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AN OUTBREAK OF HEPATITIS A IN A CHILD-CARE CENTRE

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Abstract

In September 1995, the Brisbane North / Sunshine Coast Zonal Population Health Unit was advised of two cases of hepatitis A associated with a suburban child-care centre. Normal human immunoglobulin (NHIG) was recommended for all children and staff, and written advice on hygiene precautions was provided. A total of nine cases of hepatitis A, five clinical and four subclinical, were diagnosed over a nine-week period in persons associated with the centre. Reluctance by some general practitioners to the use of NHIG and the delay in convening a group meeting of parents to emphasise hygiene measures may have contributed to the outbreak.

Introduction

Hepatitis A infection may cause an illness characterised by fever, influenza-like symptoms, malaise fatigue, anorexia, nausea and abdominal discomfort, dark urine, pale faeces, jaundice and pruritus[1]. Infants and children often experience subclinical infection. The reported case-fatality rate is low (less than 1 per 1,000) but higher case-fatality rates have been reported among children under five years of age (1.5 per 1,000). For persons over 50 years of age, the case-fatality rate is more than 27 per 1,000[2].

In the third week of September 1995, the Zonal Population Health Unit was advised that a staff member and the parent of a child at a child-care centre located in a Brisbane suburb had been diagnosed with hepatitis A the previous day. The following week, a second staff member was similarly diagnosed.

The centre caters for children from six weeks to five years of age who attend from the local area and outer suburbs. Children attending the centre were segregated into four groups: baby, 'toddler', two year old, and kindergarten / preschool. There was minimal contact between these groups. There were 44 children and 13 staff attending the centre at the time of the outbreak.

The aim of the investigation conducted by the Zonal Population Health Unit was to detect all cases of hepatitis A associated with the outbreak, implement disease control measures, and assess parental acceptance of the intervention strategy.

Methods

The case definition used was 'a person associated with the centre who tested positive for hepatitis A IgM in the absence of recent vaccination and who developed jaundice or other clinical signs of hepatitis'.

The subclinical case definition was 'a person associated with the centre who tested positive for hepatitis A IgM in the absence of recent vaccination, and who did not develop any clinical symptoms'.

On the day of notification, the director of the centre was advised of the nature of the disease and the route of transmission. Simultaneously, the director was advised that all children attending the centre should be given NHIG[3]. A fact sheet about hepatitis A, emphasising strict hygiene measures was provided for all parents on the same day.

On receipt of the first notification, the Zonal Population Health Unit determined that no staff members had previously been vaccinated against hepatitis A. It was therefore recommended that all staff be offered NHIG and / or hepatitis A vaccine. The NHMRC recommends hepatitis A vaccine for child-care workers who care for children under two years of age[4]. At least two studies have indicated the effectiveness of hepatitis A vaccines in preventing hepatitis A infection in ongoing community outbreaks[5,6]. Its effectiveness for post-exposure prophylaxis is still to be determined.

A public health nurse visited the child-care centre to evaluate hygiene measures a few days after the cases were notified. The director was asked to advise the Zonal Population Health Unit of any subsequent cases.

All cases, and their general practitioners, were interviewed. Enquires were made about personal contacts and other possible cases. When a case was identified, family members were advised to receive NHIG. The outbreak was monitored and advice was provided to the staff, parents and their general practitioners.

A search was made of the Notification of Disease database of the Queensland Health Department, in order to determine whether any other cases associated with the centre had been notified. All cases of hepatitis A that had occurred recently in the Population Health Unit zone in which the child-care centre is located were traced. No related cases were found.

All parents of 'toddlers' (children between the ages of one and two years) were asked to have their children tested for hepatitis A when the outbreak was first detected. In mid-November, parents of the two year old children were advised to have their children tested when public health staff were advised that this group may have shared meals with the 'toddlers'. A total of 11 of the 15 children in these age groups were tested. A meeting attended by about 30 parents was conducted in mid-November to discuss the outbreak.

At the end of the outbreak, a questionnaire was sent to the parents of all children at the centre to determine their response to the advice they had received.

Table 1. The distribution of the clinical & subclinical cases

Category	Total number of persons in group	Numbers tested	Clinical cases	Subclinical cases
Babies	8	0	0	no known cases
Toddlers	5	5	0	2
Two year olds	10	6	0	1
Kindergarten / preschool	21	4	0	no known cases
Staff	13	unknown	2	no known cases
Parents / grandparents	unknown	unknown	3	1
Total			5	4

Results

The distribution of the clinical and subclinical cases among children, staff and parents is shown in Table 1.

Four subclinical cases were detected: two 'toddlers', the mother of a 'toddler', and a 2 year old. There was one family cluster associated with a 'toddler' ('toddler' A) who had a subclinical infection. 'Toddler' A's mother also had a subclinical infection and the father and grandfather had clinical infections.

The epidemic curve for clinical cases of hepatitis A is shown in Figure 1.

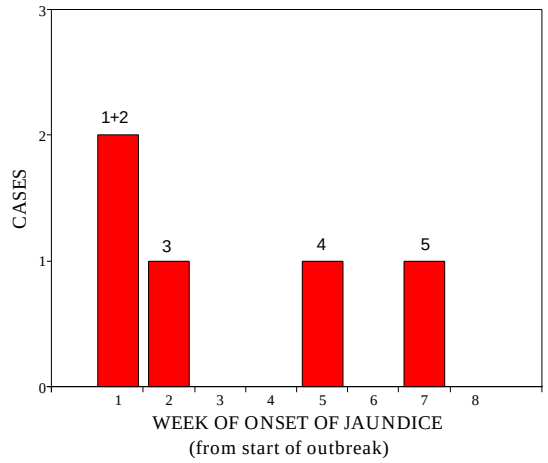
There have been no new cases identified since November 1995.

Of the 11 non-infected staff, five received NHIG and hepatitis A vaccine, four received NHIG only, and two had hepatitis A vaccine only.

Thirty-five responses to the parental questionnaire out of a possible 44 (80% response rate) were received. Of these, 23 indicated that their children had received NHIG, and 12 had not. Reasons given by the 12 parents whose children had not received the NHIG included: GP advised against it; worried about NHIG being a blood product; didn't want the child to have a painful injection; thought child was not a risk; and NHIG was too expensive. Several parents gave more than one reason.

Of those responding, 24 (69%) indicated that they had changed family hygiene practices as a result of the outbreak.

Figure 1. Clinical hepatitis A virus cases associated with the child-care centre, by week of onset of jaundice



Cases 1 and 2. Two adults (one parent and one staff member).
Case 3. One staff member.
Case 4. Grandfather of 'toddler' A.
Case 5. Father of 'toddler' A.

Discussion

Case finding was initially conducted in the 'toddler' group, as all adult cases were associated with them. This was extended to the two year old children when it was realised that staff, 'toddlers' and two year olds shared meal times. Active case finding was not conducted in the baby and kindergarten / preschool groups as investigators were confident of the segregation and infection control practices.

