ISSN 0725 - 3141

Communicable Diseases Intelligence

Bulletin number Vol. 15/No. 1
Issue date: 14 January 1991

Contents:

Editor Dr Marcus Hodge

NOTICE TO READERS

This issue sees the change to a two column format with the introduction of desktop publishing.

Issues will now be identified by volume, number and date. As this is the 15th year of publication, this issue is Number 1 of Volume 15 (14 January 1991). The order of contents has also been altered. The notes on the CDI Reporting Scheme now appear before the period report tables. Comments on these changes are welcomed.

Further minor alterations are likely when a new masthead is adopted around March 1991.

EDITORIAL STAFF:

- The Bulletin is compiled and distributed by the Communicable Diseases Section, Communicable Diseases and Social Health Branch, Telephone: (062) 89 1555, Department of Community Services and Health.
- Contributions are solicited, and do not preclude later publication elsewhere.
- Material appearing in the Bulletin is subject to Commonwealth copyright, which is administered by the Australian Government Publishing Service.
- Figures given may be subject to revision.

OVERSEAS BRIEFS

1. CHOLERA VACCINATION REQUIREMENTS FOR TRAVELLERS TO SUDAN

The WHO reports (WER 1990;47:23 November) that the Government of Sudan no longer requires a cholera vaccination certificate from international travellers. Pakistan and Pitcairn are the only country and territory still requiring a cholera vaccination certificate from travellers arriving from infected areas.

2. CHOLERA IN ZAMBIA

As at 21 December 1990 the WHO reported cholera in the northern Luapula and Copperbelt provinces of Zambia. A total of 429 cases with 49 fatalities have occurred in these provinces.

3. CHOLERA FREE - MOROCCO, ROMANIA

As at 14 December 1990 Morocco and Romania have declared all their territory to be free from cholera.

CHEMOPROPHYLAXIS OF HAEMOPHILUS INFLUENZAE TYPE B INFECTION

(Reproduced with acknowledgement from Monthly Infectious Diseases Report, RAHC February 1990 (4), editor D Isaacs)

Haemophilus influenzae type b (Hib) is the commonest cause of meningitis in childhood and this is predominantly a disease of infants and young children. The main reason for this being the age-group most often affected is that immunity to Hib is predominantly via the production of antibodies against the type b capsule which is composed of polysaccharide. Children under 2 years of age are poor at producing antibodies to polysaccharide, although in the first six months of life the incidence of Hib infection is low due to passive maternal antibody. Immunisation with Hib capsular polysaccharide (PRP) can only protect children over 2 years of age. The new conjugate vaccines are looking highly promising in inducing the production of antibodies by young babies but their introduction in Australia looks to be several years away. Until that time chemoprophylaxis must remain the principle means of preventing secondary cases of Hib infection.

Chemoprophylaxis is based on the pathogenesis of Hib infection. Around 2% of the population are nasopharyngeal carriers of Hib. In some children, however, acquisition of nasal carriage is rapidly followed by invasion, bacteraemia, and above a certain level of bacteraemia by meningitis. Concomitant viral infections of the upper respiratory tract may predispose to invasion. When a child presents with systemic Hib infection, whether meningitis, epiglottitis, facial cellulitis, septic arthritis or septicaemia without a focus there are three groups that should be considered:-

i) Household contacts: the risk to family contacts of an the population rate) and negligible over 4 years of age[1].

ii) Day care contacts: the risk to day care contacts is age-dependent and depends on the intensity of exposure (duration of contact), but there are also unexplained geographical variations. There were no secondary Hib cases in day care units in Mineapolis[2], but 2.7% of exposed Seattle day care children under 2 years old developed Hib disease[3], about the same rate as for family contacts. Colonization rates in small day care centres (less than 10 children) with an index case of Hib disease in Pittsburgh were higher than in households[4]. The highest risk for disease is in contacts younger than 2 years of age: in Seattle the risk was 2.4% aged 0-11 months, 1.2% aged 12 to 23 months and zero (0 of 738) for children aged 25 to 60 months. No child who attended day care less than 25 hours a week developed secondary disease[3].

iii) Index case: If the index case is under 2 years old he or she is unlikely to mount an adequate antibody response to type b capsular polysaccharide and conventional antibotic treatment of the infection does not eradicate carriage. About 2-3% will develop a recurrence of their infection[5].

Rifampicin

Rifampicin has been shown to be over 95% effective in eradicating nasopharyngeal carriage of Hib, although only if given as a 20 mg/kg once daily dose (maximum 600 mg) for 4 days[1]. Band et al randomised both day care and hosehold contacts of an index case to receive rifampicin at the above dose or placebo: 4 of 765 placebo but none of 1,112 rifampicin recipients developed Hib infection[1]. All 4 children with secondary disease were under 4 years old: 3 were household and one a day care contact. The difference between placebo and rifampicin groups was significant for children under 4 years old. The only other study on efficacy of rifampicin prophylaxis in prevention of disease was an uncontrolled study which suggested that rifampicin prevented secondary cases in day care contacts aged under 24 months[3].

Almost all recipients of rifampicin develop orange-red urine and tears. In one study[1] 20-25% developed adverse effects (as did 11% of placebo recipients) of which gastrointestinal upset (nausea, vomiting, diarrhoea or abdominal pain) was the commonest, followed by neurological complaints (dizziness, drowsiness, headache). Rifampicin should not, be given to pregnant women.

Another adverse effect of day care rifampicin prophylaxis not easily quantified is the increased level of anxiety engendered among the families of day care contacts.

The rationale of rifampicin prophylaxis is to eradicate nasopharyngeal carriage in the whole family of a risk case. Since conventional antibiotic treatment does not eradicate nasal carriage the index child must also receive rifampicin.

Most secondary cases within families occur within 1-2 weeks of the index case so family prophylaxis should be started immediately and the index child should be treated as soon as he or she can tolerate oral medication. Rifampicin should not, however, be given at the same time as chloramphenicol as it induces liver enzymes and leads to very low chloramphenicol levels.

Recommendations for prophylaxis

Recommendations on prophylaxis following diagnosis of an index case of Hib disease are based on limited data on the need for and efficacy of rifampicin. It is harly surprising, therefore, that the recommendations have been changed frequently but it is a source of much confusion for clinicians.

There are no firm guidelines in Australia and each clinician is expected to make his or her own decision and implement it. The 1988 Red Book (Report of the Committee on Infectious Diseases of the American Academy of Pediatrics) is fairly woolly: "the management of day care and nursery school contact groups should be individualized".

We would suggest the following guidelines:-

1) Index case: Index cases under 2 years old are at high risk of having recurrent disease, and rifampicin shold be given to the index case and all his or her household contacts including adults (unless pregnant).

2) Household contacts: If the index case has a sibling or other household contact under 48 months old, rifampicin prophylaxis should be given to the index case and all household contacts including adults.

3) Day care contacts: Rifampicin prophylaxis is only indicated after a single case of Hib infection in a day care unit or nersery school, if the contacts are under 2 years old and have been in contact with the index case at least 25 hours a week. In this unusual circumstance prophylaxis should also be given to other children and staff at the day care, i.e. they should be treated like a household. If all day care contacts are more than 2 years old prophylaxis is not indicated after a single case.

If two or more cases of systemic Hib infection occur within 60 days in a day care or nursery all children attending the facility and all staff should be given prophylaxis.

