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VACCINATION AGAINST JAPANESE ENCEPHALITIS IN THE TORRES STRAIT

Jeffrey Hanna[1], *Dianne Barnett*[2] *and Dan Ewald*[3]

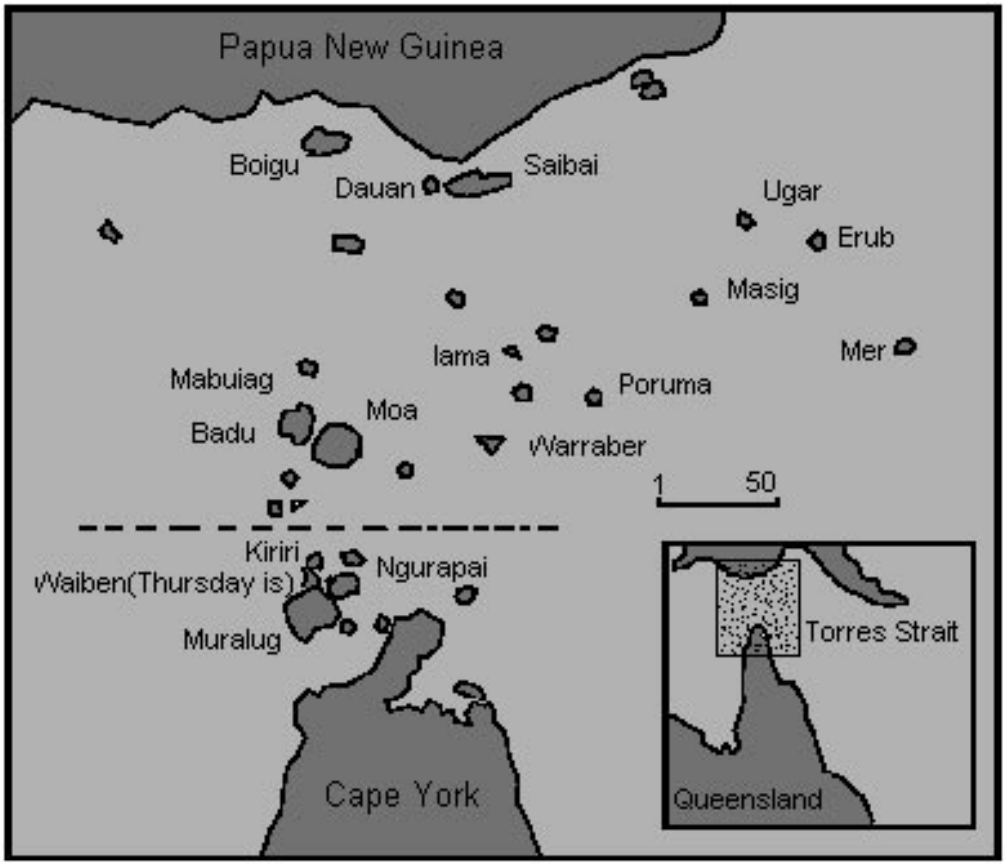
Abstract

Three cases of Japanese encephalitis (JE) occurred among residents of Badu Island in the Torres Strait in early 1995. Although this was the first recognised outbreak of locally acquired JE in Australia, there were subsequent indications that JE was widespread at that time throughout the outer Torres Strait islands. Strategies to minimise the risk of future cases of JE in the Torres Strait were implemented. As a munber of these strategies would take time to implement, vaccination of the population of the outer islands of the Torres Strait before the 1996 wet season was recommended. Vaccine was ordered, and mobile teams, each vaccinating seven communities in the area, administered the vaccine. Several clinics were established to vaccinate people who had temporarily moved away and those moving to the outer islands. JE vaccine was administered to 3,440 people. The vaccination achieved high levels of uptake, with 93% of those who commenced the series receiving two or three doses. Ongoing surveillance using sentinel pigs on the islands is in place to detect further incursions of the JE virus into the Torres Strait.

Background

Three cases of Japanese encephalitis (JE) occurred among residents of Badu Island in the Torres Strait in March-April 1995[1]. Subsequent serological surveys indicated that JE virus activity was widespread throughout the outer Torres Strait Islands at, or about, that time (unpublished data).

Figure. The Torres Strait. The outer islands are north of the arbitrary dotted line



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Although JE is widespread in Asia, the Torres Strait outbreak was the first recognised occasion of locally acquired JE in Australia. In response, a workshop was convened in mid-July 1995 to develop strategies to minimise the risk of future cases of JE in the Torres Strait. Among the short-term (relevant to the 1995-96 wet season) strategies that were considered were the vaccination of people and vaccination of pigs.

At the time, it was considered that concerns about the delayed moderately severe hypersensitivity reactions reported following the administration of the human vaccine[2], and the lack of any information concerning the risk of future outbreaks, did not justify the widespread use of the vaccine. It was proposed instead that pigs be vaccinated, thereby eliminating amplifying hosts from the community environments. Vaccinating pigs would render them unsuitable for surveillance. The Torres Health Council approved the testing of unused human sera, from blood specimens sent for routine laboratory studies, for opportunistic surveillance for JE.

By mid-October it was clear that the animal vaccine would not be available before the 1995-96 wet season and that the proposed risk-assessment studies to determine the likelihood of further incursions of the JE virus into the Torres Strait would take considerably longer than anticipated. An urgent recommendation for a mass vaccination program to cover the population of the outer islands of the Torres Strait before the wet season was forwarded to the then Commonwealth Department of Human Services and Health. On 1 November 1995, it was announced that the Federal Government would fund the purchase of JE vaccine for the people of the outer Torres Strait islands, and Queensland Health would cover the administration costs.

Implementation of the vaccination program

There are 14 communities on 13 outer Torres Strait islands (Figure).

Although there are no reliable census data for the Torres Strait, it was estimated that the population of the outer islands was about 3,000. Local sources suggested that the vaccine uptake would be poor beyond Badu Island, and therefore vaccine was ordered from the vaccine distributor for 2,000 adults and 400 children (one to three years of age).

During November a considerable effort was made to inform the residents of the outer islands on how to minimise the risk of acquiring JE through personal protection measures (repellents, mosquito coils, bed nets etc.) and about the vaccination program. Public Health staff visited each outer island community to brief councils and address community meetings to pass on the information. Two posters were developed and displayed throughout the communities.

Information pamphlets about JE and vaccination consent forms were prepared in English and in the local vernacular. Both specifically mentioned that people who have significant allergies to bee stings, foods and medicines may be more susceptible to allergic reactions to the JE vaccine, and that those who receive the JE vaccine should:

(i) not leave the clinic for 30 minutes after receiving the vaccine,

(ii) not drink alcohol for two days after administration of the vaccine, and

(iii) remain within ready access to medical care for ten days following vaccination.

The advice concerning alcohol was based on tentative findings that suggest that a greater than usual intake of alcohol after vaccination may be associated with adverse reactions (R Kass, personal communication). Community Health staff circulated these sheets in advance of the vaccination teams to obtain consent. The local media - radio and newspaper - also ran stories on the prevention of JE and on the vaccination program.

Every outer island community has a health centre staffed by local health workers; five of the communities have resident registered nurses. Medical services are provided by medical practitioners from Thursday Island visiting on a two- to four-weekly basis. Two mobile teams, each vaccinating seven communities, administered the JE vaccine. Each team consisted of a medical practitioner, at least two registered nurses and the local health workers. Extra nursing staff from outside the Torres Strait had to be assigned to the program to ensure that there were adequate staff not only to constitute the teams but also to maintain routine services. A registered nurse remained behind on each community for three days after vaccination to monitor for adverse reactions.

Prior to the start of the vaccination program, all nursing staff and senior health workers involved in the program participated in a workshop. Details of the JE vaccine, the reported adverse reactions and their management, and details of the program, including itinerary and staff movements, were discussed. Three workshops were convened, two in the Torres Strait and one in Cairns. All attendees were given protocols on JE vaccination and the management of adverse reactions. To cater for Torres Strait residents temporarily away and people moving to the outer islands (for example to commence employment), three JE vaccination clinics were established off the outer islands: in a travel medicine clinic in Brisbane, in a general practice in Cairns and in the Outpatients Department of Thursday Island Hospital.

Vaccine was recommended for all residents over one year of age of the outer Torres Strait Islands unless they had proven immunity by testing positive for JE during the serological survey, and all non-residents who would be living or working on the outer Torres Strait islands in the 1995-96 wet season for the equivalent of 30 days or more[2]. People who had past history of an allergic reaction that required systemic therapy were advised not to have the vaccine.

The vaccine schedule (three doses given on days 0, 7 and 28) required each team to vaccinate one community every day for a week, and then, without any delay, return to the first community to start the second round. The vaccine arrived in time to commence the program as planned on Sunday, 3 December. Additional refrigerator space had to be found on Thursday Island as the volume of vaccine was too great for the hospital's pharmacy. The third round commenced on 8 January (day 35) to allow communities to celebrate New Year (day 28) without undue interference.