The first three clinical cases were all associated with the 'toddler' group and were considered to have been infected from the same source. Case one and case three were staff members at the centre and case two was a parent. We considered that the fourth and fifth clinical cases in weeks five and seven were secondary cases. It was not possible to determine the date of onset for the four subclinical cases or whether they were primary or secondary cases. It was considered that 'toddler A' may have been the index case.

The public health measures instituted included education and distribution of information, improved hygiene, and advising passive immunisation of all children and staff. In addition, there was active immunisation of staff and active case finding of subclinical cases, subsequent passive immunisation of family members. The public health nurse reported that the centre had fully implemented all the hygiene measures recommended by the Zonal Population Health Unit.

The NHMRC immunisation guidelines recommend hepatitis A immunisation for individuals who work in day-care centres, particularly in situations where the children are too young to be toilet trained[3]. The fact that two staff members had clinical infections supports the

need for implementation of this recommendation in all centres.

NHIG is recommended and has been shown to have proven benefit in preventing or modifying the severity of hepatitis A infection. It must be given within seven to ten days of exposure. The usefulness of NHIG depends on the identification of persons at risk within a few days of exposure.

Many of the parents of the children expressed concern about the use of NHIG, a human blood product, because of fears that it may transmit disease. Several general practitioners expressed similar concerns.

The public health physician discussed with the parents and their general practitioners the low level of risk of receiving NHIG compared with the risks of hepatitis A. As a result, many parents and their general practitioners indicated that the children would have the NHIG. From the questionnaire, it was apparent that 12 children did not receive NHIG.

These results support the findings that infants and children who do not show symptoms of hepatitis A may be a source of infection for others[7]. Most adults develop symptoms, sometimes requiring hospitalisation. To prevent further spread, early intervention may be required, particularly after the first case is identified.

On reflection, a general meeting with all parents should have been convened immediately after the initial cases were identified. Sixty-nine percent of parents reported changes in family hygiene practice as a result of the outbreak. This may indicate that the parents recognised that their current practices were inadequate to prevent transmission of this infection. Early active hygiene education for household members should play an important role in containing such an outbreak

Measures to be considered in the future include whether to offer NHIG to all children at the centre, and adults associated with them, as in other recently reported outbreaks[8,9]. Also closer liaison with general practitioners may overcome their reluctance to use NHIG in the prevention of hepatitis A transmission in child-care centres. Should staff at child-care centres be vaccinated against hepatitis A as part of their employment conditions?

In two subsequent notifications of hepatitis A associated with other child-care centres, extensive liaison with general practitioners regarding the use of NHIG, and early group meetings with parents, appear to have assisted in interrupting transmission in the early stages of the outbreak. In both centres, no secondary cases occurred within the incubation period for hepatitis A.

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CATERING ON THE MOVE - INVESTIGATING A TYPICAL OUTBREAK OF GASTROENTERITIS

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Abstract

An outbreak of diarrhoea affected at least 33 of the 58 persons who attended a baptism luncheon. Our investigation included site inspections, microbiological tests and food questionnaires. *Clostridium perfringens* intoxication probably caused the outbreak, the result of inadequate refrigeration of meat cooked the day before the gathering. To avoid this hazard, mobile or home caterers must have adequate facilities for refrigeration and transport of food. Established guidelines must be followed.

Introduction

Gastroenteritis may come to the attention of public health practitioners as isolated cases of food poisoning, as clusters of cases related to gatherings, and community-wide outbreaks. The public health response will depend on the clinical and public health importance of the incident or outbreak. Factors that influence this response include the number of cases, the seriousness of the illness, the setting of the outbreak, the implications of the outbreak for food safety and the food industry, and whether the outbreak continues or has ceased.

Action may range from an inspection of an implicated premise to an extensive microbiological and epidemiological investigation. Resources to undertake such investigations are limited. As days after an outbreak elapse, the quality of microbiological and epidemiological information decays. How much does an extensive investigation add to the information derived from incisive questions asked early in the outbreak? Does it alter the response to an outbreak? Does such information provide useful data that contribute to understanding, controlling and preventing epidemic and endemic enteric illness in the community?

The outbreak

Three days after a baptism celebration, the organiser of the gathering reported to his local council cases of gastroenteritis among attendees. The council environmental health officer contacted the Regional Public Health Unit and the Infectious Diseases Unit of Health and Community Services (now the Department of Human Services), Victoria.

Fifty-eight family members and friends had attended the luncheon in a suburban Melbourne hall. A friend of the host had prepared the food, which included meats cooked on a spit, vegetables and cakes. The following night, a substantial proportion of attendees developed diarrhoea. In conjunction with local and regional health authorities, we undertook an investigation of the outbreak.

Methods

Local environmental health officers investigated the preparation of the food. We obtained details about the food served, and interviewed attendees or their parents to establish details of illness and the foods they had consumed. We analysed data with Epi Info version 6.01 and calculated relative risks of illness for specific foods.

We sought specimens of leftover food, and faeces from those who were ill, and examined these for foodborne enteric pathogens by culture and *C. perfringens* enterotoxin by reverse passive latex agglutination[1,2]. We examined both food and faeces for *Salmonella* spp., *Staphylococcus aureus*, enteropathogenic *Escherichia coli*, *Bacillus cereus* and *C. perfringens*; and faeces for *Shigella* spp., *Aeromonas* spp., *Campylobacter* spp., *Listeria monocytogenes*, *Vibrio* spp. and *C. perfringens* enterotoxin.

Our case definition was 'a person who attended the gathering at the hall and who reported developing diarrhoea during the following four days'.

Results

Food eaten at the gathering was prepared by a person who formerly ran a catering business but who at the time of the outbreak only catered for friends, and functions at a sporting club. His operation was not registered as a food premise with the local council.

The caterer bought snack-foods, meat (pork and beef in approximately three-kilogram portions), fruit and cheesecake three days before the function. The meat and fruit were stored in a domestic refrigerator, and the cheesecake in a freezer at his home. He stated that on the afternoon before the function, he had thoroughly cooked the meat for about four and a half hours on a gas spit oven in his carport. He did not impale the meat, but instead had turned it by hand from time to time. He had wrapped the cooked meat in foil, let it cool in the carport for one and a half to two hours, and then returned it to the refrigerator. The next morning he had

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Table. Foods eaten by persons who attended the baptism luncheon

Food	Attack rate in persons who ate item (%)	Attack rate in persons who did not eat food (%)	Relative risk (95% confidence limits)
Snack foods	21 / 33 (64)	11 / 14 (79)	0.8 (0.6-1.2)
Beef	32 / 45 (71)	1 / 6 (17)	4.3 (0.7-25.8)
Pork	30 / 38 (79)	2 / 12 (17)	4.7 (1.3-17.0)
Gravy	29 / 42 (69)	4 / 9 (44)	1.6 (0.7-3.3)
Potatoes	33 / 49 (67)	0 / 2 (0)	undefined
Carrots	32 / 46 (70)	1 / 5 (20)	3.5 (0.6-20.3)
Peas	31 / 47 (66)	2 / 4 (50)	1.3 (0.5-3.6)
Cheesecake	19 / 30 (63)	14 / 21 (67)	1.0 (0.6-1.4)
Fruit salad	17 / 22 (77)	16 / 29 (55)	1.4 (0.9-2.1)
Whipped cream	15 / 19 (79)	18 / 32 (56)	1.4 (1.0-2.1)

transported the meat in an unrefrigerated foam container to the hall, about one hour's drive away. The meat was then refrigerated for about an hour before being carved. The cut meat lay unrefrigerated for about two hours before being served from 12.30 pm. The caterer prepared the rest of the food in the hall on the morning of the function.

