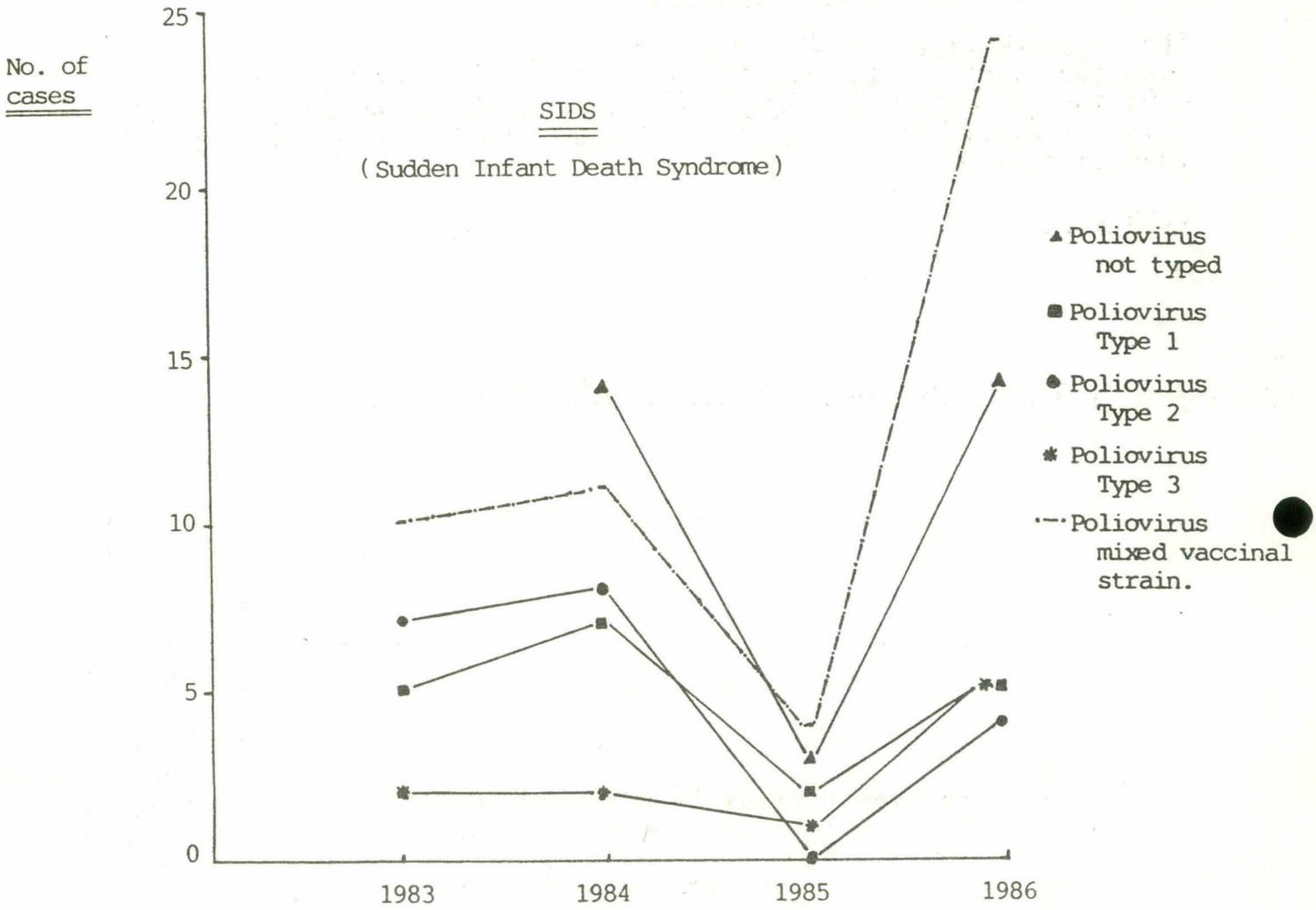


For the purpose of comparison the number of Sudden Infant Death Syndrome (SIDS) cases, from whom various strains of poliovirus were isolated at post-mortem, were notified to CDI during the period January 1983 - December 1986:

. 1983 - 24 cases of SIDS were reported
. 1984 - 42 cases of SIDS were reported
. 1985 - 10 cases of SIDS were reported
. 1986 - 32 cases of SIDS were reported

The distribution of SIDS cases according to various strains of poliovirus isolates is depicted in Figure 2.