Each vaccination clinic required at least two vaccinators working out of separate examination rooms; details of each vaccination were usually recorded by a local health worker or community member. Self-inking stamps were used to record the vaccine batch and date of administration in each client's clinic record. The client's name, sex, date of birth, community of residence, date of vaccination and vaccine batch were recorded and faxed to the Tropical Public Health Unit in Cairns. An administrative officer dedicated to the task entered the information on to an Epi Info database. Printouts for each community were faxed back for correction and used for entering information about subsequent doses in due course.

The logistics of transport (fixed-wing aircraft and occasionally helicopter) between islands, staff movements, accommodation, cold chain requirements and vaccine supply from Thursday Island to, and between, the outer islands was coordinated by a senior registered nurse dedicated to the task.

Several circumstances threatened the program:

(i) One of the chartered aircraft became inoperable without any warning during the first (sequential) two rounds. This required urgent rescheduling of flights and staff movements.

(ii) A shipment of JE vaccine was rejected soon after arrival in Australia, which could have resulted in insufficient vaccine to compete the second round. However the Australian distributor was able to obtain an urgent replacement shipment, employ staff to work overnight to relabel the vaccine to meet Australian requirements, and to courier the shipment to the Torres Strait before existing stocks were exhausted.

(iii) There was an unexpected demand for vaccine by the local residents, far exceeding predictions. The outcome was that Federally funded vaccine was virtually exhausted after the second round, leaving no vaccine to begin the third round. This shortfall was predicted soon after the end of the first round and on 12 December a request was made to the Federal Government for further funding to enable an urgent order to be placed for an additional 2,000 doses of vaccine. The funding was approved and the extra vaccine was obtained in time to commence the third round.

By 19 January, 9,046 doses of JE vaccine had been administered to 3,440 people. Remaining stock was used opportunistically during routine medical clinics, particularly to catch up on second and third doses in those who had commenced but not completed the vaccination regimen. By the end of March 1996, 2,529 people (2,455 from the outer islands) had received three doses, 732 (687 from the outer islands) had received two doses and 250 (222 from the outer islands) had received only one dose. A total of 9,301 doses of vaccine had been administered, with 8,961 being given to residents of the outer islands.

There were ten reports of mild allergic (urticarial) reactions in the 48 hours following receipt of the vaccine; these individuals were advised not to have further doses of the vaccine. There were *no* episodes of acute or delayed moderate or severe reactions of any sort; in particular there were no episodes of delayed moderately severe hypersensitivity reactions.

Summary

Regardless of the formidable logistics, and the local predictions of poor compliance, the vaccination program achieved very high levels of vaccine uptake, with 93% of those who commenced the series receiving two or three doses. No severe adverse reactions occurred. Ongoing surveillance using sentinel pigs is in place to detect any further incursions of the JE virus into the Torres Strait, and may help to determine the need for future vaccinations (for example in young children) and booster doses[3].

Acknowledgments

We wish to thank Dr Diana Lange (Queensland Health) and Dr Tony Adams (Commonwealth Department of Health and Family Services) for negotiating the funding for the vaccine. We also wish to thank CSL Vaccines, Travellers Medical and Vaccination Centre (Brisbane), Abbott Medical Clinic (Cairns), Mr Don Boldiston (Cairns Base Hospital), Mr Scott McCahon (Thursday Island Hospital), and all the Queensland Health staff who assisted with the administration of the vaccine. Particular thanks to the Health Centre managers of the outer islands, and to Dr Ted Tsai (Centers for Disease Control and Prevention, USA) for his continued support.

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REAPPEARANCE OF THE JAPANESE ENCEPHALITIS VIRUS IN THE TORRES STRAIT, 1996

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Following an outbreak of Japanese encephalitis (JE) in the Torres Strait in March - April 1995[1], the inactivated JE vaccine was offered to all residents of the outer islands of the Torres Strait in December 1995 - January 1996. Nearly 9,000 doses of vaccine were administered; 88% of the residents who commenced the vaccination regimen received at least two doses. The high uptake of the vaccine precluded the use of residual sera (from samples forwarded to the Thursday Island Hospital laboratory for clinical purposes) for surveillance to detect further incursions of the JE virus into the Torres Strait.

Queensland Department of Primary Industries (QDPI) and Queensland Health jointly established a system using pigs on three outer islands (Badu, Saibai and Erub), on one inner island (Kiriri) and on the North Peninsula Area (NPA) at the tip of Cape York, as sentinel animals for surveillance. Young pigs (those born after the 1995 outbreak) were chosen assuming them to be susceptible at the onset of the 1995-96 wet season. The three outer islands were chosen because all three have substantial numbers of domestic pigs, have local Australian Quarantine Inspection Service staff to assist with the collection of blood samples from the pigs, and they represent north-south and east-west axes across the outer Torres Strait. Further, Badu was the focus of the 1995 outbreak, and Saibai is within a few kilometers of the Papua New Guinea (PNG) coastline.

The outer island pigs were first bled in late January and early February 1996. A total of 78 pigs were sampled, 40 from Badu, 20 from Saibai and 18 from Erub. As they were sampled they were tagged with a numbered ear tag to allow identification during future samplings. The samples were initially tested by haemagglutination inhibition (HAI) assay, and then plaque reduction neutralisation assay (PRNA) was performed upon any sera with elevated HAI titres. Seven of the pigs, four from one island and three from another, tested positive for JE. The three pigs that tested positive on one island were young (about two months) and belonged to the same litter. All other pigs from the island that were tested were negative. This suggests the pigs that tested positive had maternally acquired antibodies; the mother of the litter was subsequently found to have a very high JE antibody titre, supporting the hypothesis. All four pigs on the other island were subsequently recognised as being older than originally thought and they were alive during the 1995 outbreak.

The first sampling at the end of January-early February provided no firm evidence of JE virus transmission since the 1995 outbreak. Importantly, it identified 71 pigs as being susceptible in early 1996; these pigs became the focus for further surveillance.

The second sampling took place in mid-March, and 66 pigs from the three outer islands were bled. On 29 March the Laboratory of Microbiology and Pathology, Brisbane, confirmed that 12 of the 13 pigs from Saibai with two sequential samples had seroconverted, that is there was at least a four-fold rise from a baseline HAI titre from <20 when the paired sera where tested in parallel. This finding was confirmed by PRNA. The pigs tested on Badu and Erub remained seronegative as did the sentinel pigs on Kiriri and the NPA. Thus there is clear evidence of JE virus activity on Saibai sometime during February - March 1996.

The immediate response was to inform the Saibai Council of the reappearance of JE virus in their community, and to reinforce the importance of personal protection measures (repellants, mosquito coils, bed nets etc.) to minimise the risk of mosquito bites. This response was extended to the two other northern islands - Boigu and Dauan. Although these two islands were not included in the surveillance, they are also very close to PNG and it can be assumed that the circumstances behind the reappearance of JE virus on Saibai are likely to apply to them as well.

The remaining communities of the outer Torres Strait were informed on 1 April, and advised to take similar precautions. Mosquito control activities have been in place in the Torres Strait throughout the wet season, and will be reinforced at Saibai and Boigu over the coming weeks. The sentinel pig surveillance system will be maintained over the coming months, and will possibly be expanded during the next wet season. Although vaccination has protected the majority of the outer island population, plans for ongoing elective vaccination (for example, of children who turn one year of age and therefore become eligible for vaccination) will need to be developed.

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PERTUSSIS AND THE ACELLULAR VACCINES

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Abstract

Morbidity and mortality from pertussis continue to be a problem worldwide, both because the organism is difficult to eradicate and because there is non-compliance with vaccination, often owing to concerns about side effects of the whole-cell vaccine. The new acellular vaccines have fewer side effects and appear to be efficacious, although costly. Some have been approved in other countries for use in infants and children, but none is yet available in Australia. Once they are introduced, careful surveillance will be needed to monitor any epidemiological changes.

Pertussis and pertussis vaccines

It is 90 years since Bordetella pertussis was first isolated by Jules Bordet in Brussels in 1906. Simple vaccines containing a suspension of the whole organism were made soon afterwards, yet only last year the annual incidence of pertussis was estimated at 40 million cases, with around 360,000 deaths (B. Ivanoff and SE Robertson 1995, personnal communication). Most deaths occur in children, particularly infants. The burden falls most heavily on children in developing countries, in which case-fatality rates of up to 15% have been quoted.

Why is it taking us so long to control this disease? The first commercial vaccines were developed 60 years ago in the 1930s, and effective vaccines were widely used in the 1940s and 1950s. Although these whole-cell vaccines reduced the incidence of whooping cough and the associated morbidity and mortality, they did not provide absolute protection. However they ensured that pertussis in vaccinated children was a much milder condition. In Australia, the Commonwealth Serum Laboratories (CSL) first manufactured pertussis vaccine in about 1920, and in 1953 a more potent vaccine was incorporated with diphtheria and tetanus vaccines as Triple Antigen, with a resulting fall in prevalence and mortality. In Melbourne the case-fatality rate fell from 21% in 1919 to 0.1% in 1969[1].