The kitchen in the hall was clean, and contained a sink, hot water, ample preparation and storage space, and a refrigerator that maintained a temperature below 1°C two hours after being turned on.

We interviewed 51 (88%) attendees, comprising persons from 20 households. Thirty-three persons with diarrhoea met the case definition. These comprised 18 females and 15 males, range 1 to 78 years with a median age of 35 years. The incubation period was in the range 7 to 24 hours, median 14 hours. Eighty-eight percent (29 / 33) of cases were ill for 48 hours or less.

Common symptoms included abdominal cramps or aches (28 / 33, 85%) and nausea (17 / 33, 52%). Four persons (12%) had vomited. Five persons (15%) had bloody diarrhoea. There was no significant difference in the mean duration of illness in cases who reported bloody diarrhoea (41 hours) compared with those with non-bloody diarrhoea (36.4 hours, p=0.68).

The table describes the attack rate and relative risk associated with the consumption of various foods. Data from some attendees were incomplete.

Forty-six of 51 attendees ate some meat and most of these (37 / 46) ate both beef and pork. Because few people ate only one meat, we could not distinguish statistically the effect of eating either meat. None of the five persons who did not eat meat became ill. There appeared to be an increased risk associated with eating carrots, but since most of those who consumed carrots also ate meat, the effects could not be distinguished.

We collected six faecal specimens from cases, and isolated *C. perfringens* from five (heavy or moderate growth from four, light growth from one). *C. perfringens* enterotoxin was detected by latex agglutination in three of these five samples and in the sample from which *C. perfringens* was not isolated. *C. perfringens*, but not enterotoxin, was detected in faeces from the caterer who had had diarrhoea late in the evening after the function.

We tested one sample of beef and gravy and detected 3.1 coliform organisms / g, 1.6 x 10^3 colony forming units (CFU) *Bacillus cereus* / g, and 7.4 x 10^3 CFU *C. perfringens* / g.

Discussion

C. perfringens is the second-most common cause of outbreaks of food poisoning (after *Salmonella*) reported to the national outbreak surveillance scheme in England and Wales[3]. Its relative importance as a cause of Australian outbreaks is unknown. *C. perfringens* is common in the natural environment. It also appears in faeces of persons who showed no clinical signs[4]. Type A enterotoxin-producing strains cause diarrhoea after an incubation period of 8 to 24 hours[5]. *C. perfringens* as a cause of illness is suggested when more than 10^6 *C. perfringens* organisms / g are found in faeces, together with more than 10^5 organisms / g in food, with supporting clinical and epidemiologic evidence[6].

We performed semi-quantitative culture of *C. perfringens* in faeces, and detected only 7.4 x 10^3 organisms / g in the one available sample of meat served at the gathering. However, the following evidence suggests that this outbreak was caused by enterotoxin-producing *C. perfringens* acquired from meat at the gathering:

- the illness (acute self-limiting diarrhoea after a median incubation period of 14 hours) was compatible with *C. perfringens* intoxication[5];
- the flaw in food handling (slow cooling and inadequate refrigeration of cooked meat) is a recognised cause of *C. perfringens* food poisoning[6]. Cooked fresh vegetables are a less likely vehicle for this pathogen;
- eating pork and beef at the gathering were each associated with a four-fold increased risk of illness;
- *C. perfringens* and enterotoxin were isolated from the faeces of three of six cases;

- no other bacterial pathogens were identified in the faeces of cases;
- *C. perfringens* was isolated from meat (beef) served at the function.

The attack rate and relative risk associated with eating pork indicate that pork was probably a vehicle of infection. However, there was no leftover pork available for analysis. The presence of *C. perfringens* in the sample of beef may reflect contamination of the beef, cross contamination from pork, or both.

B. cereus was also recovered from the single food sample, but food poisoning caused by this organism is usually associated with detection of more than 10^5 *B. cereus* organisms per gram of food[6]. The clinical syndrome was consistent with the diarrhoeal form of *B. cereus* intoxication, but we did not detect the pathogen in faeces from cases[5].

Bloody diarrhoea is not a feature of *C. perfringens* intoxication, nor is it a feature of illness due to any of the common agents of gastroenteritis with short incubation periods[7]. These observations may have been misinterpretations by respondents or perhaps been due to blood from another source, such as haemorrhoids.

Two of the five persons who reported bloody stools provided specimens. Neither specimen was macroscopically bloody. *C. perfringens*, but no other enteric pathogen, was isolated from the faeces of both cases.

The costs and disabilities of enteric illness to affected persons and their families can be considerable, and include medical care and loss of work, education and leisure. Further costs fall upon operators implicated in outbreaks and include recall of food products, testing and destruction of food, insurance and advertising, loss of sales and legal costs[8]. Such events readily damage reputations. The operator involved in this outbreak abandoned catering altogether after this outbreak. The use of public health resources to systematically collect, collate, analyse and disseminate knowledge gained by investigations of outbreaks may prevent illness, similar outbreaks, and the costs associated with these events.

How did the various aspects of this investigation contribute to the public health outcome of this outbreak?

The key circumstantial features of this outbreak (diarrhoea after an incubation period of less than 24 hours, and leaving cooked meat unrefrigerated) suggested the possibility of *C. perfringens* intoxication.

Microbiological examination of faeces provided supportive evidence and excluded other common bacterial causes of food poisoning. Specimens from acute cases, and secondary cases, are crucial to this component of the investigation. Late collection of faeces may add expense rather than information, as the likelihood of isolating an outbreak-associated pathogen diminishes with time after illness.

Viruses spread person to person directly or through fomites (which may include prepared food) must always be considered when diarrhoea and vomiting occur one to two days after a gathering[9]. Secondary cases among persons not exposed to the original gathering are an important (almost diagnostic) clue. In this setting, virological examination of stools of acutely ill cases is particularly important.

Epidemiologic methods are often used to determine possible sources of infection. For one or two isolated cases of gastroenteritis, foods consumed in the 72 hours before illness are usually considered. When cases are apparently related to an event, the investigation usually concentrates on the food served at the main gathering, although other related functions should also be considered. In this outbreak, data from questionnaires supported the information obtained from early interviews and inspections, and from microbiological investigations.