FIGURE 2:



Current International Policies

In the light of the current debate on OPV-associated poliomyelitis, international effort appears to concentrate heavily on case detection and case exposition rather than determining the cause and effect relationship between OPV and OPV-associated poliomyelitis[1, 3, 12].

The reactions of various countries towards OPV immunisation are summarised below:

A. In communist countries such as Hungary [4] where a national health care delivery can achieve universal vaccination, the estimated risks for each type of monovalent OPV vaccines, administered in the sequence of type 1, 3 and 2 are as follow:
   . in recipients receiving the vaccine for the first time
      - 1 case per 10^6 doses due to type 1 vaccine
      - 0.65 case per 10^6 doses due to type 2 vaccine
      - 9 cases per 10^6 doses due to type 3 vaccine

. in susceptible contacts
  - nil case due to type 1 vaccine
  - 3.62 cases per 10^6 doses due to type 2 vaccine
  - 5 cases per 10^6 doses due to type 3 vaccine

The health authorities concluded that such rates of risk are acceptable and their current OPV immunisation practices need not be altered in view of
  - the benefits provided by the live vaccine
  - the circumstances where importation of wild polioviruses may still occur from extended regions of the world.

B. In Japan, where Sabin OPV has been in use since 1961, poliomyelitis control has been routinely monitored and OPV vaccine preparation annually evaluated and tested for safety.

In one study, Shimojo (5) noted that the number of paralytic cases associated with OPV was:
  - low for cases due to type 1 virus
  - high for cases due to type 2 virus
  - high until 1969 and low since 1970 for cases due to type 3 virus. This difference was brought about by changing the batch of type 3 virus used in the preparation of OPV.

The analysis of titres of neutralising antibody showed that response
  - to type 1 virus in OPV used in 1977, and
  - to type 3 virus used in 1972 and 1973

was not adequate. Both situations were improved by changing the batches of virus used for OPV.
Although a correlation between the preparation of vaccines and the frequency of vaccine-associated cases was discussed, no reason was given for the different frequencies of paralysis induced by different vaccine preparations. These experiences suggest however that the use of appropriate batches is necessary for safety and efficacy of OPV.

In another study, Chino et al(6) evaluated the neurovirulence of OPV used in Japan during 1963 - 1982, using a five-dilution method for determining (Sabin) LID_{50} (neurovirulence for the lesion-inducing virus dose in the spinal cord in 50% of monkeys inoculated with Sabin OPV) to estimate safety and efficacy of the vaccine upon administration to children. They concluded that the degree of attenuation of the vaccine in terms of LID_{50} appeared to be related to the decrease in the vaccine-associated cases as well as the seroconversion rate. However, the five-dilution method failed to classify lot 301 Sabin OPV into less attenuated vaccine by the neurovirulence test (the frequency of vaccine-associated cases was 10 times higher than that with the other vaccines) suggesting that the five-dilution method may be too insensitive. WHO (World Health Organisation) had recommended a one-dilution method; comparative studies are currently being conducted.

It is worth noting that retrospective enquiries have revealed that lot 301 vaccine has been prepared at the level of Sabin original (SO)+3 ie three in vitro cell passages whereas other types of vaccine were prepared at the level of (SO)+2 ie two in vitro passages.