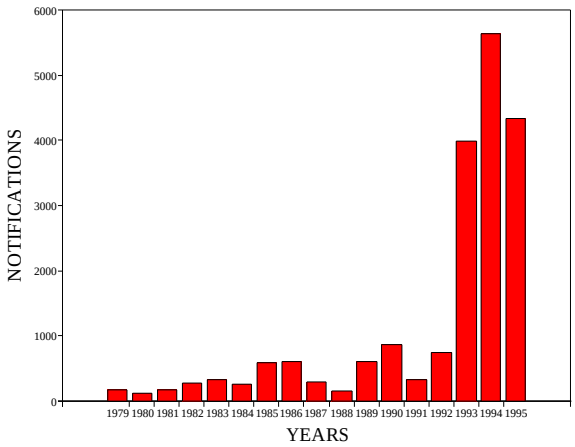
Unfortunately, immunity following vaccination does not persist. The efficacy fell from 100% to 52% five years after vaccination in one study[2], so even in well-vaccinated communities there may be pools of susceptible adults, in whom pertussis can cause persistent cough[3]. Individuals with waning immunity, such as parents or health care workers, may develop attenuated or atypical attacks of pertussis and pass the infection on to infants, but there is no evidence of long-term asymptomatic carriage. Pertussis is highly infectious: the secondary attack rate in unimmunised household contacts is about 80%[1], and chemoprophylaxis with erythromycin cannot always be relied on to eradicate *B. pertussis* from the respiratory tract[4,5]. Erythromycin prophylaxis is useful when given before the onset of the first secondary case[6] but other children in the household are often infected by the time the disease is recognised in the index case. Its use also appeared to shorten the duration of coughs in patients in one non-randomised study[7].

In the United Kingdom, the vaccine became available in the late 1940s, and the incidence of the disease declined in the 1950s following its widespread use. However a four-year cycle of epidemics peaking in autumn was still observable, even with a high uptake of vaccination. Four years is believed to be the time needed for the number of susceptible people to increase sufficiently to overcome the threshold for herd immunity[8]. In the mid-1970s, following adverse publicity about side effects, uptake fell from more than 80% to less than 40% of children. This was followed by several large outbreaks of whooping cough, the most recent of which occurred in 1993[9]. In Australia, pertussis has been epidemic since 1993 (Figure). There were 5,633 cases notified in 1994, and 4,336 in 1995[10], with increasing numbers of cases in older age groups.

Since the 1980s, there have been cyclical resurgences of pertussis in the United States of America[11]; in 1993, 6,335 cases were reported, the most in 26 years[12]. Analysis of this epidemic in Cincinnati showed that it occurred primarily among children who had been appropriately immunised, confirming the failure of whole-cell pertussis vaccine to give full protection against the disease.

In Japan there was such concern about side effects that vaccination was suspended for a short period in 1975.

Figure. Notifications of pertussis, 1979 to 1995[1]



1. Data courtesy of Communicable Diseases Network Australia New Zealand, National Notifiable Diseases Surveillance System, 1996.

This concern stimulated the development in Japan of the first acellular pertussis vaccines.

Pathogenesis and epidemiology

The organism attaches to the ciliated respiratory epithelium. Here it multiplies rapidly and produces pertussis toxin and other toxins (which cause various effects, including the characteristic lymphocytosis). The toxins damage cilia and destroy ciliated epithelial cells. It is uncertain whether the cough is due to a central toxic effect, local respiratory inflammation, or both. The organism rarely invades deeper tissues or causes bacteraemia. Complications include apnoea, dehydration, bronchitis, pneumonia, convulsions and later bronchiectasis.

The pharmacological effects mediated by the various toxins are preceded by attachment of the organism to the respiratory mucosa. Important here are:

- filamentous haemagglutinin (FHA), which is involved in adherence and which stimulates production of antibodies that protect against respiratory challenge with live organisms;
- pertactin (PRN, formerly called 69-kd protein) a non-fimbriated antigen, which also plays a role in attachment and stimulates antibody production; and
- the fimbrial agglutinogens (AGGs), surface antigens which stimulate agglutinating antibodies. There are three AGGs which infect humans (1, 2 and 3), giving rise to three serotypes – type 1,2,3; type 1,2; and type 1,3. DNA fingerprinting of the organism has recently revealed a large number of DNA types, but these do not correlate with the more widely studied serotypes[9].

It seems that serotype 1,2 predominates in unvaccinated communities. It was present in the early vaccines and was highly protective, but its use slowly led to the emergence of type 1,3 as the predominant serotype in many countries[13,14]. When uptake of immunisation declined in the United Kingdom in the 1970s, type 1,3 became less common again, suggesting that organisms bearing AGG2 have a colonising advantage in unvaccinated individuals. This effect continued in the United Kingdom until the 1980s. In Australia, a similar situation was evident in the serotypying studies carried out at Royal Alexandra Hospital for Children between 1981 and 1990. Type 1,2 was prevalent up to 1987, while type 1,3 predominated later in the decade (Maureen Gapes, personal communication).

In the early 1980s in Australia there was an increase in the number of culture-positive cases and in the proportion of these involving preschool-age groups compared with the 1970s when the highest proportion was in younger children. In 1984 the National Health and Medical Research Council (NHMRC) appointed a working party to investigate the increased number of cases and the Australia-wide change in age distribution. The working party concluded that the change in vaccination schedule from four to three doses of pertussis vaccine in 1978-79 had been partly responsible and recommended reintroduction of the fourth dose (implemented in 1985). It also found that batches of the vaccine (produced from 1978 to early 1980) had low potency and advised that this should be rectified. One study of a series of children presenting to hospital showed a vaccine efficacy of 67% after three doses of vaccine in patients aged 1-4 years, and no protection in children from the age of four years onward[15]. In 1984 aluminium adjuvant was added to the vaccine available in Australia. Following these changes, cases in vaccinated preschoolers became less common and by 1989 and 1990 serotype 1,3 again predominated in Sydney. Due to time constraints, serotyping of isolates has not been performed at Royal Alexandra Hospital for Children since 1990. Of interest is the fact that in Australia the incidence of pertussis peaks in summer months.

Pertussis vaccination

Whole-cell vaccine

Pertussis vaccination is now routine in most countries and is incorporated into the World Health Organization's Expanded Programme on Immunisation (EPI). Fine[8] has noted that, although the vaccine has been responsible for significantly reducing the disease incidence and the morbidity, the public's concern has now 'paradoxically' focused on the uncommon side effects of vaccination, rather than on the common complications of this increasingly rare disease.

Whole-cell vaccine is a brew containing whole organisms. It also contains variable but significant amounts of lipopolysaccharide endotoxin. This small amount of endotoxin probably plays a major role in causing the fever, local reactions, pain and crying which follow vaccination. A study by the Commonwealth Serum Laboratories (CSL) several years ago showed that an endotoxin-depleted whole-cell vaccine had a much lower incidence of side effects. None of the acellular vaccines contains this endotoxin. The side effects of Triple Antigen (DTP), which contains whole-cell pertussis vaccine, used in a recent Australian study are shown in Table 1[16]. A previous larger Australian study found that about 0.1% of infants had a convulsion and a further 0.1% had a hypotonic-hyporesponsive episode after DTP[17]. The rate of anaphylaxis is estimated at about 1 in 50,000 doses and of acute encephalopathy at 0-10.5 cases per million vaccinations.

Vaccine potency, efficacy and serotype

The World Health Organization (1979) recommends that whole-cell vaccine includes AGGs 1, 2 and 3, and for potency passes the mouse protection test, in which the degree of protection induced by the test vaccine is compared with that provided by a reference vaccine when the mice are challenged by intracerebral inoculation with the live organism[18]. Each dose should contain not less than four international units. However, to date there are no clear serological correlates with protection or efficacy. For example, although subjects vaccinated with acellular vaccines may have higher antibody lev-

Table 1. Side effects of Triple Antigen containing whole-cell pertussis vaccine (DTP) in 591 Australian children

Reaction	Percentage[1]
Systemic	
Fever ≥ 38°C[2]	16
Irritability	90
Crying - intermittent, inconsolable	40
Crying - persistent high-pitched	8
Vomiting	11
Hypotonic-hyporesponsive episode	0
Convulsions	0
Local	
Redness ≥ 2.4 cm	27
Induration ≥ 2.4 cm	30
Swelling	45
Tenderness	46

1. Mean after first three doses at two, four and six months of age, to the nearest whole number.
2. All children were given at least two doses of paracetamol around the time of each vaccination.

els, they cannot be said to be more or less likely to be protected than subjects vaccinated with whole-cell vaccines. There are no efficacy data published for the whole-cell vaccine currently marketed in Australia.