Investigations of large gatherings are time consuming (and therefore expensive), and are subject to logistic and other difficulties. Data collection may be incomplete and biased towards affected persons, and food recall may be inaccurate unless the investigation is undertaken swiftly. Results may be inconclusive. However, in many outbreaks of gastroenteritis, the symptoms often do not point to a particular pathogen, faults in food handling may not be evident (or may be numerous!), and microbiological tests may be unhelpful. Food recall histories may be particularly useful in this setting.

In practice, the usefulness of an epidemiological analysis of food histories cannot be anticipated, and therefore this component of the investigation should be considered (and usually undertaken) as soon as possible after an outbreak is reported.

Furthermore, epidemiological analyses of food recall histories may play a key role in investigations of widely dispersed outbreaks of enteric illness - clusters which may only be identified by analysis of patterns in surveillance data. Low level microbiological contamination of foods, a relatively low attack rate, and unanticipated pathogens, make investigation of these clusters difficult[10]. In this setting, epidemiological analysis of food recall histories of cases may help identify the causative vehicle.

We propose that this particular outbreak was caused by *C. perfringens* intoxication resulting from slow cooling and inadequate refrigeration of cooked meat by a mobile or home caterer. Recently published guidelines draw attention to this particular hazard, and ways of avoiding it by ensuring cooked meat is rapidly cooled to less than 5^oC and kept adequately refrigerated until just before serving[11,12].

As national surveillance for outbreaks of enteric illness is implemented, investigating authorities should consider how they may most efficiently and accurately investigate outbreaks, both for surveillance purposes and to facilitate an appropriate response.

Acknowledgments

We thank local and regional environmental health officers for their involvement in this investigation.

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Tropical Influenza Surveillance

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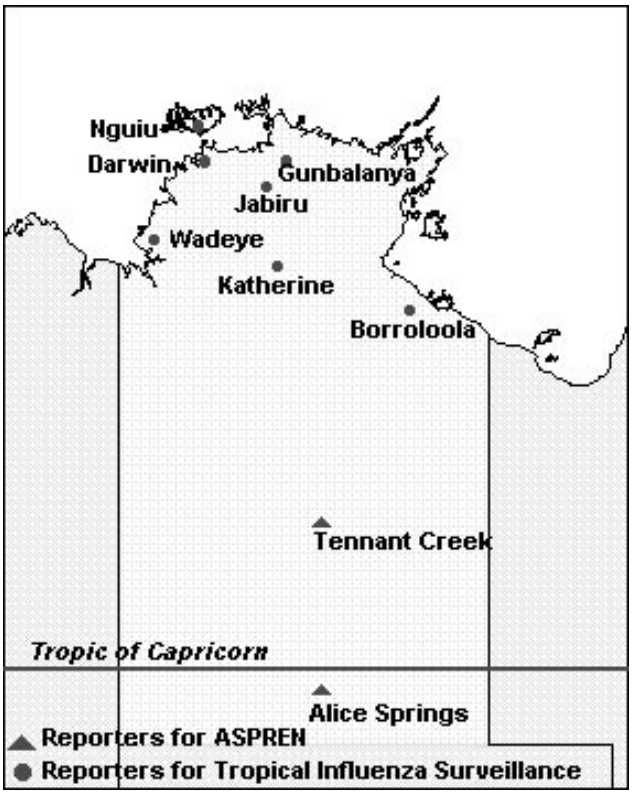
An outbreak of influenza A in the Top End of the Northern Territory three months prior to the usual winter season of influenza in Australia in 1995 highlighted the need for local surveillance. Influenza surveillance schemes in tropical countries such as Singapore and Thailand document a different seasonal pattern to the winter epidemics that are a feature of temperate areas. In tropical Australia, the pattern of influenza has not been documented.

Darwin Disease Control has established a scheme for reporting influenza-like illness through sentinel general practitioners (GPs). It was initially piloted with five GPs between June and November 1995. It commenced in January 1996 with the enrolment of 29 reporters located throughout the tropical Top End of the Northern Territory (Figure 1).

The scheme's three objectives are to:

- document the seasonal pattern of influenza activity and asses the appropriateness of current timing of influenza vaccine administration;
- enable early recognition of influenza outbreaks to facilitate a timely public health response; this includes the early collection of appropriate specimens to characterise the causative agent and to compare it with those included in the current vaccine formulation;
- compare the timing of influenza outbreaks in the Top End with outbreaks in the rest of Australia and to assess if the Top End is in a unique position to provide early information for each Australian influenza season.

Figure 1. Geographical distribution of sentinel general practitioners

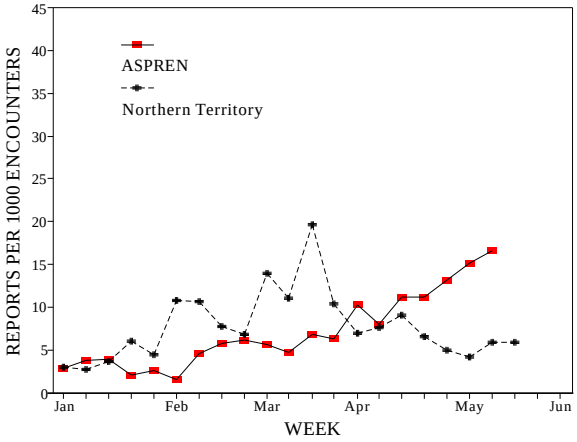


The clinical case definition for this scheme is the one used by the Australian Sentinel Practice Research Network (ASPREN), the only national sentinel practitioner surveillance scheme for influenza, so the data from both systems are directly comparable. Six of the following are required:

- sudden onset of symptoms (within 12 hours)
- cough
- fever
- rigor or chills
- prostration and weakness
- myalgia or widespread aches and pains
- no significant respiratory physical signs other than redness of the nasal mucosa and throat
- influenza in close contacts

Participating GPs report the number of patients seen each week who meet this case definition as well as their total number of consultations. These data are used to calculate the rate of influenza per thousand GP consultations per week in the Top End. In the event of an increase in influenza-like illness, arrangements are made through the emergency department of Royal Darwin Hospital and participating general practitioners to collect diagnostic specimens to identify the cause of the outbreak. So far this year, the scheme has documented a peak in March that was due to influenza A (Figure 2). Tropical Influenza Surveillance will be included in National Influenza Surveillance in future.

Figure 2. Sentinel general practitioner influenza surveillance, 1996, Northern Territory and ASPREN



OVERSEAS BRIEFS

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

Cholera

Malaysia: As of 24 May 1996, the Ministry of Health, Malaysia had reported an additional 133 confirmed cases of cholera to WHO. This has brought the total number of confirmed cholera cases in Malaysia to 1,222 since the outbreak began on 10 May 1996. The outbreak is mainly confined to Penang State.

The Ministry of Health, Malaysia has instituted preventive and control measures according to WHO guidelines. There have been no deaths so far. Visitors to the area should follow normal recommendations regarding food, water and hygiene to avoid risks of contracting the disease.

Meningococcal Infection

Balearic Isles, Spain: Two children who visited Mallorca in May have been reported to have died from suspected meningococcal infection. Included was a 13 year old from the United Kingdom (onset 19 May) and an 11 year old tourist from Germany (onset 21 May). Both children stayed in the same hotel.