References

1. Band JD et al. Prevention of *Haemophilus influenzae* type b disease. *JAMA* 1984;**251**:2381-6.

2. Osterholm MT et al. The risk of subsequent transmission of Haemophilus influenzae type b disease among children in day care. *N Eng J Med*;1987;**316**:1-5.

3. Fleming DW et al. Secondary *Haemophilus influenzae* type b in day-care facilities risk factors and prevention. *JAMA* 1985;**254**:509-14.

4. Li KI, et al. Hib colonization in household contacts of infected and colonized children enrolled in day care. *Pediatrics* 1986;**78**:15-20.

5. Dashefsky B, et al. Management of contacts of children in day care with invasive *Haemophilus influenzae* type b disease. *Pediatrics* 1986;**78**:939-41.

VACCINES AGAINST HAEMOPHILUS INFLUENZAE TYPE B

(Based on CDR 90/49, 7 December 1990)

Infections due to encapsulated *Haemophilus influenzae* type b (Hib) are an important cause of morbidity in young children. Clinical presentations include meningitis, bacteraemia, epiglottitis and other features of invasive disease such as pneumonia, septic arthritis, osteomyelitis and cellulitis. The estimated annual incidence in children under five years of age is 33.4 per 100,000. This represents a cumulative incidence of one invasive infection in 600 children before their fifth birthday.

Hib vaccines containing capsular polysaccharide antigens were first developed in the early 1970s. A large field trial of a polysaccharide vaccine, involving 98,272 children aged between three months and five years, was conducted in Finland from 1974 to 1976[2]. This trial showed that the vaccine was effective in preventing invasive Hib disease in children over 18 months of age, however for children under 18 months no protective effect was observed. As most cases of Hib disease occur in children less than two years of age, these early Hib vaccines were not widely incorporated into immunisation schedules.

The efficacy of Hib vaccines in young children has now been improved by covalent linkage of the polysaccharide antigen to a carrier protein. These conjugate Hib vaccines are protective in children from two months of age[3]. In addition, they appear to generate immunological memory, which should provide long term protection. They are now routinely used in young infants in Finland, the USA and Canada. Side effects are uncommon and are generally mild[4]. Permanent sequelae associated with Hib vaccine have not been reported, although an increased susceptiblity to invasive Hib disease in the week following vaccination has been observed[5].

To date, only one clinical trial of a conjugated Hib vaccine has been conducted in the United Kingdom (UK). One hundred and three children in Oxford received three doses of vaccine at three, five, and ten months of age, at the same time as the standard diphtheria/tetanus/pertussis (DTP) and polio vaccines[6]. All but two infants developed antibody levels greater than 1.0 ug/ml, the level thought to be associated with long term protection. Since this trial was completed, the childhood immunisation schedule has changed; DTP and polio vaccines are now given at two, three anf four months of age[7]. It is clearly desirable that any Hib vaccine schedule should be compatible with the rest of the programme.

For this reason, further UK trials are now in progress to evaluate Hib vaccines when given according to the new two, three, four month schedule. The studies are being conducted in Gloucester and Oxford. Four vaccines are being evaluated (table). Studies to determine immune responses in different populations will also be required as suboptimal protection has been demonstrated in certain ethnic groups, notably Alaskan Eskimos[8]. If the results of the UK trials are satisfactory, a Hib vaccine is likely to be available for routine use within two to three years.

Table 1. Candidate conjugate Haemophilus influenzae type b vaccines

MANUFACTURER	CARRIER PROTEIN
Connaught	Diphtheria toxoid (PRP-D)
Merieux	Tetanus toxoid (PRP-T)
Praxis	Non-toxic mutant diphtheria toxin (HbOC)
Merck Sharpe and Dohme	Group B meningococcal outer membrane protein (PRP-OMP)

References

1. Tudor-Williams G, Frankland J, Isaacs D et al. *Haemophilus influenzae* type b disease in the Oxford region. *Arch Dis Child* 1989;**64**:517-9.

2. Peltola H, Kayhty H, Sivonen A, Makela PH. *Haemophilus influenzae* type b capsular polysaccharide vaccine in children: a double-blind field of study of 100,000 vaccinees 3 months to 5 years of age in Finland. *Pediatrics* 1977;**60**:730-7.

3. Makela PH, Eskola J, Peltola H. Clinical experience with *Haemophils influenzae* type b conjugate vaccines. *Pediatrics* 1990;**85** (suppl):651-3.

4. Vadheim CM, Greenberg DP, Marcy SM, Froeschle J, Ward JI. Safety evaluation of PRP-D *Haemophilus influenzae* type b conjugate vaccine in children immunised at 18 months of age and older: follow-up study of 30,000 children. *Pediatr Infect Dis J* 1990;**9**:555-61.

5. Sood SK, Daum RS. Disease caused by *Haemophilus influenzae* type b in the immediate period after homologous immunisation: immunologic investigation. *Pediatrics* 1990;**85** (Suppl):698-704.

6. Tudor-Williams G, Frankland J, Isaacs D et al. *Haemophilus influenzae* type b conjugate vaccine trial in Oxford: implications for the United Kingdom. *Arch Dis Child* 1989;**64**:520-4.

7. Department of Health, Welsh Office, Scottish Home and Health Department. Immunisation against infectious disease. London: **HMSO** 1990.

8. Ward J, Brenneman G, Letson GW, Heyward WL and The Alaska *H. influenzae* Vaccine Study Group. Limited efficacy of a *Haemophilus influenzae* type b conjugate vaccine in Alaska native infants. *N Engl J Med* 1990;**323**:1393-1401.

PERTUSSIS IN WESTERN AUSTRALIA DURING THE EIGHTIES

(P Masters, P Campbell, B Wild - Princess Margaret Hospital for Children, J Gill - Health Department of Western Australia)

Recent articles published in **CDI** (90/6 & 90/13) have shown that pertussis continues to present a problem in Australia. This infection has caused concern in other developed countries, but their experiences have been different from ours.

The incidence of pertussis in the eighties has been lower in the USA and higher in the UK, Sweden and Japan, than in Australia, for reasons which are well known to readers of **CDI**. One experience which has been widespread is the increased recognition of pertussis in adults. It has been reported from the USA, Sweden and from Sydney, Australia[1].

The experiences of the UK, Sweden and Japan have demonstrated another point: the present whole-cell pertussis vaccine has considerable protective value, so that its side-effects, however unwelcome, should not be allowed to reduce the proportion of babies being immunised.

The estimations of pertussis incidence in the recent articles in **CDI** were based on isolations of *B. pertussis*, hospital discharge data and notifications by clinicians. These must be considerable underestimates of the incidence of infection in school children and adults. Both positive cultures and hospital admissions for pertussis are mainly seen in infancy, while doctors are reluctant to diagnose the infection in older children and adults on clinical findsings alone. Only by obtaining a higher proportion of laboratory diagnoses in out-patients can the pertussis immunisation programme be adequately monitored.

Measuring pertussis-specific IgA antibody in nasopharyngeal aspirates (NPAs) provides a promising way of extending the laboratory diagnosis of pertussis, and was first described by Canadian workers. The method has two advantages: the same sample is used for both culture and antibody tests, and specific IgA antibodies are produced only in response to pertussis infection and not to vaccination.

In 1988 the authors reported early experience with a quantitative modification of the ELISA antibody test on NPA described by Goodman, Wort and Jackson[2,3]. Between 1/10/84 and 31/3/85 the authors obtained NPA's on clinical presentation from 543 children.