Antigenic modification of attenuated Sabin type 1 poliovirus by in vitro passages at supraoptimal temperatures has been demonstrated by Crainic et al [7] in mutants (LSc2ab strain) capable of growing at high temperatures (39.5°C). This mutant continued to be neuroviralent in spite of the complete conversion of its neutralisation epitope formula to the Sabin virus pattern.

C. A number of developing countries including Brazil[8] and Africa[9, 10] which have adopted OPV as their mass immunisation practices against poliomyelitis, have reported spectacular successes which warrant the continuation of OPV immunisation strategy for the foreseeable future. However, India[11] has reported a failure to control poliomyelitis using OPV as a result of inappropriate immunisation strategy and low rates of antibody response to vaccine virus. The switch over to IPV cannot be effected because of high cost. Meanwhile the current annual immunisation strategy using OPV (which was found to be highly effective when used in pulse immunisation) is still recommended for national control of poliomyelitis.

Developed countries including the United States[12] have maintained that vaccine-associated paralytic poliomyelitis is rare and the risk due to OPV is small. The greatest likelihood of paralysis occurs in association with the 1st dose of OPV and that likelihood is reduced in subsequent doses more for the recipients than for their contacts.

Discussion

Health authorities in Holland, Finland and Sweden (which have used IPV exclusively) would undoubtedly argue that OPV vaccine-associated poliomyelitis could be avoided by widespread adoption of formalin-fixed vaccine as Salk has suggested[13]. Experience with this vaccine in Finland and Sweden has shown excellent protection from paralytic disease, eradication of wild type virus and very few side effects[14, 15]. The question remains whether the results would be as good in larger countries (-larger in terms of population such as the United States - or larger in terms of geographical areas such as Australia) where the logistics of health care delivery is much more complex.

Most public health authorities, including those in Australia, would consider the small risk associated with the oral trivalent vaccine (OPV) to be acceptable. The present concern however, emphasises that warnings of risks associated with OPV use should not be limited to parents but should include other nonimmune adults who have close contact with vaccinees, since it has been established that:

1. paralytic poliomyelitis is more likely to occur in nonimmune adult contacts than in infants who receive the vaccine[2] hence it has been suggested that

susceptible parents be given OPV along with their infants [16],

2. the attenuated virus used for immunisation can become virulent after passage in vaccinees [17],
3. most of the recent contact cases have been due to type 2 and type 3 possibly indicating a greater propensity for the attenuated type 2 and type 3 virus to become more virulent after passage through vaccinees [4, 18].

Conclusion

Based on the currently practised immunisation policy in Western countries such as the United States [19], Canada, England and Europe, a change in the current NH&MRC (National Health and Medical Research Council) schedule of national immunisation policy [20] in relation to OPV use in Australia does no seem warranted.

However, given
(1) the increasing concern in relation to the prevalence of possible cases of OPV-associated poliomyelitis;
(2) the possibility that Sabin original poliovirus undergoing 3 or more in vitro cell passages may become intolerably more neurovirulent, unlike other viruses which become more attenuated on subsequent in intro cell passages and;
(3) Sabin poliovirus vaccine strains increase in neurovirulence after passages through vaccinees;
the following measures may need to be considered to monitor the safety and adverse effects of OPV use:

1 ADRAC may need to increase scrutiny its current register to detect each case of suspected OPV-associated poliomyelitis in order to determine:
- whether the case occurred in
  . recipient (if recipient, ascertain which dose administered)
  . contact (define contact and identify
    1) recipient
    2) when was OPV given
    3) age of contact person
    4) immune status of contact in relation to poliomyelitis
  . immune deficient individuals (contact/recipient)
  . others (attempt to define history of receiving OPV or contact with recent OPV recipients)

- which type of poliovirus the case has been infected with (ie poliovirus type 1, 2 or 3 or mixed vaccinal strain or wild type poliovirus or a mutated form of the vaccinal strains of Sabin type poliovirus) by examining laboratory results on:
  1) viral identification and typing
  2) serological/immunological profile
  3) virus isolation and characterisation including oligonucleotide mapping.