Meningococcal infection has also been reported for two other tourists from the United Kingdom staying in different hotels. One was a 5 year old girl from whom *Neisseria meningitidis* group C was identified (onset 23 May). The second confirmed case was a 3 year old boy. Neither of these had a history of contact with each other or the two fatal cases.

Spanish authorities have instituted appropriate control measures in Mallorca. Visitors to Mallorca should seek medical advice immediately if symptoms, especially fever, appear.

United Kingdom: The Public Health Laboratory Service has commented that the current period of reports of meningococcal meningitis is one of the highest reported in the United Kingdom. The incidence has remained high during May. The majority of cases were diagnosed as meningococcal meningitis Group B. However there has been a shift towards more Group C and an upward shift in the age distribution of cases.

Lassa fever

Sierra Leone: As of 28 May 1996, ten additional suspected cases of Lassa fever and one death have been reported in Kenema. The WHO Collaborating Centre for Reference and Research on Special Pathogens at the Centers for Disease Control and Prevention, Atlanta, United States of America has confirmed Lassa fever in 9 of 20 specimens obtained from 12 earlier patients.

CORRESPONDENCE

Accelerated primary immunisation schedule

Dr Michael Sorokin, Traveller's Medical and Vaccination Centre Pty Ltd, 29 Gilbert Place Adelaide SA 5000

Since Burgess and Forrest, and Sloan quote the same reference to draw opposite conclusions about the incidence of pertussis in the United Kingdom, perhaps you could have some editorial comment in the next edition, or perhaps publish the paper by Miller, Vurdien and White if that is possible and permissible.

Reply from the author

Dr David Sloan, Central Queensland Public Health Unit, PO Box 946, Rockhampton Qld 4700

Thank you for your query about my recent letter in *Communicable Diseases Intelligence*[1].

A reader suggests that I draw opposite conclusions from a reference article to those drawn by M Burgess and J Forrest in an article in the same issue[2].

In my letter I used the reference article to support my comment that in the United Kingdom following the introduction of the accelerated schedule in 1990 'the incidence of pertussis fell[3]. Burgess and Forrest use the same reference in stating that the adoption of the accelerated schedule in the United Kingdom 'has not yet reduced the number of cases in children under the age of one year'. These are different types of conclusions and are not necessarily mutually exclusive.

My comment is supported on page R153 paragraph one and Figure 1 of the reference article.

The comment by Burgess and Forrest appears to be based on page 153, paragraph 4, 'the proportion of cases in children in cases aged 3 - 11 months has not declined (Figure 2)'.

The stability of the number and of the proportion of cases is not necessarily the same. In this situation, total number of pertussis cases are declining, and so while the proportion has not declined, the number of 3 - 11 month old cases is likely to have fallen.

To complicate matters, the proportion of cases in younger infants (under three months) has actually risen since 1990 (Figure 2 of the reference article). However, as the total uptake of immunisation increases and total cases fall, the proportion of cases from this group, who are too young for any or complete protection, is quite likely to rise.

Rather than dwell too much on the interpretations of this article, it may be more beneficial to recognise that it was written some while ago and note that an editorial in *CDI* has recently suggested that Australia should consider an accelerated schedule[4] (remembering as well the other benefits of higher uptake and lower rates of side effects). Perhaps the editor might consider inviting Dr Elizabeth Miller (et al.) to comment on the latest situation in the United Kingdom vis-a-vis pertussis.

References

1. Sloan D. Accelerated primary immunisation schedule [letter]. *Comm Dis Intell* 1996: 20;199.
2. Burgess M, Forrest J. Pertussis and the acellular vaccines. *Comm Dis Intell* 1996: 20; 192-196.
3. Miller E, Vurdien TE, White TM. The epidemiology of pertussis in England and Wales. *Comm Dis Rep* 1992: R152-164.
4. Hall R. Editorial: measuring immunisation coverage. *Comm Dis Intell* 1996: 20; 219.

Editorial comment

An update on the effect of the accelerated immunisation schedule on pertussis in England and Wales has since been published in *Communicable Disease Report* on 24 May 1996[1]. Dr Miller is a co-author.

In May 1990, an accelerated schedule was introduced in the United Kingdom with primary immunisation at two, three and four months of age rather than three, five and ten months of age as had previously been the case. There has been concern that the accelerated schedule might lead to inadequate immunity against pertussis in the preschool years and as a consequence could cause more disease in siblings under six months of age and, in particular, those under three months.

The report highlights a marked reduction in the incidence of pertussis in all age groups and a change in the age distribution of cases. The epidemic year of 1994 had the smallest number of cases so far recorded: only 3,964 were notified compared to 65,810 in the epidemic year of 1984. Improved vaccine coverage in preschool children led to a fall in the number of cases aged between six months and five years. Rates of infection for both this age group and those under six months remained extremely low. The rates in children aged two months and under, who are too young to be vaccinated, fell from 161 per 100,000 during the period 1985 to 1989 to 115 per 100,000 during 1990 to 1994.

Vaccine efficacy estimates of 96% were calculated for infants between six and 11 months of age and 93% for children aged one to four years. There was no significant decline in vaccine efficacy with age. The authors point to a number of factors that may have led to an over-estimation of vaccine efficacy and highlight the need for continued surveillance of immunity beyond five years of age.

The authors note a continuing decline in disease rates in all age groups, including those under six months of age and that the data suggests children no more than five years of age who were vaccinated under the accelerated schedule are not getting mild pertussis that could in turn be transmitted to young infants.

Reference

1. White JM, Fairley CK, Owen D *et al*. The effect of an accelerated immunisation schedule on pertussis in England and Wales. *Comm Dis Rep* 1996; 6:R86-90.