On clinical review 395 children were considered to have had pertussis and 148 children not to have had the infection. Of 395 NPA's from the infected children 36% were culture-positive and 24% were antibody positive.

In spite of the 40% false negatives, clinicians responded by providing, for the first time, more samples from out-patients than from in-patients. In-patients comprised only 12% of the 395 infected children tested, or 20% of those yielding positive culture or antibody results. In subsequent years positive yields from the antibody test have been higher because of an increasing proportion of older children and adults tested (see Figure 1.).

During the 1984/85 summer season nearly all outpatients tested attended Princes Margaret Hospital (PMH) or private rooms of paediatricians. In subsequent seasons an increasing proportion attended general practitioners, and their NPAs were collected by private laboratory staff and sent to PMH. During the 1989/90 season 109 out of 267 positive samples were so collected.

The unexpectedly large number of infected children in their second year of life during the 89/90 season (see Figure 1.) prompted an investigation of the immunisation histories of those aged between 6 months and 3 years at the onset of pertussis. This was done retrospectively by telephone or letter and compared with the immunisation status of the same age groups infected in the 84/85 season(see Table 1.).

The main purpose of the figure is to indicate two possible causes for the increased prevalence of pertussis in Australia during the eighties: abandoning the fourth DTP injection at the beginning of 1979 (restored at the beginning of 1985 in WA), and substandard batches of pertussis vaccine.Potency has been assayed by the mouse-brain challenge test, and was found to be diminished in most batches of vaccine over a two year period centred on 19794.

However, lower rates of pertussis vaccination in infants may also have been a contributing factor. Surveys of immunisation coverage in WA show 95% acceptance of DTP/CDT, but it has not been possible to estimate DTP coverage.

Table 1.: Immunisation History of Cases of Pertussis, 1984/85 Season

Season	84/85				89/90			
Age ranges (months)	6 -	12 -	24 - 36	Totals	6 -	12 -	24 - 36	Totals
DPTs 0	2	3	4	9	5	2	3	10
1 or 2	2	1	0	3	8	1	2	11
3 or 4	5	9	8	22	3	10	2	15
	9	13	12	34	16	13	7	36
Not determined	0	1	4	5	6	13	5	24
TOTALS	9	14	16	39	22	26	12	60

The data suggest that the higher number of 6-12 month children infected in the 89/90 season may have been due to a larger proportion failing to complete their DPT courses by six months of age. The even higher number of 12-24 months children infected during the 89/90 season cannot be explained in this way, but contact with the larger number of younger infected babies is a probable explanation.

The period October to March was selected because of the pronounced summer incidence of pertussis in WA; most cases occur during November, December, January and February. Note the peak incidence for the 1979 cohort in the 84/85 and 86/87 seasons.

The same applies to the 85/86 season, while there were insufficient cases of pertussis during the 87/88 and 88/89 seasons to produce meaningful age-related data. Only recently was it considered that the large number of admissions for pertussis in the 81/82 season (See Table 2.) might provide evidence regarding substandard vaccine issued during 1979.

Figure 1.

PERTUSSIS IN WA AGE INCIDENCE IN THREE SUMMER SEASONS BASED ON LABORATORY DIAGNOSIS AT P.M.H.

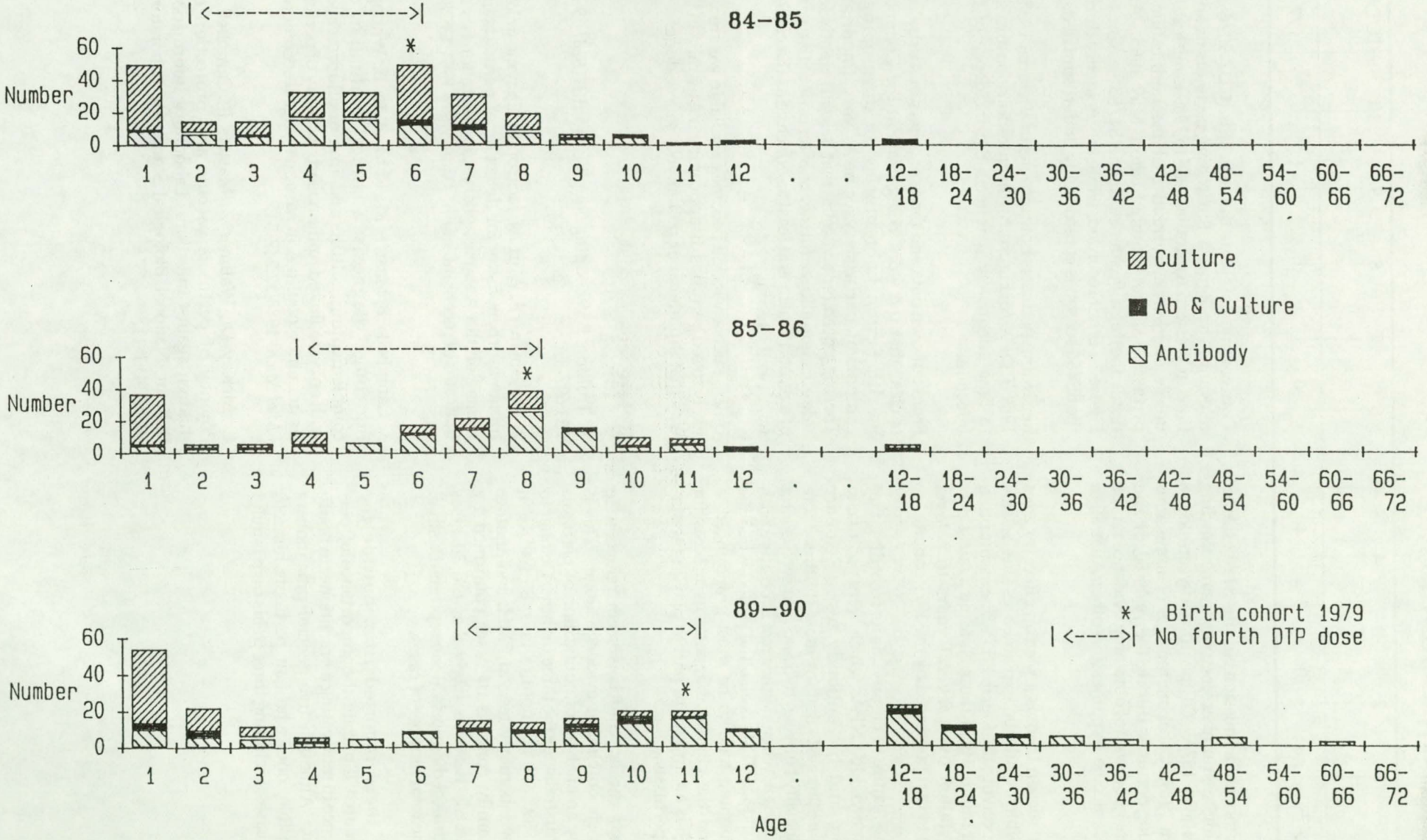


Table 2. Hospitalised pertussis cases 1981/82

YEARS OF AGE	0	1	2	3	4	5	6	7	8	9	10	TOTAL
ADMISSIONS	47	6	10	3	4	2	0	2	2	1		77

The total number of admissions for the 81/82 season was 77, the highest number since we began recording pertussis data in 1974. Of the 10 two-year-olds, 8 were born in 1979. Checking the ages of admissions to hospital for pertussis during the years 80, 81 and 82, could be a good way of obtaining evidence for the substandard vaccine hypothesis in other Australian States.