- the infectivity and pathogenicity (including neurovirulence) of the poliovirus strain isolated/recovered from the case as evidenced by:
  1) clinical/anatomical signs and symptoms
  2) laboratory tests results
  3) serological/immunological profile
  4) literature and scientific reports

2 National Biological Standards Laboratory (NBSL) - Virology to consider regularly reviewing, monitoring, enquiring and commenting on neurovirulence data of each batch of Sabin OPV (manufactured by Smith Kline & French and distributed by the Commonwealth Serum Laboratories) used in Australia, in particular in relation to the effect of various cell passages.

Ideally NBSL would consider conducting neurovirulence tests on suspect batches of Sabin OPV. However, present financial constraint would prevent NBSL from accommodating such a request.

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MEASLES OUTBREAK CONTINUES - CALIFORNIA (USA)

(Based on California Morbidity #16, 1 May 1987)

By late April 1987, nearly 500 cases of measles have been recorded in California's largest measles outbreak of the past decade. The outbreak, of several months duration, began in November 1986 in the Lake Isabella area of Kern County, spreading westward into Bakersfield and eastward into the desert communities of Tehachapi, Mojave and Ridgecrest, and then into northern San Bernadino and Los Angeles counties.

Most spread has been in secondary schools:
- 57% of cases have been aged 10-19 years, and almost
- 75% of cases have been non-preventable, mostly because they have a personal or school record indicating measles immunisation on or after the first birthday.

Since several recent studies indicated higher attack rates for persons immunised between ages 12 and 14 months, outbreak control efforts in schools with cases began with mandatory exclusion (unless they accepted reimmunisation) of pupils immunised at this age. Also excluded, on a community wide basis, were pupils lacking a legally adequate school measles immunisation record indicating at least the month and year of immunisation received on or after the first birthday. When provisional data suggested no increased attack rate for pupils immunised between ages 12 and 14 months in the outbreak, exclusion of these pupils was stopped.

As part of the outbreak control program in Kern County, records of over 35,000 pupils in 53 schools were audited between November 1986 and April 1987 - 6% of pupils were found to have legally inadequate immunisation records or exemptions to immunisation and were given school exclusion noticed. Despite these efforts, the outbreak moved rapidly through multiple secondary schools.

To evaluate measles attack rates in relation to school immunisation record status, a study was made in the two high schools initially affected. A total of 36 cases occurred among 2403 pupils:
- unimmunised pupils with personal beliefs exemptions to immunisation comprised only 0.5% of the student body but had an attack rate of 38.5% and accounted for 14% of the cases;
- pupils with legally inadequate records (eg incomplete or absent records, immunisation before the 1st birthday) comprised 8% of the cases;
- pupils with legally adequate records made up 93% of the student body and had an attack rate of 1.3%, 95% lower than that for pupils with exemptions; yet they accounted for 78% of the cases in these schools.

It is somewhat startling that such large measles outbreaks can occur in schools with high immunisation levels. However, it has been demonstrated that opportunities for exposure are sufficiently great in secondary schools that sizable measles outbreaks can occur even when over 99% of pupils have immunisation records and over 95% are actually seropositive for measles antibody[1].

Attempts were made to identify subgroups of pupils with legally adequate records but still at higher risk:
- pupils immunised between ages 12 and 14 months had an attack rate of 2.1%, compared to 1.0% for pupils immunised at age 15 months and older. Such pupils accounted for 20% of the student body and 28% of the cases,
- pupils immunised prior to 1979 (a better stabilising agent was added to measles vaccine in 1979(had an attack rate of

1.6%, compared to zero for those immunised in 1979 or later (p 0.005). Pre-1979 vaccinees (which obviously also included all pupils immunised at ages 12-14 months) comprised 72% of the student body and accounted for 78% of the cases - including all cases in pupils with legally adequate immunisation records.