COMMUNICABLE DISEASES SURVEILLANCE

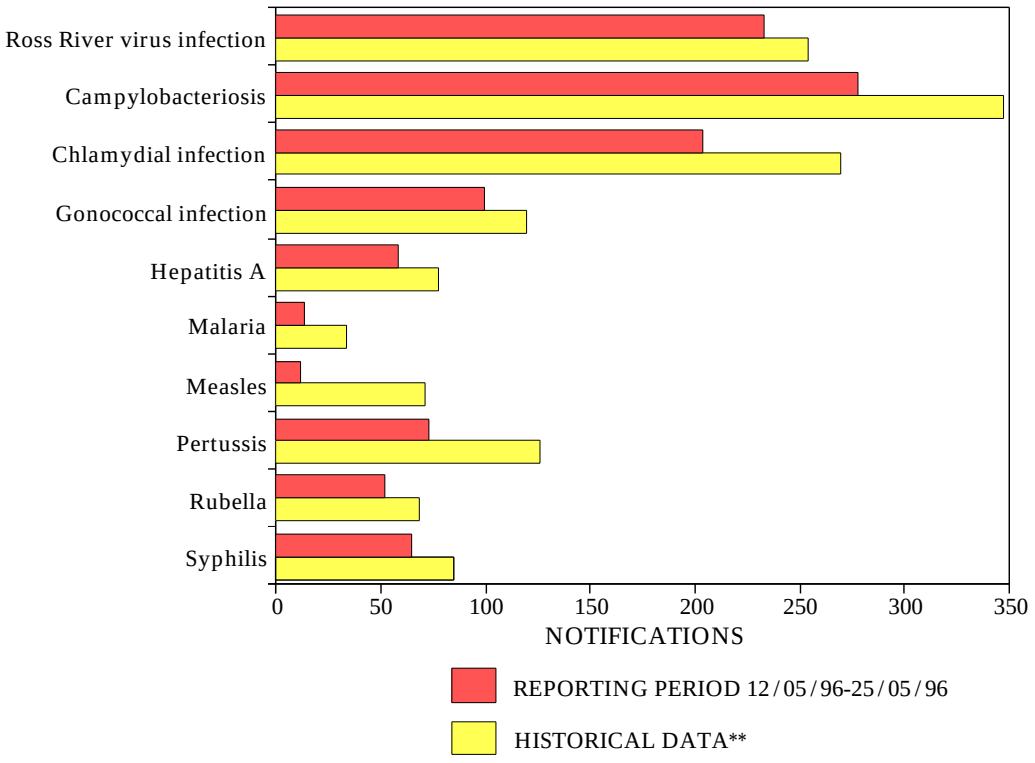
National Notifiable Diseases Surveillance System

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia-New Zealand. The system coordinates the national surveillance of 41 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislation. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information see *CDI* 1996; 20: 9-10.

There were 1,604 notifications received for this two week period (Tables 1, 2 and 3). The numbers of reports for selected diseases has been compared with averaged data for previous years (Figure 1). No reports were received from Victoria for the current period. This should be taken into account in interpreting the figure.

The recent epidemic of **Ross River virus infection** continues to decline (Figure 2). There were 233 reports received for the current period, the highest number of reports being from the Queensland Statistical Divisions of Brisbane (68) and Northern (38). The age group distribution for cases during the epidemic is similar to that observed over the last year and a half (Figure 3).

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.

Figure 2. Ross River virus infection notifications 1993 to 1996, by month of onset

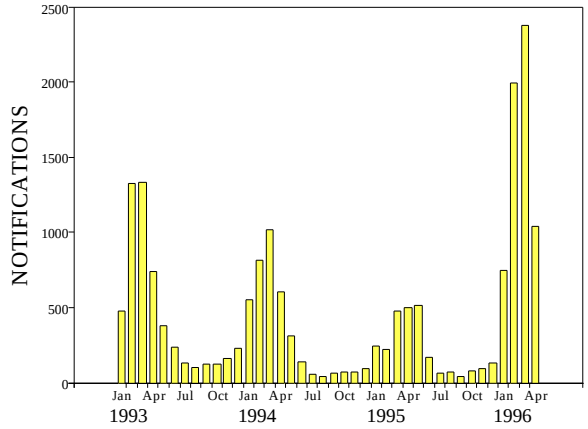


Figure 3. Ross River virus infection notifications 1995 and 1996, by age group and sex

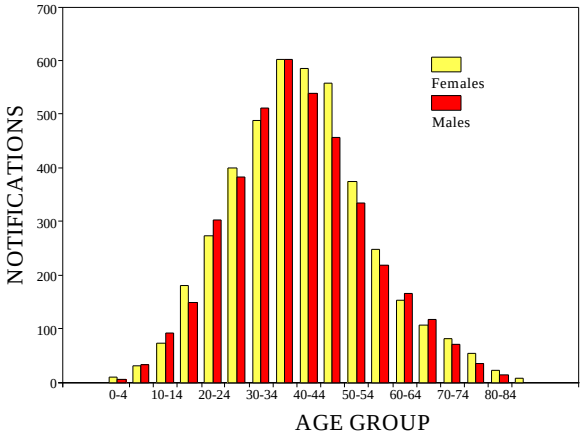


Figure 4. Campylobacteriosis notifications 1995 and 1996, by age group and sex

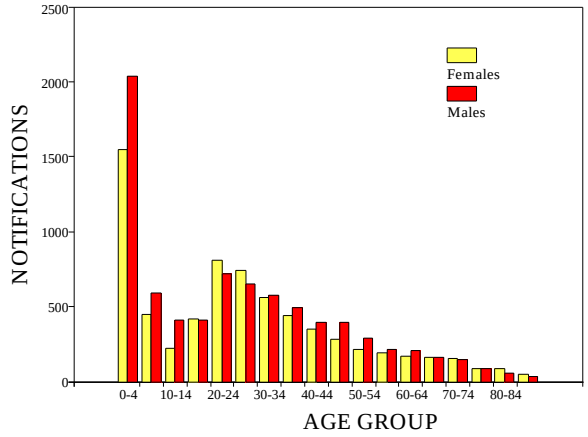


Figure 5. Hepatitis A notifications 1991 to 1996, by month of onset

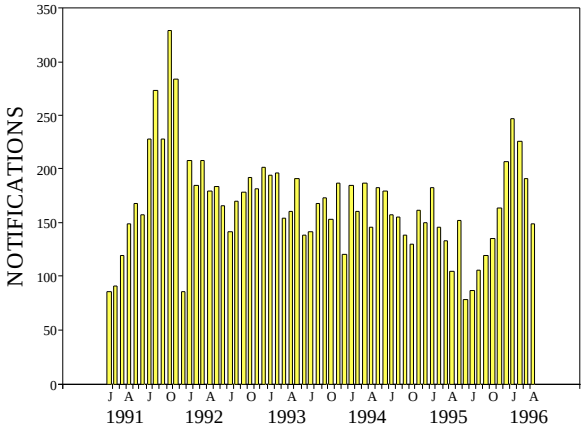


Figure 6. Measles notifications 1993 to 1996, by month of onset

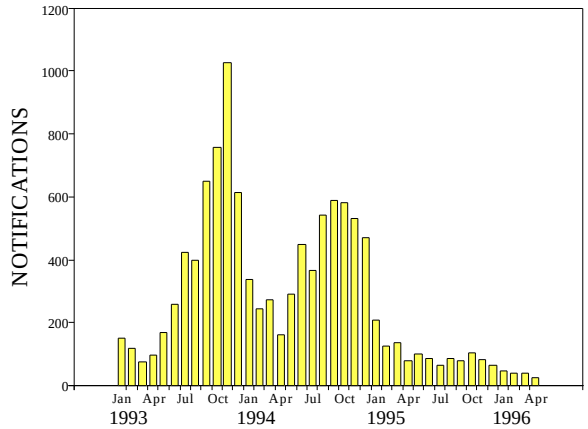
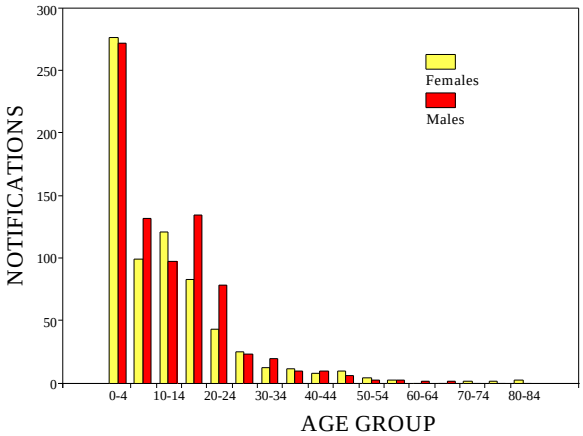


Figure 7. Measles notifications 1995 and 1996, by age group and sex



There were 278 reports of **campylobacteriosis** for the period, bringing the number received since January 1995 to 15,023 cases. The highest rate of notification was from South Australia. The predominant age group was 0-4 years. A smaller second peak in numbers of notifications was seen in young adults (Figure 4). This age group distribution is typical of patterns seen in other developed countries. In developing countries the majority of notifications are for children in the first two years of life. In these countries, most persons have numerous symptomatic campylobacter infections in early childhood, and there are few symptomatic infections in later life.