British workers have found that when whole-cell vaccine is used widely, the predominant serotype in the community changes from 1,2 to 1,3. They believe that acellular vaccines should be shown to be efficacious against both serotypes before being licensed for infants[19]. Some data on this are available from the German studies[20]. Vaccine efficacy is conventionally expressed as protection from disease, rather than as protection from infection. Fine and Clarkson[21] believe that there is evidence that whole-cell vaccine protects to a lesser extent against infection than against disease. This gives the organism the ability to continue to circulate in the community. However, Preston argues that the very low rates of disease in some Eastern European countries after many years of high compliance with whole-cell vaccination is against this hypothesis[22].

Acellular vaccines

Once the biologically active components of the organism had been identified, development of acellular pertussis vaccines became possible. Those which have been produced commercially contain inactivated concentrates from the culture fluid of the organism, and may contain one to five components which include FHA, one or two agglutinogens, pertactin and pertussis toxin. It is not certain which components are most correlated with protection from infection, but it is likely that both FHA and pertactin are important.

Acellular vaccines were licensed for use in Japan in 1981. Initially they were used only in two year old children; in 1988 they became available for infants. There has been a dramatic decline in the prevalence of the disease since their introduction. Protective efficacy has been high and reactogenicity low, and protection has been shown to last for up to ten years. However, in Sweden, which had ceased using its whole-cell vaccine in 1979 because of limited efficacy[23], the first trials of acellular vaccines did not confirm the high rate of protection seen in Japan. In 1985, an early trial of two acellular vaccines (a single-component (PT) vaccine and a two-component (PT & FHA) vaccine) showed efficacies of only 54% and 69% respectively[24].

In the 10 years since the Swedish studies, the vaccines have been further improved and subjected to a series of rigorous trials. Most now contain two to five pertussis antigens, together with aluminium and a preservative. They generate much higher antibody titres than the whole-cell vaccines; they also provoke levels of cellular immunity similar to those seen after natural infection. Importantly, they are associated with fewer and milder side effects than the whole-cell vaccine. In the large European trials, the frequency of side effects with the acellular vaccines was similar to the frequency after the control vaccine which contained only diphtheria and tetanus toxoids (DT) (Table 2)[25,26]. The rate of extremely rare adverse events such as encephalopathy after acellular vaccines is not yet known and will only be evident with careful post-marketing surveillance.

Because the question of efficacy was not resolved at the time, the United States in 1991 licensed acellular vaccines only for the fourth and fifth doses of the childhood immunisation schedule. Recent United States trials of 13 acellular vaccines in infants showed that they were associated with fewer and less severe adverse reactions than whole-cell vaccines[27,28]. The United States of America is currently proceeding to license a three-component acellular vaccine for use in infants, and Germany has already done so. The effects of this change will require close monitoring.

Efficacy studies of acellular vaccines

Even though the acellular vaccines had been shown to stimulate development of high levels of antibodies (that is, they were immunogenic), their ability to prevent clinical infection (their efficacy) still had to be demonstrated. Six National Institutes of Health-sponsored large-scale phase III efficacy trials, which included both a DT control group and a whole-cell comparison group, are now completed or near completion. Preliminary results of these randomised double-blind trials, involving 25,000 children in Europe, show clear evidence of efficacy (in Sweden 85% for a five-component vaccine (PT, FHA, pertactin, AGGs 1 and 2) and 59% for a bivalent vaccine (PT and FHA)[26]; and in Italy 84% for two 3-component vaccines (PT, FHA, pertactin)) and fewer side effects compared with the control group that received whole-cell pertussis vaccine[25,26,29]. According to Edwards and Decker[30], these results suggest that the presence of fimbrial pro-

Table 2. Side effects[1] of two 3-component acellular pertussis vaccines (DTaP) compared with whole-cell pertussis vaccine (DTP) and DT vaccine in 14,751 Italian infants[2]

Reaction	Percentage			
	Acellular vaccine 1 (13,761 doses)	Acellular vaccine 2 (13,713 doses)	Whole-cell vaccine (13,520 doses)	DT vaccine (4,540 doses)
Systemic				
Fever ≥ 38°C[3]	7.2	4.3	40.5	3.4
Crying - persistent ≥ 3 hours	0.04	0.07	0.4	-
Hypotonic hyporesponsive episode	-	0.007 (1 case)	0.07 (9 cases)	0.04 (2 cases)
Seizures (within 48 hours)	0.007 (1 case)	-	0.02 (3 cases)	-
Local				
Swelling	9	7	26	6
Tenderness	4.6	4.6	30	4.5

1. Within 48 hours of each of the first three doses.
2. Based on reference 25.
3. Rectal temperature.

teins in the five-component vaccine did not improve efficacy, but the presence of pertactin increased protection in the trivalent vaccine compared with the bivalent vaccine. In these studies, the whole-cell vaccine (a product licensed and manufactured in the United States of America and not available in Australia) showed efficacies of 36% in Italy and 48% in Sweden. Trials under other sponsors are also under way in Germany, Senegal and Sweden. One recent German study of a three-component vaccine given at ages three, four and five months showed an 88.7% efficacy in children exposed within their household to a case of pertussis, compared with 98% for a German whole-cell vaccine[31].

In most of these trials, the acellular pertussis vaccines were combined with diphtheria and tetanus toxoid, and the resulting combination (DTaP) was compared with the conventional DTP which contains whole-cell pertussis vaccine. Tetravalent and pentavalent vaccines, in which Haemophilus influenzae type B and hepatitis B vaccines are added to DTaP, have also been developed and tested. Such combinations are difficult to make with the acellular vaccines. They will reduce the number of immunisations required in infancy.

Eradication: is it possible?

On both practical and theoretical grounds, eradication seems unlikely in the immediate future. Infants who are too young to have completed their primary schedule need to be protected by herd immunity. Commencing vaccination in the neonatal period has not proved successful and the adoption of an accelerated infant schedule in Britain in June 1990 (doses given at two, three and four months) has not yet reduced the number of cases in children under the age of one year[14].

It is possible that some acellular vaccines may prove more efficacious than whole-cell vaccine and be more suitable for use as adult boosters. Future vaccines which enhance specific IgA production and are administered orally are being developed. So are synthetic peptide vaccines aimed at inducing an immune response which will block adhesion of the organism to the cell. But these are still to be evaluated in clinical trials and are likely to be a decade away.

Current situation in Australia

Not until a vaccine is licensed will the National Health and Medical Research Council decide whether acellular vaccines are to be recommended for use in the routine infant schedule in Australia. Their widespread use may depend on whether their appreciably higher cost will be publicly funded in the same way that other routine childhood vaccines now are. For the present, we must continue to use whole-cell DTP. Serious reactions to vaccination, which contraindicate further doses of whole-cell pertussis vaccine, including anaphylaxis and unexplained encephalopathy, are very rare[32]. Doctors should strongly discourage the inappropriate deferral of pertussis immunisation due to mild illness, and the omission of pertussis vaccine because of inappropriate contraindications.

Once the new acellular vaccines are licensed, they are likely to be more acceptable to parents, especially when they are included in combination with other antigens. They are also likely to be useful for boosting immunity in adults, perhaps combined with the ten-yearly boosters of adult diphtheria and tetanus.

Acknowledgements

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BOVINE SPONGIFORM ENCEPHALOPATHY AND POSSIBLE HUMAN RISKS

Based on a World Health Organization press release of 3 April 1996

At a consultation organised by the World Health Organization (WHO) in Geneva on 2-3 April 1996, a group of international experts reviewed the public health issues related to bovine spongiform encephalopathy (BSE) and the emergence of a new variant of Creutzfeldt-Jakob disease (CJD), as officially reported by the United Kingdom on 20 March 1996.

The consultation made recommendations, based on the latest scientific information, to minimise transmission of BSE among animals and to reduce as much as possible any exposure of humans to the BSE agent.

Bovine Spongiform Encephalopathy

BSE is a transmissible spongiform encephalopathy (TSE) in cattle which was first identified in the United Kingdom in 1986. It is one of a group of similar degenerative diseases which occur in several animal species. Transmission of BSE to cattle appears to have been via contaminated meat and bone meal in concentrate feed, with sheep or cattle being the original source. The United Kingdom is the only country with a high incidence of the disease, and the epidemic there appears to have been due mainly to feeding affected bovine material to cattle before the ruminant (cattle, sheep and goats) feed ban in July 1988 took effect. There is no evidence to date of either maternal or horizontal transmission of BSE.

The incidence of the disease is declining significantly in the United Kingdom, although the measures introduced have not halted the epidemic. The worldwide distribution of BSE is not known precisely, but it has been reported at a much lower incidence than in cattle in other European countries. In these latter countries only part of the BSE cases could be related to the consumption of possibly BSE-contaminated feed.