In the 89/90 season there is a plateau effect for ages six to ten which could be explained as due to this cohort not receiving the fourth DTP. The assumption is made that immunity of these children against pertussis had fallen over several years to a level comparable to that possessed by the 1979 cohort.

A misleading feature of Figure 1. is its recording that a few children yielded NPAs which were positive to both culture and the antibody test. Evidence presented here suggests that for most children there is a period, usually between two and four weeks after onset of the cough, when neither test is positive with present techniques. This is also the period when the bouts of coughing tend to be most pronounced, a reminder that the clinical diagnosis as a basis for notification has an important part to play in the surveillance of pertussis.

As mentioned earlier, clinicians seem reluctant to notify pertussis on clinical evidence alone. This is supported by an analysis of notification of pertussis in Western Australia for the three months, October to December 1989. Of 117 notifications, 83 had been confirmed by laboratory tests at PMH, and another four were family contacts of cases confirmed by laboratory tests. It seems likely that the 30 cases notified on clinical diagnosis alone represent a small fraction of the total number of cases.

The ten of these reported from outside the metropolitan area illustrate the importance of personal experience in motivating a practitioner to notify this infection. Although five general practitioners were responsible, one, who had pertussis himself, notified five cases, including three in his own family!

Two comments about the article in CDI 90/6 are relevant. The Figure 4, illustrating pertussis notifications in Western Australia, 1985 to 1989 is misleading unless one keeps in mind that the notification of pertussis was not required before May 1985. As a result the high incidence in the 84/85 season (see Figure 1.) is not recorded, while the incidence during the 85/86 season is considerably underestimated.

Regarding pertussis notifications in Victoria, 1985 to 1989 (CDI 90/6, Figure. 5), the question also arises as to how adequately it reflects the incidence of the infection.

From our own data and those of others in Australia it is clear that the work being done at the Commonwealth Serum Laboratories on developing a component pertussis vaccine is very important. There is a need for a vaccine that has sufficiently few side effects to allow a further booster dose at five years of age, and which is at least as effective as the current whole-cell vaccine.

Unless such a vaccine becomes available we are unlikely to see pertussis disappear from Australia in the way that diphtheria or poliomyelitis have done.

References

1. Trollfors B Whooping cough in adults. *BMJ* 1981; 283:697-8.

2. Goodman YE, Wort AJ, Jackson FL Enzyme linked immunosorbent assay for detection of pertussis immunoglobulin A in nasopharyngeal secretions as an indicator of recent infection. *J Clin Micro* 1981;13:286-93.

3. Campbell PB, Masters PL, Rohwedder E. Whooping cough diagnosis: a clinical evaluation of complementing culture and immunofluorescence with enzyme linked immunosorbent assay of pertussis immunoglobulin A in nasopharyngeal secretions. *J Med Micro* 1988;27:247-54.

4. McKay DJ, Wallmer Z, Masters P, Campbell P Potency of pertussis vaccine and protection of children against infection. Laboratory Information Bulletin, National Biological Standards Laboratory 1988;1(4):1-11.

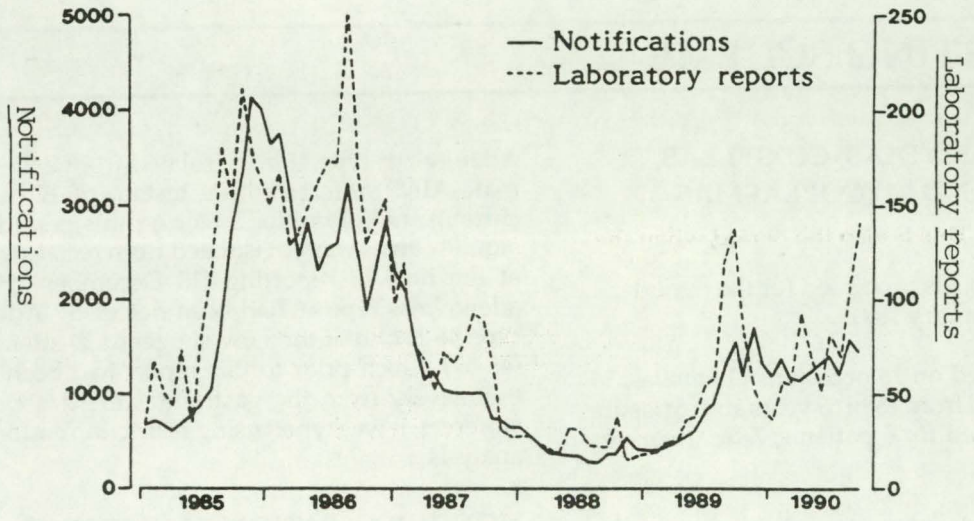
WHOOPING COUGH SURVEILLANCE

(Based on CDR 90/41 October 1990)

The current upsurge in pertussis is continuing. Laboratory reports have doubled in the past eight weeks, from 67 (weeks 30-33) to 140 (weeks 38-41). This increase has occurred in several Regions, especially Mersey and North East Thames. Notifications have not shown a similar increase. Increased laboratory reporting in the absence of a corresponding rise in notifications has been observed previously, in years of both high and low incidence.

Since the beginning of the year, 13,187 cases have been notified in England and Wales. Six deaths have been recorded, a case fatality ratio of 0.05 per 100 notifications, higher than in recent years. Five deaths were in infants aged three months or less.

In an effort to reduce the morbidity and mortality from whooping cough in early infancy, the Department of Health has recently changed the immunisation schedule to start at two months, with an interval of one month between each dose. It is important that infants are vaccinated without unnecessary delay. Pertussis vaccine should also be considered for older unimmunised children, both for their own protection and for that of young siblings under the age of vaccination. There is no upper age limit for pertussis vaccination.



ROUTINE SCREENING OF PREGNANT WOMEN FOR HIV

(Australian National Council on AIDS Bulletin no.8)

Mother-to-child transmission of human immunodeficiency virus (HIV) has become of major importance in sub-Saharan Africa and the Caribbean and in certain cities of the United States and Europe.

Transmission of HIV from an infected woman to her baby is an efficient process, the rate being between 25 and 50%. HIV-infected infants have a poor prognosis and most become symptomatic before one year of age.

Although in Australia the risk of a pregnant woman being infected with HIV is small; because the outcome is so important for the baby, it is likely that most women would want to know if they are infected. HIV infection also has serious implications for the future health of the pregnant woman and her sexual partner(s).

It is often impossible to detect if a pregnant woman is at risk of HIV infection from interview or examination eg. if she has a bisexual partner, or if she or her sexual partner is or has been an intravenous drug user who has shared needles. Therefore, testing for HIV should be offered to all pregnant women, not only those perceived to be risk.

Testing for HIV should be offered alongside other routine antenatal tests and performed with the patient's knowledge and consent. As with other screening tests, it is important that the woman's doctor explain the rationale of the test and be prepared to answer any queries.

In the event of a pregnant woman being found to test positive, the speical medical problems associated with HIV infection during pregnancy and after delivery may require expert counselling and management. If the woman's medical adviser is not skilled in this area the patient should be referred to an obstetrician and counsellor with appropriate expertise.