It is possible that directing re-immunisation efforts at pupils immunised at ages 12-14 months and/or before 1979, in addition to pupils with legally inadequate records or with exemptions to immunisation, may help control school measles outbreaks.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 15-6-87 to 28-6-87 BULLETIN NUMBER 87/13
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	12	1	5		3	1	13	1	36
0101	ADENOVIRUS TYPE 1	1	1				2			4
0102	ADENOVIRUS TYPE 2				2				3	5
0103	ADENOVIRUS TYPE 3	1					1		2	4
0105	ADENOVIRUS TYPE 5						1			1
0106	ADENOVIRUS TYPE 6								2	2
0107	ADENOVIRUS TYPE 7	1								1
0109	ADENOVIRUS TYPE 9								1	1
0112	ADENOVIRUS TYPE 12	1								1
0122	ADENOVIRUS TYPE 22	1								1
0199	ADENOVIRUS TYPING PENDING	2	1			2				5
0201	INFLUENZA A VIRUS	6		4						10
0203	INFLUENZA B VIRUS	1				1				2
0301	PARAINFLUENZA VIRUS TYPE 1				1	3	1			5
0302	PARAINFLUENZA VIRUS TYPE 2					1		2	2	5
0303	PARAINFLUENZA VIRUS TYPE 3	4				9	3	9		25
0399	PARAINFLUENZA VIRUS TYPING PENDING	1				1				2
0400	RESPIRATORY SYNCYTIAL VIRUS (RS)	31	9	6	1	14	56	27	19	163
0500	RHINOVIRUS (ALL TYPES)	1		1	3	10	2	1	4	22
0600	MYCOPLASMA PNEUMONIAE	12				1	1		4	18
0700	ORNITHOSIS-PSITTACOSIS	1								1
0901	COXSACKIEVIRUS B1								3	3
0902	COXSACKIEVIRUS B2						1			1
0903	COXSACKIEVIRUS B3	5	1	1	1	4				12
1003	ECHOVIRUS TYPE 3	2								2
1005	ECHOVIRUS TYPE 5	4								4
1006	ECHOVIRUS TYPE 6	1								1
1009	ECHOVIRUS TYPE 9	2								2
1011	ECHOVIRUS TYPE 11				4	1				5
1012	ECHOVIRUS TYPE 12								1	1
1014	ECHOVIRUS TYPE 14	2								2
1018	ECHOVIRUS TYPE 18	1				3				4
1025	ECHOVIRUS TYPE 25	1								1
1029	ECHOVIRUS TYPE 29								1	1
1100	POLIOVIRUS NOT TYPED					3		1		4
1101	POLIOVIRUS TYPE 1								1	1
1102	POLIOVIRUS TYPE 2				1					1
1200	MUMPS VIRUS								1	1
1300	HERPES VIRUS GROUP-NOT TYPED	31						1	3	35
1301	HERPES SIMPLEX VIRUS NOT-TYPED	1	3		1				1	6
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	11	2		1	1			13	28
1303	VARICELLA-ZOSTER VIRUS	6		2	1		1		2	12
1306	HERPES SIMPLEX TYPE 1	38		6	40		15	46	26	171
1307	HERPES SIMPLEX TYPE 2	189		27	59		17	54	74	420
1399	HERPES VIRUS TYPING PENDING					2				2
1401	COXIELLA BURNETI	14							1	15
1502	PICORNA VIRUS-NOT TYPED	32	1	7				12		52
1521	MEASLES VIRUS								1	1
1522	RUBELLA VIRUS	3			2		2		3	10
1532	HEPATITIS B ANTIGEN	121		3	18		9	24	9	184
1535	HEPATITIS A ANTIBODY	8			9		1		2	20
1541	CHLAMYDIA A - C TRACHOMATIS	63		2	15		20	34	51	185
1543	CHLAMYDIA A - LGV TYPE	2						8		10
1556	CMV - CYTOMEGALOVIRUS	6	2	11	36	5	8	1	8	77
1564	ROTAVIRUS	24	2	12	3	10	26	13	7	97
1599	ENTEROVIRUS TYPING PENDING	2		7		8				17
9901	ARBO. GROUP A.(UNSPECIFIED)						1			1
9992	ROSS RIVER VIRUS	3							1	4
9994	SMALL VIRUS (LIKE) PARTICLE	2								2
9995	DENGUE								1	1
Total		650	23	94	198	82	169	246	248	1,710