Variant Creutzfeldt-Jakob disease (V-CJD)

The group reviewed the clinical and pathological data from the ten cases in the United Kingdom. The disease has occurred at younger ages than is usual for classical CJD and shows several clinical and pathological differences. Based on findings in these ten cases, the group established a case definition to facilitate better surveillance, which is necessary to determine the incidence and distribution of this syndrome.

The group concluded that there is no definite link between BSE and V-CJD, but that circumstantial evidence suggests exposure to BSE may be the most likely hypothesis. Further research on both diseases is urgently required.

Possible exposure to BSE has already been greatly reduced by measures taken in the United Kingdom. Implementation of the recommendations by this consultation should further reduce risk from exposure to BSE to minimal levels.

Recommendations

Bovine Spongiform Encephalopathy

1. No part of any animal which has shown signs of TSE should enter any food chain, human or animal. All countries must ensure the slaughter and safe disposal of TSE-affected animals so that TSE infectivity cannot enter any food chain. All countries should review their rendering procedures to ensure that they effectively inactivate TSE agents.
2. All countries should establish continuous surveillance and compulsory notification for BSE according to recommendations established by the Office International des Epizooties in Paris. In the absence of surveillance data, the BSE status of a country must be considered as unknown.
3. Countries where BSE exists in native cattle should not permit tissues that are likely to contain the BSE agent to enter any food chain.
4. All countries should ban the use of ruminant tissues in ruminant feed.
5. With respect to specific products:
   - Tests on milk from BSE-infected animals have not shown any BSE infectivity, and there is evidence from other animal and human spongiform encephalopathies to suggest that milk will not transmit this disease. Milk and milk products, even in countries with high incidence of BSE, are therefore considered safe.
   - Gelatine is considered safe for human consumption since its preparation involves a chemical extraction process that destroys BSE infectivity.
   - Tallow is likewise considered safe if effective rendering procedures are in place.
6. With respect to medicinal products, which differ from food in that they can be injected as well as taken orally, measures to minimise the risk of transmitting the BSE agent were developed at a previous WHO consultation in 1991 and continue to be applicable.
   - As more information becomes available these measures will be reviewed and strengthened if necessary.

- The importance of obtaining materials destined for the pharmaceutical industry from countries which have a surveillance system in place and which report either no or sporadic cases of BSE is reiterated.
- Removal and inactivation procedures contribute to the reduction of the risk of infection. But it must be recognised that the BSE agent is remarkably resistant to physico-chemical procedures which destroy the infectivity of common microorganisms.

7. Research on TSE should be promoted, especially on rapid diagnosis, agent characterisation, and epidemiology of TSEs in humans and animals.

Variant Creutzfeldt-Jakob disease (V-CJD)

1. The geographic distribution of V-CJD, although reported at present only in the United Kingdom, needs to be further investigated.
2. While the most likely hypothesis at present for this newly recognised variant is exposure to the BSE agent, further data from scientific studies on these variant cases are urgently required to establish a link. More monitoring and surveillance studies on all forms of CJD are required throughout the world, modelled on current European collaborative studies.
3. Exposure to BSE from beef and beef products has already been substantially reduced by the measures taken in the United Kingdom. Exposure to BSE has always been lower in other countries. The group considered that implementation of their recommendations will ensure that any continuing risk of exposure to BSE in beef and beef products will be reduced to a minimum.

As surveillance worldwide is increased for both BSE and V-CJD, more information will become available in the coming months. WHO will keep these developments under review and update the recommendations as appropriate.

Australian Task Force

The task force established by the Federal Government (see *Communicable Diseases Intelligence* 1996;20:170) will consider this report together with scientific information from other sources when providing advice to the Government.

A toll-free telephone line is available so that members of the public can enquire about products which may be suspect and about diseases BSE and CJD. The toll-free number is 1800 02 06 13 and is open from 8.30am to 8.30 pm every day.

OVERSEAS BRIEFS

In the past fortnight the following information has been provided by the World Health Organization (WHO).

Cholera

Zaire: An outbreak of cholera has been reported by the Ministry of Health that has affected four districts in Kinshasa Province (Barumbu, Kinshasa, Limete/Kingaba and Kingwala - all newly infected areas). A total of 147 cases with 33 deaths had been reported to 29 March. An interdisciplinary team have donated money and supplies to help control the outbreak. Measures relating to water quality and sanitation.

Other countries reporting cholera in the past week are Benin, Mali and Niger.

Buffalopox in India

Five suspected paediatric cases of buffalopox virus infection from two villages in Beed district, Maharashtra State were under investigation in March. The cases coincided with infections in cattle. During 1992-1994, three cases of buffalopox were confirmed in humans and seven in buffalos in two districts of Maharashtra State. The clinical features in human cases included fever, lymphadenopathy and pox lesions on the hands whereas lesions in affected animals were mainly on the teats and udder. Sero-surveys in the affected villages revealed neutralising antibodies in sera of affected humans and in 70% of their contacts. The investigations were conducted by the National Institute of Virology in Pune in collaboration with the Maharashtra State Public Health and Animal Husbandry Departments.

CORRESPONDENCE

Accelerated Primary Immunisation Schedule

Dr David Sloan, Epidemiologist, PO Box 946, Rockhampton, Queensland 4700

As a newcomer from the United Kingdom, I don't want to appear presumptuous, but may I argue the case for introducing the accelerated primary immunisation schedule to Australia.

This schedule was introduced in 1990[1] in the United Kingdom with diphtheria, pertussis and tetanus (DPT) and polio vaccines being given at two, three and four months, instead of at three, five and nine months. When *Haemophilus influenza* type b (Hib) vaccine was later introduced, it was added to this schedule[2]. Reasons for its introduction were to assure earlier protection, especially against pertussis which is more severe in young infants, and to improve uptake[2]. Those reasons were endorsed by the WHO and backed up by research and experience elsewhere[3]. Subsequently, a higher uptake was indeed confirmed[4] and the incidence of pertussis fell[5]. Additionally, there was a lower level of side effects[6].

The problem of the mobility of young families who move out of the district before completion of the primary course is addressed by the schedule[2].

Follow-up serological studies have shown that while the antibody levels were lower than they were following the previous schedule, the levels remained protective; and after a year there was little difference between the levels[7,8]. It is anticipated that antibody levels will remain adequate until the preschool booster, though some have argued for a booster in the second year of life[8]. The relatively low level of immunisation which has been demonstrated in a recent survey[9] has been highlighted as a major public health problem in Australia[10].

Major epidemics of pertussis in 1993 and 1994 (with 3,990 and 5,633 notified cases respectively[11,12]) mean that young infants are at a significant risk of infection which a primary course completed earlier should prevent. If the United Kingdom experience of a higher uptake of the primary course is duplicated in Australia, then herd immunity will be lifted and there should be fewer cases and less risk of transmission. The concerns expressed by some in the United Kingdom about falling antibody levels are even less relevant here with boosters of DPT and Hib, and DPT at 18 months and prior to school entry respectively[13], as opposed to a preschool booster of polio and DT (only) in the United Kingdom[2].

No doubt your readers will indicate any reasons, if there are any, for not instituting such a change.

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NOTICES TO READERS

Development of a National Communicable Diseases Surveillance Strategy

Extension of time for the lodgement of submissions

A National Communicable Diseases Surveillance Strategy (NCDSS) is being developed to enhance Australia's capacity to manage communicable diseases. The NCDSS aims to provide a comprehensive annual picture of the burden of communicable diseases that will improve national capacity to prioritise communicable disease threats and to develop control strategies. Issues being considered in the development of the Strategy include current surveillance arrangements and national co-ordination of surveillance activities. Laboratory issues are being considered in a related review. The Strategy is being developed on behalf of the Chief Health Officers of Australia. The process has been endorsed by the Australian Health Ministers' Advisory Council.

Submissions are invited on the development of a NCDSS. Further information, including terms of reference can be obtained by calling (06) 289 8351 or by faxing a request to (06) 289 7791. Please include your name, address and telephone number.

How to make your submission

Please make your submission in writing, word processing documentation diskette, or on audio tape, and include your name, address and phone number. Submissions should be sent to:

Dr Graeme Oliver, Secretary
NCDSS Committee
AIDS / Communicable Diseases Branch
MDP 15
Department of Human Services and Health
GPO Box 9848
Canberra ACT 2601

The new closing date for receipt of submissions is Monday 29 April 1996.

All submissions will be held in a register of submissions which can be accessed by the public. If you would like your submission to be treated as confidential, please indicate this clearly. Submissions may however be subject to release under the Freedom of Information Act 1992.

Seventh Arbovirus Research in Australia/Second Mosquito Control Association of Australia Conference

Ocean Blue Resort
Surfers Paradise, Gold Coast, Queensland, Australia
25-29 November 1996

Call for papers

The Arbovirus Research in Australia symposia are recognised internationally as the only specialist conference available to the broad range of disciplines working on arboviruses, of both human and veterinary importance. The Mosquito Control Association of Australia is a national organisation of field practitioners and researchers.