Enquires regarding the Bulletin to Secretary, Australian National Council on AIDS, GPO Box 9848, Canberra ACT 2601, telephone (06)289 7767.

This bulletin was prepared in conjunction with the Royal Australian College of Obstetricians and Gynaecologists.

CDI REPORTING SCHEME

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTS

There were 1673 reports processed for the period (6 December to 1 January 1991).

Q fever was reported on 16 occasions (2 females, 14 males). Ages ranged from 15 to 69 years and exposure details were provided for 8 patients; 7 meatworkers and 1 animal handler.

Coxsackievirus B4 was isolated from the cerebrospinal fluid of a pregnant (20 weeks), 30-year-old woman who presented with meningitis. Infection with coxsackievirus B during pregnancy has been associated with lower birth weight, urogenital and cardiovascular anomalies in the fetus, and with spontaneous abortion or still-birth with infections late in the pregnancy.

Cytomegalovirus, presumably congenitally acquired, was isolated from the urine of a 7-month-old female with cerebral atrophy.

Rubella was reported in an adult female who was 8 weeks pregnant.

Adenovirus type 46 was isolated from a 55-year-old male AIDS sufferer with a history of 12 months of chronic anal ulceration. During this period Herpes simplex virus was not isolated from rectal swabs, and at the time of reporting (13 December 1990) only adenovirus type 46 had been detected. Adenovirus type 46 is one of the new subgenus D adenoviruses (43-47) which prior to this report had been isolated exclusively from the gastrointestinal tract of AIDS sufferers; it was typed using restriction endonuclease analysis.

NON-VIRAL PATHOGEN REPORTS

16 positive blood culture reports have been received since CDI 90/25 (12 from Toowoomba Base Hospital Pathology Laboratory and 4 from Nambour Hospital Laboratory. The following organisms were isolated:

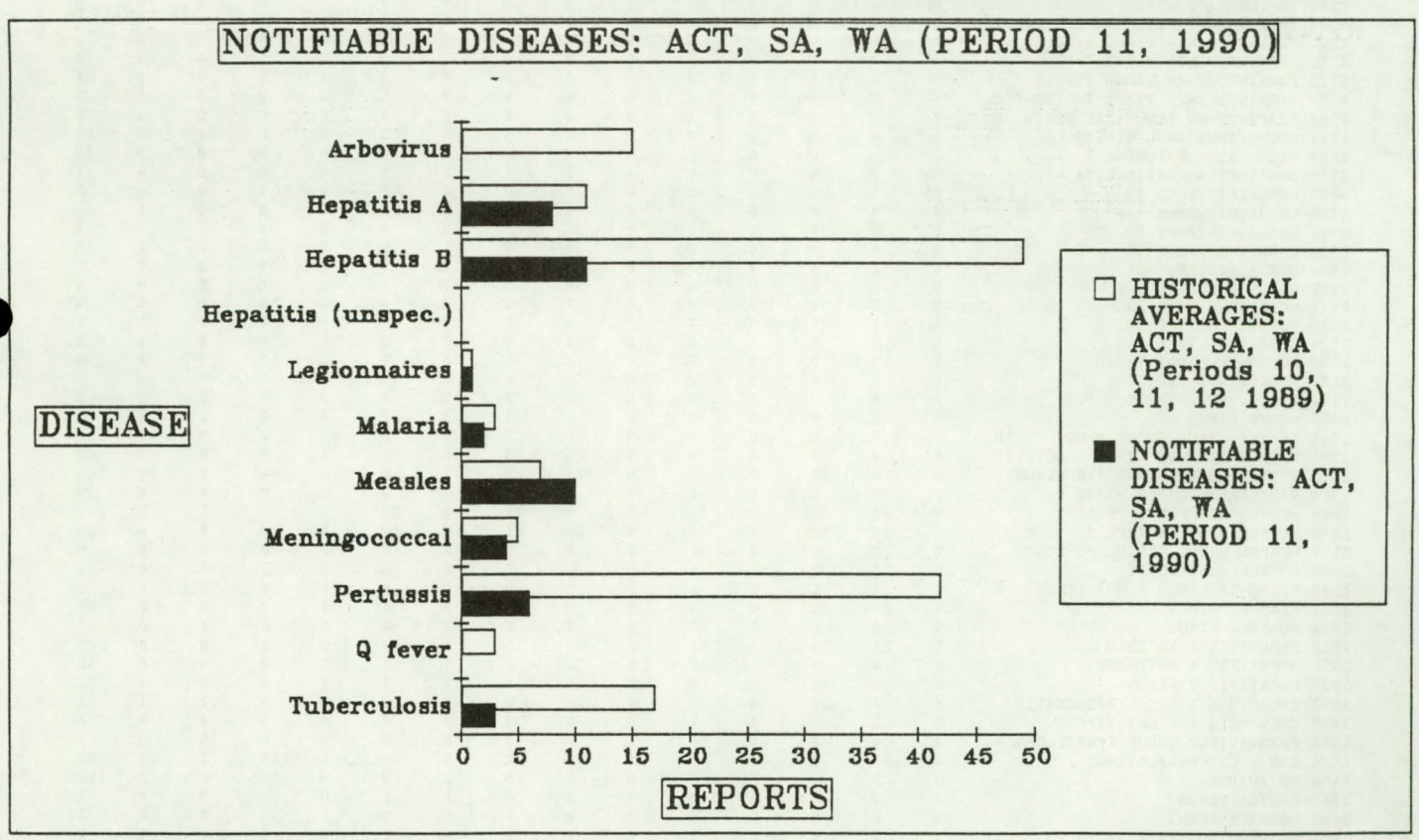
- *Brucella abortus* from a 47 year old male kangaroo shooter also exposed to goats and pigs;
- *Enterobacter aerogenes* from an 81 year old female with obstructive jaundice;
- *Escherichia coli* from 4 patients, a 20 year old female, a 49 year old female with pyelonephritis an 80 year old anaemic diabetic male and an 81 year old female;

- *Haemophilus influenzae* type b from a 12 month old female with purulent otitis media and *Giardia lamblia* in the faeces;
- *Morganella morganii* from a 77 year old male with cancer of the bladder;
- *Salmonella group B* from a 5 year old male, this organism was also isolated from a faecal specimen;
- *Staphylococcus aureus* from 2 male patients, one a 69 year old dialysis patient the other 70 years old;
- *Staphylococcus epidermidis* from an 81 year old female with subacute bacterial endocarditis;
- *Streptococcus durans* from a 75 year old female;
- *Streptococcus mitis* from a newborn female, this organism was also isolated from the gastric aspirate and skin swabs, there was a history of 4 days premature rupture of membranes.
- *Streptococcus pneumoniae* from 2 patients, a 29 year old female (organism also isolated from sputum) and a 68 year old male with leukemia.

Further interesting reports include:

- One case of meningitis reported from Toowoomba Pathology Laboratory in a 2 year old female. *Haemophilus influenzae* type B was isolated from the CSF.
- 2 reports of Cryptococcal antigen positive patients (ICPMR, Westmead) were received, a 36 year old female and a 43 year old male, no risk factors were reported.
- *Shigella sonnei* was isolated from the faeces of a 48 year old female.
- *Bordetella pertussis* was detected by serology in a 24 year old male and a 54 year old female, both staff at a Thoracic/Renal Unit.