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 16-6-87 to 28-6-87 BULLETIN NO 87/13

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					2				
0102 ADENOVIRUS TYPE 2.....		3							1		
0103 ADENOVIRUS TYPE 3.....		3									
0105 ADENOVIRUS TYPE 5.....		1									
0106 ADENOVIRUS TYPE 6.....	1										1
0107 ADENOVIRUS TYPE 7.....							1				
0112 ADENOVIRUS TYPE 12.....	1										
0201 INFLUENZA A VIRUS.....	2	4									
0203 INFLUENZA B VIRUS.....		1	1								
0301 PARAINFLUENZA VIRUS TYPE 1....		5									
0302 PARAINFLUENZA VIRUS TYPE 2....	1	4									
0303 PARAINFLUENZA VIRUS TYPE 3....		25									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	14	147									1
0500 RHINOVIRUS (ALL TYPES).....	1	3									
0600 MYCOPLASMA PNEUMONIAE.....	4	13									
0901 COXSACKIEVIRUS B1.....		1									
0902 COXSACKIEVIRUS B2.....				1							
0903 COXSACKIEVIRUS B3.....	1	4	1	5			2				
1003 ECHOVIRUS TYPE 3.....			1	1							
1005 ECHOVIRUS TYPE 5.....	1		1	1			1				
1006 ECHOVIRUS TYPE 6.....		1									
1009 ECHOVIRUS TYPE 9.....		1		1							
1011 ECHOVIRUS TYPE 11.....			1	3							1
1014 ECHOVIRUS TYPE 14.....		1		1							
1018 ECHOVIRUS TYPE 18.....		1									1
1025 ECHOVIRUS TYPE 25.....			1								
1102 POLIOVIRUS TYPE 2.....		1									
1200 MUMPS VIRUS.....						1					
1301 HERPES SIMPLEX VIRUS NOT-TYPED											4
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	9							4			
1303 VARICELLA-ZOSTER VIRUS.....	1							1			8
1306 HERPES SIMPLEX TYPE 1.....	8	5								1	8
1307 HERPES SIMPLEX TYPE 2.....	23							1			92
1401 COXIELLA BURNETI.....	9										
1521 MEASLES VIRUS.....	1										
1522 RUBELLA VIRUS.....	1										5
1532 HEPATITIS B ANTIGEN.....	79							99			
1535 HEPATITIS A ANTIBODY.....	7							13			
1541 CHLAMYDIA A - C.TRACHOMATIS...	22										
1543 CHLAMYDIA A - LGV TYPE.....	8	2									
1556 CMV - CYTOMEGALOVIRUS.....	12	16		1		1		2	1	7	3
1564 ROTAVIRUS.....	8						88				
9992 ROSS RIVER VIRUS.....	2										
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
Total.....	216	243	6	14		2	96	120	2	8	200