The meeting will be held in the week after the XIV International Congress for Tropical Medicine and Malaria to be held in Nagasaki.

The five-day program will be structured around vectors and control during the first part of the week, with a plenary session by internationally recognised speakers covering broad topics of global relevance. The following two days will cover epidemiology, virology, clinical medicine and molecular biology.

Papers (up to 15 minutes long) are invited. Submissions are particularly welcome on the subjects of dengue viruses, Japanese encephalitis virus, Ross River virus, arbovirus diseases affecting livestock, emerging diseases, innovative mosquito control, living with insect growth regulators, vector biology, application technology and other operational issues, surveillance and management plans, and contemporary diagnostics.

Further information and registration forms can be obtained from the conference convenor

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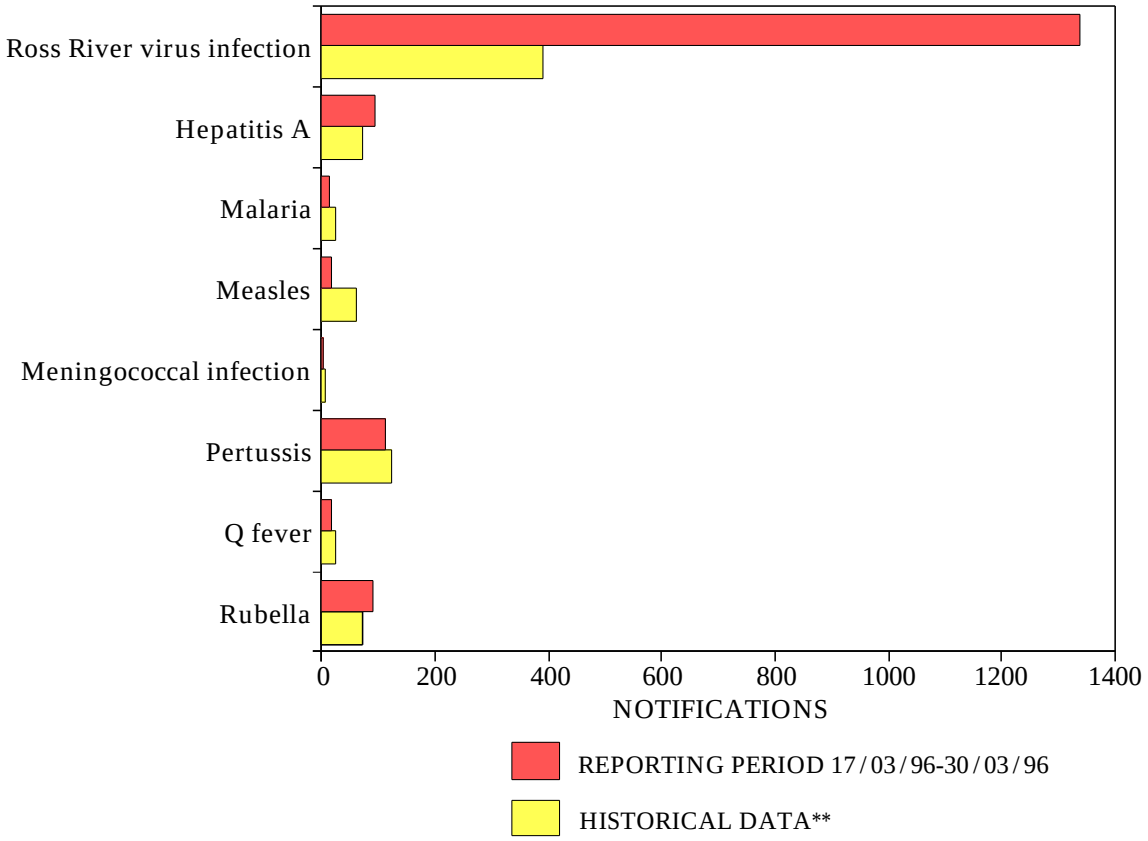
COMMUNICABLE DISEASES SURVEILLANCE

National Notifiable Diseases Surveillance System, 17 to 30 March 1996

There were 3,420 notifications received for this two week period (Tables 1, 2 and 3, and Figure 1).

- There were 1,338 notifications of **Ross River virus infection**, 20% more than the number reported for the previous fortnight. The male:female ratio was 1:1.2. As for the previous three reporting periods, all age groups were affected, although 59% of cases were aged between 30 and 54 years. The greatest number of cases was reported from Queensland (especially the Southern and Central Coastal Statistical Divisions, and Darling Downs). In New South Wales the Northern and Richmond-Tweed Statistical Divisions reported most cases. In Western Australia the greatest numbers and rates were reported from the South West Statistical Division.
- Seventy-eight cases of **Barmah Forest virus infection** were reported from Queensland and one case from the Australian Capital Territory; 61% of the cases were in the age range 30-54 years.
- Two cases of **brucellosis** were reported from Queensland and one from Victoria. Two cases were in males in the age group 30-34 years, and one case in a female in the age group 55-59 years.
- Notifications of **campylobacteriosis** remain at a high level, 447 cases being reported in the current fortnight. The male:female ratio was 1.2:1; all age groups were affected, with 26% of cases being aged less than five years.
- There were 273 notifications of **chlamydial infection** received, 52% of them being reported from Queensland. The male:female ratio was 1:2.2; 85% of the cases were aged between 15 and 29 years.
- There were 127 notifications of **gonococcal infection** received; 86 cases were male and 40 cases were female, the sex of the remaining case being not reported; 66% of the cases were aged between 15 and 29 years.
- Two cases of ***Haemophilus influenzae* type b infection** were reported, both females, a child aged one year from New South Wales and an infant from the Northern Territory.
- There were 94 cases of **hepatitis A** reported, 65% of them in males. The cases were from all age groups from 0-4 years to 60-64 years, with one case in an older female; half of the cases (46) were in males aged from 20 to 44 years. The majority of cases were reported from the Metropolitan Statistical Divisions of Sydney (32 cases), Melbourne (15 cases) and Brisbane (12 cases).
- Eight cases of **hepatitis B (incident)** were reported; four were males and three were females, the sex of the remaining case not being reported. Their ages were from five age groups between 15-19 years and 45-49 years.
- One case of **hydatid disease** was notified, an elderly male from the Victorian Statistical Division of Melbourne.
- Seven cases of **legionellosis** were reported. Four cases were in males and two in females, the sex of the remaining case being not reported. They were from age groups ranging from 35-39 years to 80-84 years. The cases were reported from five separate statistical divisions in four States and Territories.
- Twelve cases of **leptospirosis** were reported. Their ages ranged from 30 to 70 years. All but one were males. Three cases were reported from the Hunter Statistical Division of New South Wales, and the remainder from eight separate Statistical Divisions in Queensland, Victoria and Tasmania.
- Three cases of **listeriosis** were reported; three were males and the sex of the remaining case was not reported. All were over 60 years of age. The cases were reported from the Statistical Divisions of Melbourne, Victoria and Northern, South Australia.
- Fourteen notifications of **malaria** were received; 11 were males and three were females. The ages of cases ranged from 16 years to 87 years. The cases were reported from seven separate Statistical Divisions in four States and Territories.
- Eighteen cases of **measles** were reported; six cases were male and 12 cases were female. Their ages ranged from less than one year (3 cases) to 45 years, seven cases being under five years of age. There was one apparent cluster of two cases reported from the same postcode area in Victoria during the current reporting period.
- There were three cases of **meningococcal infection** reported from separate statistical divisions in three States. There were two males in the age group 15-19 years and one female aged less than one year. There was one apparent cluster of two cases reported from the same postcode area in Queensland during the current and previous two-week reporting periods.
- There were 112 notifications of **pertussis**; 38 cases were male and 73 cases were female, the sex of the remaining case being not reported. All age groups but one from 0-4 years to 75-79 years were represented. Eight cases were aged less than one year, and a further seven cases were less than five years of age. Nine apparent clusters of two or three cases each were reported from the same postcode area during the reporting period; the apparent clusters occurred in four separate States and Territories.

Figure 1. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods: the corresponding periods of the last 3 years and the periods immediately preceding and following those.

Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 17 to 30 March 1996

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[1]			
									This period 1996	This period 1995	Year to date 1996	Year to date 1995
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae b infection	0	1	1	0	0	0	0	0	2	7	19	26
Measles	0	5	0	3	0	0	8	2	18	59	136	592
Mumps	0	1	0	NN	0	0	1	1	3	5	35	32
Pertussis	1	35	1	28	25	0	22	0	112	133	793	1323
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0
Rubella	6	11	0	39	5	1	27	2	91	71	861	767
Tetanus	0	0	0	0	0	0	0	0	0	0	1	2

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

NN Not Notifiable.