(The above 3 reports were received from Toowoomba Base Hospital Pathology Laboratory).



AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 06/12/90 TO 01/01/91

CODE 018 - MICROBIOLOGICAL DIAGNOSTIC UNIT, UNIVERSITY OF MELBOURNE (VIC)
CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 065 - STATE HEALTH LABORATORY SERVICES, PERTH (WA)
CODE 066 - PRINCESS MARGARET HOSPITAL, PERTH (WA)
CODE 110 - INSTITUTE OF MEDICAL & VETERINARY SCIENCE, ADELAIDE (SA)
CODE 111 - ROYAL CHIDLDRENS HOSPITAL, MELBOURNE (VIC)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 113 - PRINCE HENRY/PRINCE OF WALES HOSPITALS, SYDNEY (NSW)
CODE 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)
CODE 400 - DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON (QLD)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

	018	019	065	066	110	111	112	113	114	115	116	400	RHH	TPL	TOTAL
0100 ADENOVIRUS NOT TYPED	0	2	3	8	2	10	0	7	0	6	0	0	0	0	38
0101 ADENOVIRUS TYPE 1	0	2	0	0	0	0	1	1	0	0	0	0	1	0	5
0102 ADENOVIRUS TYPE 2	0	0	0	0	0	0	2	0	0	0	0	0	0	0	2
0103 ADENOVIRUS TYPE 3	0	1	0	0	1	0	3	0	0	0	0	0	0	0	5
0104 ADENOVIRUS TYPE 4	0	2	0	0	0	0	0	0	0	0	0	0	3	0	5
0108 ADENOVIRUS TYPE 8	0	14	0	0	0	0	0	0	0	0	0	0	0	0	14
0109 ADENOVIRUS TYPE 9	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
0111 ADENOVIRUS TYPE 11	0	0	0	0	0	0	4	0	0	0	0	0	0	0	4
0117 ADENOVIRUS TYPE 17	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
0119 ADENOVIRUS TYPE 19	0	2	0	0	0	0	0	0	0	0	0	0	0	0	2
0128 ADENOVIRUS TYPE 28	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
0129 ADENOVIRUS TYPE 29	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
0146 ADENOVIRUS TYPE 46	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1
0199 ADENOVIRUS TYPING PENDING	0	0	0	0	0	4	0	2	2	0	0	0	0	0	8
0201 INFLUENZA A VIRUS	0	0	0	0	1	0	3	0	0	2	0	1	0	0	7
0202 INFLUENZA A VIRUS SUBTYPE H3N2	0	14	0	0	0	0	1	0	1	0	0	0	1	0	17
0203 INFLUENZA B VIRUS	0	4	0	0	3	1	0	0	0	0	0	0	0	0	8
0299 INFLUENZA VIRUS - TYPING PENDI	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
0301 PARAINFLUENZA VIRUS TYPE 1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
0302 PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	3	0	0	0	0	0	0	0	0	0	3
0303 PARAINFLUENZA VIRUS TYPE 3	0	6	0	1	12	3	6	0	5	4	0	0	0	0	37
0399 PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	2	0	0	0	1	0	0	0	0	3
0400 RESPIRATORY SYNCYTIAL VIRUS (R	0	4	0	2	1	0	4	0	0	0	0	0	2	0	13
0500 RHINOVIRUS (ALL TYPES)	0	8	2	0	0	5	3	0	3	3	0	0	0	0	24
0600 MYCOPLASMA PNEUMONIAE	0	3	6	0	1	4	6	3	0	0	0	3	0	0	26
0700 ORNITHOSIS-PSITTACOSIS	0	7	0	0	0	0	2	1	0	0	1	0	0	0	11
0809 COXSACKIEVIRUS A9	0	0	0	0	0	0	2	0	0	0	0	0	0	0	2
0816 COXSACKIEVIRUS A16	0	4	0	0	0	0	0	0	0	0	0	0	0	0	4
0902 COXSACKIEVIRUS B2	0	0	0	0	0	0	1	0	2	0	0	0	0	0	3
0904 COXSACKIEVIRUS B4	0	9	0	0	0	0	0	0	1	0	0	0	0	0	10
0906 COXSACKIEVIRUS B6	0	2	0	0	0	0	0	0	0	0	0	0	0	0	2
1005 ECHOVIRUS TYPE 5	0	0	0	0	4	0	0	0	0	0	0	0	0	0	4
1011 ECHOVIRUS TYPE 11	0	2	0	0	2	0	0	0	0	0	0	0	0	0	4
1022 ECHOVIRUS TYPE 22	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1
1024 ECHOVIRUS TYPE 24	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1
1100 POLIOVIRUS NOT TYPED	0	0	0	0	0	0	0	4	0	0	0	0	0	0	4
1102 POLIOVIRUS TYPE 2	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
1103 POLIOVIRUS TYPE 3	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1
1200 MUMPS VIRUS	0	2	0	0	0	0	4	1	0	0	0	0	0	0	7
1300 HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	2	3	0	0	11	0	0	0	16
1301 HERPES SIMPLEX VIRUS - NOT TYP	0	0	0	5	0	0	26	0	6	2	0	3	0	0	42
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	0	5	20	0	36	8	19	3	1	8	0	44	0	0	144
1303 VARICELLA-ZOSTER VIRUS	0	10	4	1	1	0	3	7	1	0	0	0	0	0	27
1306 HERPES SIMPLEX TYPE 1	0	102	32	1	56	3	7	19	0	31	0	0	1	0	252
1307 HERPES SIMPLEX TYPE 2	1	82	57	0	45	0	26	21	0	22	0	0	0	0	254
1399 HERPES VIRUS TYPING PENDING	0	0	0	0	0	4	0	0	0	0	0	0	0	0	4
1401 COXIELLA BURNETII	0	3	1	0	0	0	4	0	0	5	0	3	0	0	16
1502 PICORNIA VIRUS - NOT TYPED = E	0	0	1	0	0	0	2	9	0	8	0	0	0	0	20
1521 MEASLES VIRUS	0	20	1	0	0	0	0	3	0	0	0	0	0	0	24
1522 RUBELLA VIRUS	0	9	1	0	1	2	3	0	0	0	0	3	0	0	19
1532 HEPATITIS B ANTIGEN	0	25	12	0	20	2	33	15	1	23	5	1	2	0	139
1535 HEPATITIS A ANTIBODY	0	4	9	0	7	0	6	0	1	1	2	0	0	0	30
1536 HEPATITIS C VIRUS	0	0	12	0	0	0	0	0	0	0	0	0	0	0	12
1541 CHLAMYDIA A - C. TRACHOMATIS	0	0	27	0	13	0	28	6	0	15	0	0	1	1	91
1543 CHLAMYDIA A - LGV TYPE	0	1	0	0	0	0	0	0	0	0	0	5	0	0	6
1555 PAPOVAVIRUS GROUP (PAPILLOMA -	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
1556 CMV - CYTOMEGALOVIRUS	0	52	2	7	8	1	12	12	5	14	1	10	1	0	125
1564 ROTAVIRUS	0	1	0	3	20	6	9	16	2	0	0	49	5	1	112
1565 CALICI VIRUS	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
1566 NORWALK AGENT	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
1571 ENTEROVIRUS TYPE 71 (BCR)	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
1599 ENTEROVIRUS TYPING PENDING	0	0	0	0	1	10	0	16	4	0	0	0	0	0	31
4556	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
9906 BARMAH FOREST VIRUS	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
9981 DENGUE TYPE 1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
9983 DENGUE TYPE 3	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
9992 ROSS RIVER VIRUS	0	2	1	0	0	0	0	1	0	7	0	1	0	0	12

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 06/12/90 TO 01/01/91

NSW: ICPMR; PHH/POW; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP.
VIC: FAIRFIELD; RCH; MDU, UNI MELB.
QLD: STATE LAB, BRIS; TOOWOOMBA PATH LAB; ROYAL BRIS HOSP; DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON.
WA: STATE LAB, PERTH; PMH.
SA: IMVS.
TAS: ROYAL HOBART HOSP; DIAGNOSTIC SERVICES, LAUNCESTON; LAUNCESTON GEN HOSP; DIAGNOSTIC SERVICES, HOBART; HOBART PATH; MERSEY GEN HOSP, LATROBE.
ACT: WVH.