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 15-6-89 to 28-6-87 BULLETIN NO 87/13
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
0101 ADENOVIRUS TYPE 1.....										1
0102 ADENOVIRUS TYPE 2.....										1
0103 ADENOVIRUS TYPE 3.....	1									
0106 ADENOVIRUS TYPE 6.....	1									
0109 ADENOVIRUS TYPE 9.....	1									
0122 ADENOVIRUS TYPE 22.....		1								
0201 INFLUENZA A VIRUS.....					1		1	1	1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....							3		1	
0600 MYCOPLASMA PNEUMONIAE.....				1				1		
0700 ORNITHOSIS-PSITTACOSIS.....									1	
0901 COXSACKIEVIRUS B1.....							1	2		
0903 COXSACKIEVIRUS B3.....								1		
1003 ECHOVIRUS TYPE 3.....									1	
1011 ECHOVIRUS TYPE 11.....								1		
1012 ECHOVIRUS TYPE 12.....										1
1018 ECHOVIRUS TYPE 18.....								2		
1029 ECHOVIRUS TYPE 29.....										1
1100 POLIOVIRUS NOT TYPED.....										1
1101 POLIOVIRUS TYPE 1.....									1	
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			5	2				6	3	
1303 VARICELLA-ZOSTER VIRUS.....			1						1	
1306 HERPES SIMPLEX TYPE 1.....	3	69					1			
1307 HERPES SIMPLEX TYPE 2.....		304		1						
1401 COXIELLA BURNETI.....							1	2	3	
1502 PICORNA VIRUS-NOT TYPED.....									1	
1522 RUBELLA VIRUS.....			2		1				1	
1532 HEPATITIS B ANTIGEN.....		2		1					3	
1541 CHLAMYDIA A - C.TRACHOMATIS...		162							1	
1556 CMV - CYTOMEGALOVIRUS.....	1	1		1		4	2		28	
1564 ROTAVIRUS.....									1	
*9992 ROSS RIVER VIRUS.....					2					
9995 DENGUE.....								1		
Total.....	7	541	9	5	4	4	9	17	47	5

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 1 - 1 January 1987 to 24 January 1987

Bulletin 87/13

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis				2				1	3	3
Ankylostomiasis					2		NN		2	2
Anthrax										
Arbovirus infection			21		5				26	26
Brucellosis			2						2	2
Campylobacter infections	175		NN	79	16	NN	2	NN	272	272
Chancroid				NN						
Cholera										
Congenital rubella syndrome			NN			NN		NN		
Diphtheria			1				2		3	3
Donovanosis			4	NN	7		2		13	13
Giardiasis	26	1	NN	41	21	NN	NN	NN	89	89
Genital herpes	56		29		NN	NN		NN	85	85
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN		
Gonorrhoea	89		109	53	131	2	24	10	418	418
Hepatitis A (infectious)	18	5	11	14	14				62	62
Hepatitis B (serum)	34	17	38	10	28				127	127
Hepatitis – unspecified	5		1	2	NN	NN			8	8
Hydatid disease										
Lassa fever			NN			NN		NN		
Legionnaires disease	1		NN			NN		NN	1	1
Leprosy					1				1	1
Leptospirosis	2	3	3	1		3			12	12
Lymphogranuloma venereum					NN	NN		NN		
Marburg disease			NN			NN		NN		
Malaria	8	8	16	5	4		1		42	42
Meningococcal infections	1			3	5	NN			9	9

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	234		NN	NN	NN	NN	NN	NN	234	234
Ornithosis										
Pertussis (whooping cough)		1	NN	4	59	NN		NN	64	64
Plague										
Poliomyelitis										
Q. fever	10		13						23	23
Rabies				NN		NN		NN		
Salmonella infections	94	9	49	24	27	5	7	1	216	216
Shigella infections	19	2	11	2	8		5		47	47
Smallpox										
Syphilis	30		43	3	24		15	2	117	117
Tetanus										
Trachoma		NN	1		3	NN	NN		4	4
Tuberculosis (all forms)	14	29	12	4	11			1	71	71
Typhoid fever	7								7	7
Typhus (all forms)										
Vibrio parahaemolyticus infections			NN			NN		NN		
Yellow fever										
Yersinia infections	9		NN	1		NN		NN	10	10

NN - Not Notifiable

(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.)