The number of reports of **hepatitis A**, 58 for the period, is returning to average levels (Figures 1 and 5). Twenty-eight cases (48% of the total) were reported for the age range 20-34 years (20 males and 8 females).

There were 12 reports of **measles** received during the period. The number of reports has remained low since the beginning of 1995 (Figure 6). During this period 48% of reported cases were in persons over the age of ten years (Figure 7); 11% of cases were in children under one year of age. The overall male:female ratio was 1.1:1; however for the age range 15 to 24 years the ratio was 1.7:1.

Since the end of the reporting period, a case of **cholera** (*V. cholerae* serogroup O1, biotype El Tor, serotype Ogawa) has been reported from Queensland. The case was a female who visited Penang, Malaysia during the epidemic period in May 1996.

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 12 to 25 May 1996

DISEASE	ACT	NSW	NT	Qld	SA	Tas	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
Haemophilus influenzae B infection	0	0	0	0	0	0	0	0	1	22	35
Measles	0	6	0	3	1	0	2	12	43	183	746
Mumps	0	0	0	NN	2	0	0	2	5	48	52
Pertussis	1	38	1	3	28	0	2	73	145	1114	1783
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0
Rubella	6	9	0	27	3	3	4	52	71	1156	1036
Tetanus	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 12 to 25 May 1996

DISEASE	ACT	NSW	NT	Qld	SA	Tas	WA	TOTALS FOR AUSTRALIA[1]			
								This period 1996	This period 1995	Year to date 1996	Year to date 1995
Arbovirus Infection (NEC)[3,4]	0	6	2	2	0	0	1	11	17	287	302
Barmah Forest virus infection	0	0	-	17	0	0	-	17	32	379	190
Ross River virus infection	1	30	6	177	0	-	19	233	304	6486	1590
Dengue	1	0	1	0	0	-	0	2	5	21	13
Campylobacteriosis[5]	10	-	12	78	90	13	75	278	373	4399	4207
Chlamydial infection (NEC)[6]	3	NN	24	127	0	13	37	204	261	2691	2581
Donovanosis	0	NN	3	1	NN	0	0	4	0	23	37
Gonococcal infection[7]	0	11	46	38	0	0	4	99	77	1408	1212
Hepatitis A	2	25	6	18	1	0	6	58	46	1003	703
Hepatitis B incident	0	0	1	1	0	0	1	3	9	93	147
Hepatitis B unspecified	4	0	0	32	0	4	13	53	68	601	707
Hepatitis C incident	0	0	0	-	0	-	-	0	10	8	39
Hepatitis C unspecified	16	NN	8	68	NN	3	37	132	329	3340	3278
Hepatitis (NEC)	0	0	0	0	0	0	NN	0	0	9	11
Legionellosis	0	4	0	2	0	0	0	6	7	75	90
Leptospirosis	0	4	0	5	0	0	0	9	8	102	52

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 12 to 25 May 1996, continued

DISEASE	ACT	NSW	NT	Qld	SA	Tas	WA	TOTALS FOR AUSTRALIA[1]			
								This period 1996	This period 1995	Year to date 1996	Year to date 1995
Listeriosis	0	0	0	0	0	0	0	0	0	23	34
Malaria	1	8	0	0	2	0	2	13	20	315	238
Meningococcal infection	0	3	0	2	1	0	1	7	11	100	122
Ornithosis	0	NN	0	0	0	0	0	0	3	35	63
Q fever	0	9	0	3	0	0	0	12	24	183	179
Salmonellosis (NEC)	1	32	35	100	17	3	17	205	214	2809	3253
Shigellosis[5]	0	-	14	10	0	0	2	26	25	269	360
Syphilis	0	33	12	19	0	0	1	65	73	590	794
Tuberculosis	0	3	2	8	8	0	1	22	35	429	467
Typhoid8	0	0	0	0	0	0	1	1	3	35	34
Yersiniosis (NEC)5	0	-	0	3	0	0	0	3	9	105	161

1. For HIV and AIDS, see Tables 4 and 5. 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.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 12 to 25 May 1996

DISEASES	Total this period	Reporting States or Territories	Year to date 1996
Botulism	0		0
Brucellosis	1	NSW	13
Chancroid	0		1
Cholera	0		2
Hydatid infection	1	WA	17
Leprosy	0		6
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.

HIV and AIDS Surveillance

Methodological note

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information.

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 December 1995, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
										This period 1995	This period 1994	Year to date 1995	Year to date 1994
HIV diagnoses	Female	0	4	0	0	0	0	0	0	4	9	72	81
	Male	1	27	0	1	1	0	14	2	46	59	744	854
	Sex not reported	0	0	0	0	0	0	0	0	0	0	9	9
	Total[1]	1	31	0	1	1	0	14	2	50	68	827	945
AIDS diagnoses	Female	0	0	0	1	0	0	0	0	1	2	27	41
	Male	0	17	0	1	0	0	5	0	23	67	619	864
	Total[1]	0	17	0	2	0	0	5	0	24	69	648	909
AIDS deaths	Female	0	0	0	0	0	0	1	0	1	2	36	36
	Male	1	12	1	5	5	0	10	0	34	59	556	676
	Total[1]	1	12	1	5	5	0	11	0	35	61	593	717

1. Persons whose sex was reported as transsexual are included in the totals.

Table 5. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 December 1995, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	15	547	4	94	44	4	157	69	934
	Male	167	9908	80	1549	557	70	3322	746	16399
	Sex not reported	0	2047	0	0	0	0	42	0	2089
	Total[1]	182	12509	84	1648	601	74	3530	817	19445
AIDS diagnoses	Female	5	130	0	28	18	2	47	17	247
	Male	71	3708	25	626	260	32	1307	270	6299
	Total[1]	76	3848	25	656	278	34	1361	289	6567
AIDS deaths	Female	2	96	0	21	13	2	32	9	175
	Male	50	2610	20	432	177	21	1024	199	4533
	Total[1]	52	2712	20	455	190	23	1062	209	4723

1. Persons whose sex was reported as transsexual are included in the totals.

More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly *Australian HIV Surveillance Report*, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 332 4648 Facsimile: (02) 332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for December 1995, as reported to 31 March 1996, are included in this issue of *CDI* (Tables 4 and 5).