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 17 to 30 March 1996

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This period 1996	This period 1995	Year to date 1996	Year to date 1995
Arbovirus infection												
Ross River virus infection	1	168	11	908	3	-	36	211	1338	99	4351	647
Dengue	0	0	0	0	0	-	0	0	0	0	13	6
Barmah Forest virus infection	1	0	0	78	0	0	0	0	79	23	254	121
NEC[3,4]	0	17	1	6	0	0	18	3	45	68	128	109
Campylobacteriosis[5]	22	-	18	107	115	19	92	74	447	397	3072	2779
Chlamydial infection (NEC)[6]	2	NN	19	143	0	14	42	53	273	217	1782	1611
Donovanosis	0	NN	0	0	NN	0	0	0	0	1	17	25
Gonococcal infection[7]	1	26	25	34	0	0	7	34	127	108	891	759
Hepatitis A	3	37	3	23	0	0	22	6	94	68	698	500
Hepatitis B	4	0	0	1	0	2	1	0	8	15	70	91
Hepatitis C incident	0	1	0	0	0	0	0	0	1	1	7	15
Hepatitis C unspecified	13	0	1	115	0	9	57	34	229	315	2262	2135
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	0	5	8
Legionellosis	0	2	1	1	0	0	3	0	7	18	46	67
Leptospirosis	0	3	0	5	0	1	3	0	12	1	64	35
Listeriosis	0	0	0	0	1	0	3	0	4	6	16	27
Malaria	1	5	0	0	0	0	5	3	14	29	186	145
Meningococcal infection	0	1	0	1	0	0	1	0	3	7	62	80
Ornithosis	0	NN	0	0	0	0	3	0	3	14	27	45
Q fever	0	10	0	6	0	0	1	0	17	18	120	121
Salmonellosis (NEC)	2	41	23	112	22	6	31	39	276	336	1917	2294
Shigellosis[5]	0	-	3	14	2	0	1	4	24	47	186	259
Syphilis	0	27	15	27	0	2	0	6	77	54	358	473
Tuberculosis	1	9	4	9	2	1	3	6	35	49	258	299
Typhoid[8]	0	0	0	0	0	0	2	0	2	2	30	26
Yersiniosis (NEC)[5]	0	-	0	9	0	0	0	0	9	16	81	122

1. For HIV and AIDS, see Tables 5 and 6. For rarely notified diseases, see Table 3 .
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. Tas: includes Ross River virus and dengue.
4. WA, NT and Vic: includes Barmah Forest virus.
5. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
6. WA: genital only.
7. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
8. NSW, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 17 to 30 March 1996

DISEASES	Total this period	Reporting States or Territories	Year to date 1996
Botulism	0		0
Brucellosis	3	Qld 2, Vic 1	9
Chancroid	1	Tas	1
Cholera	0		4
Hydatid infection	1	Vic	11
Leprosy	0		2
Lymphogranuloma venereum	0		0
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1994.

- Seventeen notifications of **Q fever** were received. Sixteen cases were reported from eight separate rural Statistical Divisions in New South Wales and Queensland. One case was reported from the metropolitan Statistical Division of Melbourne. All but one of the cases were males. All but two of the age groups between 20-24 years and 60-64 years were represented.
- There were 91 cases of **rubella** reported; 53 cases were male and 34 cases were female, the sex of the remaining cases being not reported. The recorded ages of cases were from all five-year age groups up to 60-64 years; 36% of the cases (33 cases) were reported in males 15-24 years of age, and 21% (19 cases) in women aged 15 to 44 years.
- There were 276 cases of **salmonellosis** reported; 126 cases were male and 143 cases were female; the sex of the remaining seven cases was not reported; 48% of the cases were aged less than five years.
- Seventy-seven cases of **syphilis** were reported; 39 cases were male and 38 cases were female. All age groups but one from 15-19 years to 80-84 years were represented. There was one case reported in a male child in the age group 5-9 years.
- There were 35 cases of **tuberculosis** reported; 12 cases were male and 20 were female, the sex of the remaining cases being not reported. All age groups between 10-14 years and 85-89 years were represented. There was one case reported in a female child under 5 years of age.
- Two cases of **typhoid** were reported; both were female. The cases were reported from the Statistical Division of Melbourne.
- Nine cases of **yersiniosis** were reported. One case was male, and eight were female. Two cases were reported in children under five years of age, the remainder of the cases being aged between 10 and 49 years.

Australian Sentinel Practice Research Network

Data for weeks 11 and 12 ending 17 and 24 March respectively are included in this issue of *CDI* (Table 4). The rate of reporting of influenza-like illness continues to rise (Figure 2).

Figure 2. ASPREN consultation rate for influenza, 1996

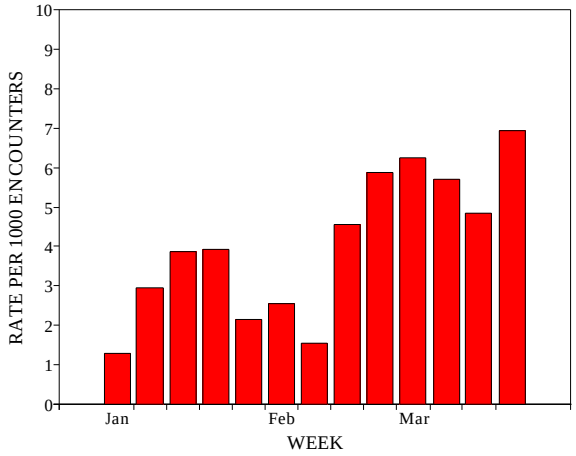


Table 4. Australian Sentinel Practice Research Network, weeks 11 and 12, 1996

Condition	Week 11, to 17 March 1996		Week 12, to 24 March 1996	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	45	4.8	67	6.9
Rubella	0	0.0	2	0.2
Measles	1	0.1	0	0.0
Chickenpox	3	0.3	16	1.7
Pertussis	6	0.7	6	0.6
Gastroenteritis	144	15.5	151	15.6

Australian Encephalitis: Sentinel Chicken Surveillance Programme

AK Broom[1], *J Azuolas*[2], *JS Mackenzie*[3], *L Melville*[4], *DW Smith*[5] *and PI Whelan*[6]

November and December, 1995

Sentinel chicken serology was carried out for 15 of the 22 flocks in Western Australia in November and December 1995. There were no seroconversions during this period.

Eight flocks of sentinel chickens from the Northern Territory were tested in November and December. There was one new seroconversion to Kunjun virus from Howard Springs in Darwin in December.

The Sentinel Chicken Surveillance Programme started again in Victoria in November and there were no seroconversions during November and December. However, the program in New South Wales is not being carried out in 1996.

January and February 1996

Sentinel chicken serology was carried out for 20 of the 22 flocks in Western Australia in January and February. There were no seroconversions during this period.

Eight flocks of sentinel chickens from the Northern Territory were also tested during this period. There were no new seroconversions.

There were no seroconversions to flaviviruses in the sentinel chicken flocks in Victoria during January and February 1996.

1. Department of Microbiology, The University of Western Australia
2. Veterinary Research Institute, Victoria
3. Department of Microbiology, The University of Queensland
4. Berrimah Agricultural Research Centre, Darwin, NT
5. Pathcare, Perth
6. Medical Entomology Branch, Department of Health and Community Services, Darwin, NT.

Virology and Serology Reporting Scheme

There were 771 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 5, 6 and 7).

- **Ross River virus** was reported for 117 patients this fortnight. Diagnosis was by IgM detection (84), single high titre (27), fourfold change in titre (6). One hundred and fourteen reports were from Western Australia and 3 from Victoria. Of the 1,625 cases of **Ross River virus** for 1996, 870 (54%) were aged between 25 and 44 years (Figure 3).
- One report of **Barmah Forest virus** was reported this period for a 35 year old female from southwest Western Australia. Diagnosis was by IgM detection.
- **Parainfluenza virus type 1** was reported for 9 patients this reporting period. Diagnosis was by virus isolation (4) and antigen detection (5). Reports have remain low throughout 1995 and early 1996 (Figure 4).