		NSW	VIC	QLD	WA	SA	TAS	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	7	12	6	11	2	0	0	38
0101	ADENOVIRUS TYPE 1	2	2	0	0	0	1	0	5
0102	ADENOVIRUS TYPE 2	2	0	0	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	3	1	0	0	1	0	0	5
0104	ADENOVIRUS TYPE 4	0	2	0	0	0	3	0	5
0108	ADENOVIRUS TYPE 8	0	14	0	0	0	0	0	14
0109	ADENOVIRUS TYPE 9	0	1	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	4	0	0	0	0	0	0	4
0117	ADENOVIRUS TYPE 17	1	0	0	0	0	0	0	1
0119	ADENOVIRUS TYPE 19	0	2	0	0	0	0	0	2
0128	ADENOVIRUS TYPE 28	0	1	0	0	0	0	0	1
0129	ADENOVIRUS TYPE 29	1	0	0	0	0	0	0	1
0146	ADENOVIRUS TYPE 46	0	0	0	0	1	0	0	1
0199	ADENOVIRUS TYPING PENDING	4	4	0	0	0	0	0	8
0201	INFLUENZA A VIRUS	3	0	3	0	1	0	0	7
0202	INFLUENZA A VIRUS SUBTYPE H3N2	2	14	0	0	0	1	0	17
0203	INFLUENZA B VIRUS	0	5	0	0	3	0	0	8
0299	INFLUENZA VIRUS - TYPING PENDI	0	1	0	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	1	0	0	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	3	0	0	3
0303	PARAINFLUENZA VIRUS TYPE 3	11	9	4	1	12	0	0	37
0399	PARAINFLUENZA VIRUS TYPING PEN	0	2	1	0	0	0	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	4	4	0	2	1	2	0	13
0500	RHINOVIRUS (ALL TYPES)	6	13	3	2	0	0	0	24
0600	MYCOPLASMA PNEUMONIAE	9	7	3	6	1	0	0	26
0700	ORNITHOSIS-PSITTACOSIS	3	7	0	0	0	0	1	11
0809	COXSACKIEVIRUS A9	2	0	0	0	0	0	0	2
0816	COXSACKIEVIRUS A16	0	4	0	0	0	0	0	4
0902	COXSACKIEVIRUS B2	3	0	0	0	0	0	0	3
0904	COXSACKIEVIRUS B4	1	9	0	0	0	0	0	10
0906	COXSACKIEVIRUS B6	0	2	0	0	0	0	0	2
1005	ECHOVIRUS TYPE 5	0	0	0	0	4	0	0	4
1011	ECHOVIRUS TYPE 11	0	2	0	0	2	0	0	4
1022	ECHOVIRUS TYPE 22	1	0	0	0	0	0	0	1
1024	ECHOVIRUS TYPE 24	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	4	0	0	0	0	0	0	4
1102	POLIOVIRUS TYPE 2	1	0	0	0	0	0	0	1
1103	POLIOVIRUS TYPE 3	1	0	0	0	0	0	0	1
1200	MUMPS VIRUS	5	2	0	0	0	0	0	7
1300	HERPES VIRUS GROUP - NOT TYPED	5	0	0	0	0	0	11	16
1301	HERPES SIMPLEX VIRUS - NOT TYP	32	0	5	5	0	0	0	42
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	23	13	52	20	36	0	0	144
1303	VARICELLA-ZOSTER VIRUS	11	10	0	5	1	0	0	27
1306	HERPES SIMPLEX TYPE 1	26	105	31	33	56	1	0	252
1307	HERPES SIMPLEX TYPE 2	47	83	22	57	45	0	0	254
1399	HERPES VIRUS TYPING PENDING	0	4	0	0	0	0	0	4
1401	COXIELLA BURNETII	4	3	8	1	0	0	0	16
1502	PICORNIA VIRUS - NOT TYPED = E.	11	0	8	1	0	0	0	20
1521	MEASLES VIRUS	3	20	0	1	0	0	0	24
1522	RUBELLA VIRUS	3	11	3	1	1	0	0	19
1532	HEPATITIS B ANTIGEN	49	27	24	12	20	2	5	139
1535	HEPATITIS A ANTIBODY	7	4	1	9	7	0	2	30
1536	HEPATITIS C VIRUS	0	0	0	12	0	0	0	12
1541	CHLAMYDIA A - C. TRACHOMATIS	34	0	16	27	13	1	0	91
1543	CHLAMYDIA A - LGV TYPE	0	1	5	0	0	0	0	6
1555	PAPOVAVIRUS GROUP (PAPILLOMA -	1	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	29	53	24	9	8	1	1	125
1564	ROTAVIRUS	27	7	50	3	20	5	0	112
1565	CALICI VIRUS	1	0	0	0	0	0	0	1
1566	NORWALK AGENT	1	0	0	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	0	1	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	20	10	0	0	1	0	0	31
4556		0	0	1	0	0	0	0	1
9906	BARMAH FOREST VIRUS	0	0	1	0	0	0	0	1
9981	DENGUE TYPE 1	0	0	1	0	0	0	0	1
9983	DENGUE TYPE 3	0	0	1	0	0	0	0	1
9992	ROSS RIVER VIRUS	1	2	8	1	0	0	0	12
9993	ASTROVIRUS	3	0	0	0	0	0	0	3
9994	SMALL VIRUS (LIKE) PARTICLE	3	0	0	0	0	0	0	3
TOTAL		421	474	282	219	240	17	20	1673

NOTE: DIRECT COMPARISION BETWEEN STATES IS NOT POSSIBLE SINCE:

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 06/12/90 TO 01/01/91

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	6	0	0	0	19	0	0	0	2	27
0101	ADENOVIRUS TYPE 1	2	2	0	0	0	1	0	0	0	0	5
0102	ADENOVIRUS TYPE 2	0	1	0	0	0	1	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	1	0	0	0	0	3
0104	ADENOVIRUS TYPE 4	0	4	0	0	0	0	0	0	0	0	4
0109	ADENOVIRUS TYPE 9	0	0	0	0	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	3	0	0	1	0	4
0117	ADENOVIRUS TYPE 17	0	0	0	0	0	1	0	0	0	0	1
0128	ADENOVIRUS TYPE 28	0	0	0	0	0	1	0	0	0	0	1
0129	ADENOVIRUS TYPE 29	0	0	0	0	0	1	0	0	0	0	1
0146	ADENOVIRUS TYPE 46	0	0	0	0	0	0	0	0	0	1	1
0199	ADENOVIRUS TYPING PENDING	0	4	1	0	0	1	0	0	0	0	6
0201	INFLUENZA A VIRUS	1	4	0	0	0	0	0	0	0	0	5
0202	INFLUENZA A VIRUS SUBTYPE H3N2	1	10	0	0	0	0	0	0	0	0	11
0203	INFLUENZA B VIRUS	0	5	0	0	0	0	0	0	0	0	5
0299	INFLUENZA VIRUS - TYPING PENDI	0	1	0	0	0	0	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	1	0	0	0	0	0	0	0	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	3	0	0	0	0	0	0	0	0	3
0303	PARAINFLUENZA VIRUS TYPE 3	2	34	0	0	0	0	0	0	0	0	36
0399	PARAINFLUENZA VIRUS TYPING PEN	0	3	0	0	0	0	0	0	0	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	5	7	0	0	0	0	0	0	0	0	12
0500	RHINOVIRUS (ALL TYPES)	1	19	0	0	0	0	0	0	0	0	20
0600	MYCOPLASMA PNEUMONIAE	3	19	0	0	0	0	0	0	0	0	22
0700	ORNITHOSIS-PSITTACOSIS	1	9	0	0	0	0	0	0	0	0	10
0809	COXSACKIEVIRUS A9	0	1	0	0	0	1	0	0	0	0	2
0816	COXSACKIEVIRUS A16	0	2	0	0	0	0	0	0	0	2	4
0902	COXSACKIEVIRUS B2	1	1	0	0	0	0	0	0	0	0	2
0904	COXSACKIEVIRUS B4	1	2	0	6	0	0	0	0	0	0	9
0906	COXSACKIEVIRUS B6	0	0	0	1	0	0	0	0	0	0	1
1005	ECHOVIRUS TYPE 5	0	3	0	0	0	1	0	0	0	0	4
1011	ECHOVIRUS TYPE 11	0	2	0	1	0	1	0	0	0	0	4
1022	ECHOVIRUS TYPE 22	0	1	0	0	0	0	0	0	0	0	1
1024	ECHOVIRUS TYPE 24	0	1	0	0	0	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	4	0	0	0	0	4
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	1	0	0	0	0	1
1103	POLIOVIRUS TYPE 3	1	0	0	0	0	0	0	0	0	0	1
1200	MUMPS VIRUS	4	0	0	1	1	0	0	0	0	0	6
1300	HERPES VIRUS GROUP - NOT TYPED	4	0	0	0	0	0	0	0	0	5	9
1301	HERPES SIMPLEX VIRUS - NOT TYP	6	3	0	1	0	0	0	0	0	20	30
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	58	12	1	0	0	0	5	0	0	0	76
1303	VARICELLA-ZOSTER VIRUS	7	0	1	0	1	0	0	0	0	15	24
1306	HERPES SIMPLEX TYPE 1	1	10	0	0	0	0	1	2	1	169	184
1307	HERPES SIMPLEX TYPE 2	9	1	0	0	0	1	0	0	0	92	103
1399	HERPES VIRUS TYPING PENDING	0	0	0	1	0	0	0	0	0	3	4
1401	COXIELLA BURNETII	3	0	0	1	0	0	1	0	0	1	6
1502	PICORNIA VIRUS - NOT TYPED = E	0	6	0	0	1	10	0	0	0	0	17
1521	MEASLES VIRUS	7	0	0	0	0	0	0	0	0	16	23
1522	RUBELLA VIRUS	3	0	0	0	0	0	0	0	0	2	5
1532	HEPATITIS B ANTIGEN	75	0	0	0	0	0	60	0	0	0	135
1535	HEPATITIS A ANTIBODY	10	0	0	0	0	0	14	0	0	0	24
1536	HEPATITIS C VIRUS	10	0	0	0	0	0	2	0	0	0	12
1541	CHLAMYDIA A - C. TRACHOMATIS	19	0	0	0	0	1	0	0	0	0	20
1543	CHLAMYDIA A - LGV TYPE	3	0	0	0	0	0	0	0	0	0	3
1555	PAPOVAVIRUS GROUP (PAPILLOMA -	0	0	0	0	0	0	0	0	1	0	1
1556	CMV - CYTOMEGALOVIRUS	13	20	0	0	0	1	4	1	8	1	48
1564	ROTAVIRUS	9	1	0	0	0	100	0	0	0	0	110
1565	CALICI VIRUS	0	0	0	0	0	1	0	0	0	0	1
1566	NORWALK AGENT	0	1	0	0	0	0	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	1	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	1	5	0	1	0	18	0	0	0	1	26
4556		1	0	0	0	0	0	0	0	0	0	1
9906	BARMAH FOREST VIRUS	1	0	0	0	0	0	0	0	0	0	1
9981	DENGUE TYPE 1	1	0	0	0	0	0	0	0	0	0	1
9983	DENGUE TYPE 3	1	0	0	0	0	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	4	1	0	0	0	0	0	0	0	0	5
9993	ASTROVIRUS	0	0	0	0	0	3	0	0	0	0	3
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	3	0	0	0	0	3
TOTAL		272	204	3	14	3	176	87	3	11	330	1103

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 06/12/90 TO 01/01/91

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	4	0	1	0	0	0	1	3	2	0	11
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	0	1	0	0	2
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	14	0	0	0	0	0	0	0	0	0	14
0119	ADENOVIRUS TYPE 19	2	0	0	0	0	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	0	2	0	0	2
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	0	0	2	0	2
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	0	0	0	0	6	0	0	6
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	0	2	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	1	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	0	1	0	1
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	2	1	4
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	1	2	1	0	4
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	0	1	0	0	1
0902	COXSACKIEVIRUS B2	0	0	0	0	1	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	0	0	0	0	0	0	1	0	0	1
0906	COXSACKIEVIRUS B6	0	0	0	0	0	0	0	1	0	0	1
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	7	0	0	0	0	0	0	0	0	7
1301	HERPES SIMPLEX VIRUS - NOT TYP	2	10	0	0	0	0	0	0	0	0	12
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	38	4	1	0	3	10	12	0	68
1303	VARICELLA-ZOSTER VIRUS	1	0	0	0	0	0	0	1	1	0	3
1306	HERPES SIMPLEX TYPE 1	13	51	0	0	0	0	0	0	4	0	68
1307	HERPES SIMPLEX TYPE 2	1	148	0	0	0	0	0	1	1	0	151
1401	COXIELLA BURNETII	0	0	0	0	2	0	0	8	0	0	10
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	1	0	2	0	3
1521	MEASLES VIRUS	0	0	0	0	0	0	0	1	0	0	1
1522	RUBELLA VIRUS	1	0	1	0	2	0	1	0	9	0	14
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	4	0	4
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	1	0	5	0	6
1541	CHLAMYDIA A - C. TRACHOMATIS	0	71	0	0	0	0	0	0	0	0	71
1543	CHLAMYDIA A - LGV TYPE	0	3	0	0	0	0	0	0	0	0	3
1556	CMV - CYTOMEGALOVIRUS	0	1	0	2	0	9	11	13	41	0	77
1564	ROTAVIRUS	0	0	0	0	0	0	0	2	0	0	2
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	1	1	2	1	0	5
9992	ROSS RIVER VIRUS	0	0	0	0	5	0	0	1	1	0	7
TOTAL		40	291	41	6	11	10	20	60	89	1	569