Figure 3. Ross River virus laboratory reports, 1996, by age group and sex

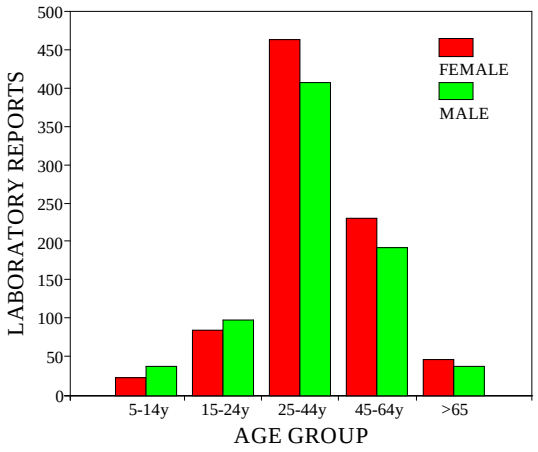


Figure 4. Parainfluenza virus type 1 laboratory reports, 1992 to 1996, by month of specimen collection

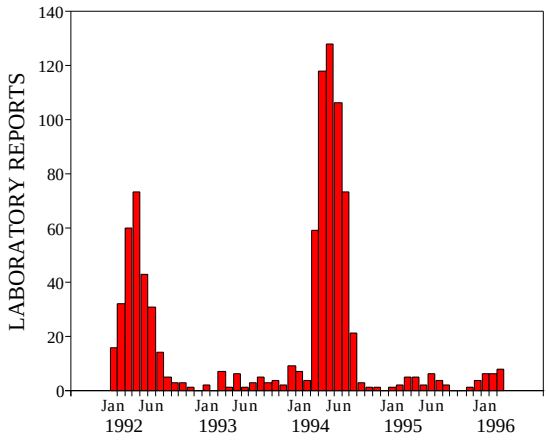
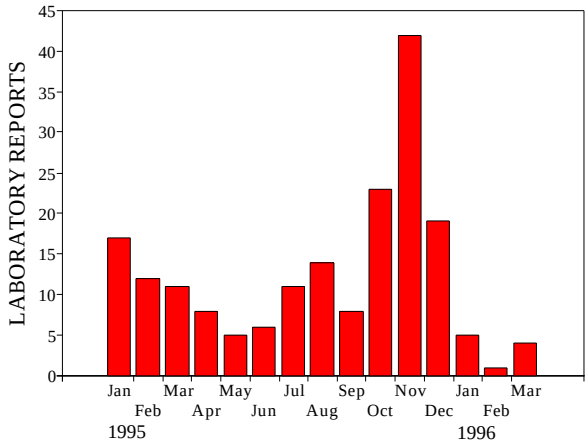


Figure 5. *Chlamydia psittaci* laboratory reports, 1995 to 1996, by month of specimen collection



- Nineteen reports of ***Bordetella*** were received this reporting period, 18 ***Bordetella pertussis*** and 1 ***Bordetella* species.**

  Diagnosis was by antigen detection (one), IgA detection (16), single high titre (one) and method not stated (one). Included were 5 females and 14 males.

- **Norwalk Agent** was reported for 4 patients this period all from Victoria. Three of the cases were male and one sex not stated. These are the first reports of **Norwalk Agent** since December.

- Four reports of ***Chlamydia psittaci*** were received this fortnight with all reports coming from Victoria. Diagnosis was by single high titre (2) and fourfold change in titre (2). The number of reports has remained low since the peak in November (Figure 5).

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 21 March to 3 April 1996, historical data[2], and total reports for the year

	State or Territory[1]						Total this fortnight	Historical data [2]	Total reported this year
	NSW	NT	Qld	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA									
Measles virus					2		2	11.7	22
Rubella virus					3	4	7	11.2	210
HEPATITIS VIRUSES									
Hepatitis A virus		1			1	5	7	11.7	174
Hepatitis B virus					10		10	79.5	432
ARBOVIRUSES									
Ross River virus		1			3	113	117	77.0	1,653
Barmah Forest virus						1	1	8.8	90
ADENOVIRUSES									
Adenovirus type 2					1		1	0.8	17
Adenovirus type 5					1		1	0.0	1
Adenovirus type 37					2		2	0.0	3
Adenovirus type 40					2		2	0.0	2
Adenovirus not typed / pending	7				7	10	24	29.5	448

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 21 March to 3 April 1996, historical data[2], and total reports for the year, continued

	State or Territory[1]						Total this fortnight	Historical data [2]	Total reported this year
	NSW	NT	Qld	Tas	Vic	WA			
HERPES VIRUSES									
Herpes simplex virus type 1	11	11	1	7	65	52	147	148.3	1,893
Herpes simplex virus type 2	13	2		2	39	63	119	154.3	1,877
Herpes simplex not typed / pending						1	1	19.3	135
Cytomegalovirus	2		2		14	9	27	53.0	478
Varicella-zoster virus	3				10	4	17	34.7	401
Epstein-Barr virus	4	1			2	31	38	58.0	628
Herpes virus group - not typed						4	4	0.5	46
OTHER DNA VIRUSES									
Parvovirus					1		1	1.3	38
PICORNA VIRUS FAMILY									
Coxsackievirus A9				1			1	0.0	6
Rhinovirus (all types)	2				12		14	30.7	173
Enterovirus not typed / pending					6	17	23	46.7	282
ORTHO / PARAMYXOVIRUSES									
Influenza A virus					4	1	5	6.3	65
Parainfluenza virus type 1	1				6	2	9	14.7	26
Parainfluenza virus type 2						1	1	5.7	15
Parainfluenza virus type 3	1				3	4	8	12.5	228
Parainfluenza virus typing pending						1	1	2.0	4
Respiratory syncytial virus	16				6	6	28	30.7	285
OTHER RNA VIRUSES									
HIV-1						4	4	2.3	47
Rotavirus	2			1	1	3	7	20.0	259
Norwalk agent					4		4	0.0	18
OTHER									
Chlamydia trachomatis not typed	8	16		5	5	43	77	69.3	989
Chlamydia psittaci					4		4	4.3	45
Mycoplasma pneumoniae					7	4	11	18.2	157
Coxiella burnetii (Q fever)					1	1	2	4.8	41
Rickettsia australis			1	1			2	0.0	8
Streptococcus group A					1		1	9.7	148
Bordetella pertussis					17	1	18	25.0	141
Bordetella species						1	1	2.7	129
Legionella longbeachae						1	1	0.3	9
Treponema pallidum	9					3	12	17.5	101
Entamoeba histolytica					1		1	0.5	10
Toxoplasma gondii				1			1	1.5	7
Schistosoma species					4	3	7	1.8	111
TOTAL	79	32	4	18	245	393	771	1,026.8	11,852

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 6. Virology and serology laboratory reports by clinical information for the reporting period 21 March to 3 April 1996

	Meningitis	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	TOTAL
MEASLES, MUMPS, RUBELLA										
Measles virus				1	1					2
Rubella virus					2				5	7
HEPATITIS VIRUSES										
Hepatitis A virus				3					4	7
Hepatitis B virus				2					8	10
ARBOVIRUSES										
Ross River virus					1		9		107	117
Barmah Forest virus									1	1
ADENOVIRUSES										
Adenovirus type 2		1								1
Adenovirus type 5		1								1
Adenovirus type 37						2				2
Adenovirus type 40			1						1	2
Adenovirus not typed/pending		8	13						3	24
HERPES VIRUSES										
Herpes simplex virus type 1		2			107	6		14	18	147
Herpes simplex virus type 2					73			39	7	119
Herpes simplex not typed/pending		1								1
Cytomegalovirus		6							21	27
Varicella-zoster virus					13				4	17
Epstein-Barr virus	1	16		1					20	38
Herpes virus group - not typed					3			1		4
OTHER DNA VIRUSES										
Parvovirus							1			1
PICORNA VIRUS FAMILY										
Coxsackievirus A9					1					1
Rhinovirus (all types)		13							1	14
Enterovirus not typed/pending	2	12	4		1				4	23
ORTHO/PARAMYXOVIRUSES										
Influenza A virus		4							1	5
Parainfluenza virus type 1		9								9
Parainfluenza virus type 2									1	1
Parainfluenza virus type 3		5							3	8
Parainfluenza virus typing pending		1								1
Respiratory syncytial virus		27							1	28
OTHER RNA VIRUSES										
HIV-1									4	4
Rotavirus			7							7
Norwalk agent			4							4

Table 6. Virology and serology laboratory reports by clinical information for the reporting period 21 March to 3 April 1996, continued

	Meningitis	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	TOTAL
OTHER										
Chlamydia trachomatis not typed								59	18	77
Chlamydia psittaci		2							2	4
Mycoplasma pneumoniae		8							3	11
Coxiella burnetii (Q fever)									2	2
Rickettsia australis									2	2
Streptococcus group A									1	1
Bordetella pertussis		18								18
Bordetella species		1								1
Legionella longbeachae									1	1
Treponema pallidum									12	12
Entamoeba histolytica									1	1
Toxoplasma gondii									1	1
Schistosoma species			1						6	7
TOTAL	3	135	30	7	202	8	10	113	263	771

Table 7. Virology and serology laboratory reports by contributing laboratories for the reporting period 21 March to 3 April 1996

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	South West Area Pathology Service, Liverpool	77
Tasmania	Northern Tasmanian Pathology Service, Launceston	3
	Royal Hobart Hospital, Hobart	14
Victoria	Microbiological Diagnostic Unit, University of Melbourne	5
	Monash Medical Centre, Melbourne	20
	Royal Children's Hospital, Melbourne	65
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	161
Western Australia	PathCentre Virology, Perth	241
	Princess Margaret Hospital, Perth	20
	Western Diagnostic Pathology	165
TOTAL		771