National Influenza Surveillance

Australian Sentinel Practice Research Network; Communicable Diseases Intelligence Virology and Serology Reporting Scheme Contributing Laboratories, New South Wales Department of Health; Victorian Department of Health; World Health Organisation Collaborating Centre for Influenza Reference and Research.

National Influenza Surveillance is conducted from May to September each year. Data are combined from a number of sources to provide an indication of influenza activity. Included are sentinel general practitioner surveillance, absenteeism data from a national employer, and laboratory data from LabVISE and the World Health Organization Collaborating Centre for Influenza Reference and Research. For further information, see *CDI* 20 1996, pages 9-12.

Figure 8. Sentinel general practitioner influenza reports per 1,000 encounters 1996, by week

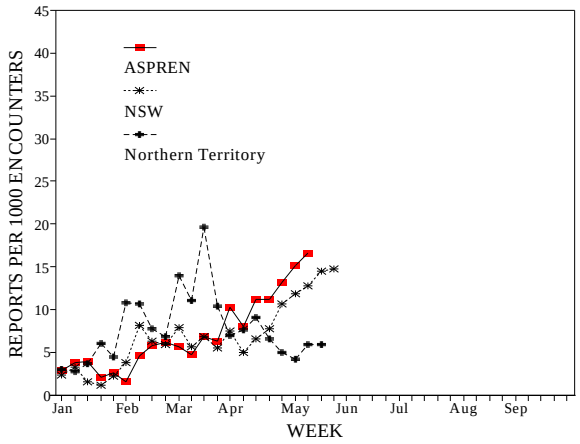


Figure 9. Australia Post absenteeism, 1996, by week

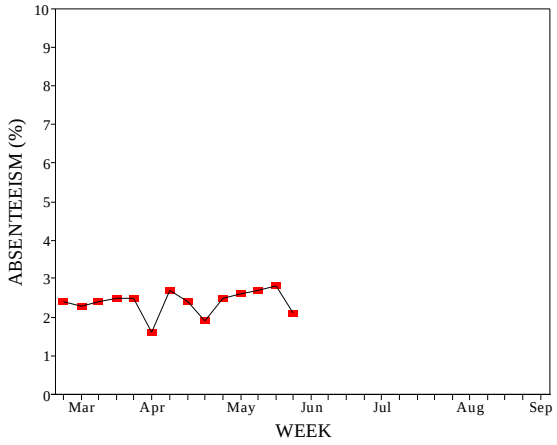


Figure 10. Influenza A laboratory reports, 1996, by method of diagnosis and week of specimen collection

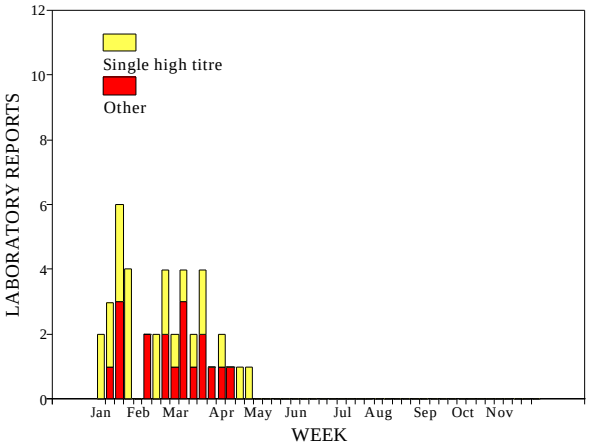
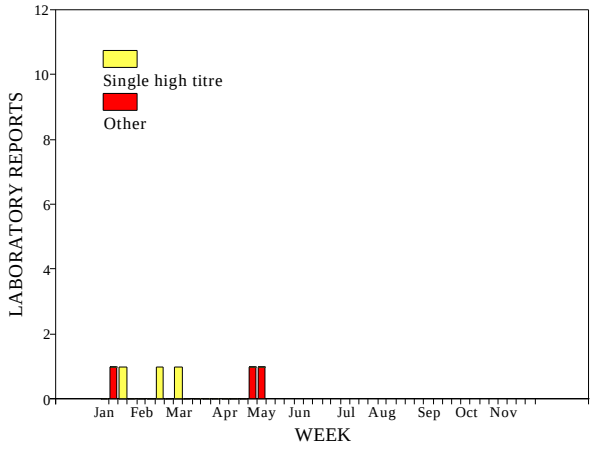


Figure 11. Influenza B laboratory reports, 1996, by method of diagnosis and week of specimen collection



The consultation rates for influenza like illness recorded by the ASPREN and New South Wales sentinel general practitioners schemes continues to rise (Figure 8). However that for the Northern Territory has declined after peaking in mid March. The absenteeism rate for a national employer remains stable (Figure 9). With respect to laboratory based surveillance low numbers of reports continue to be received (Figures 10 and 11).

The World Health Organization (WHO) Collaborating Centre For Influenza Reference Research, Melbourne has received few viruses for further identification so far this season. All of the influenza A strains have been of the H_3N_2 sub-type. Two of the four analysed were identical to the A / Johannesburg vaccine strain. A further two appear closer to A / Wuhan / 359 / 95, a variant which shows some antigenic drift from A / Johannesburg (serological studies at the Centre indicate that vaccines containing an A / Johannesburg-like strain produce good responses to the A / Wuhan virus).

The two influenza B isolates are antigenically close to the B / Beijing vaccine strain.

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) comprises 99 sentinel general practitioners from throughout the country. A total of approximately 9,000 consultations are recorded each week for 12 conditions. Of these, *CDI* reports the consultation rate for influenza, rubella, measles, chickenpox, pertussis and gastroenteritis. For further information including case definitions see *CDI* 1996;20:98-99.

Data for week 20 ending 19 May are included in this issue of *CDI* (Table 6). The rate of reporting of influenza-like illness has continued to rise to 16.6 per 1000 consultations for week 20 (Figure 8), the highest rate recorded by the scheme this year. The rate of reporting of pertussis continues at a low level, while that for gastroenteritis is also lower than in recent weeks.

Table 6. Australian Sentinel Practice Research Network, week 20, 1996

Condition	Week 20, to 19 May 1996	
	Reports	Rate per 1000 encounters
Influenza	137	16.6
Rubella	10	0.1
Measles	0	0
Chickenpox	17	2.1
Pertussis	5	0.6
Gastroenteritis	105	12.7

Australian Encephalitis: Sentinel Chicken Surveillance Programme serological results; March and April 1996

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

Sentinel chicken serology was carried out for 21 of the 22 flocks in Western Australia in March and April 1996. There were no seroconversions during this period.

Eight flocks of sentinel chickens from the Northern Territory were also tested in March and April. During this period there were a number of seroconversions to flaviviruses in the flock from Coastal Plains Research Station approximately 100 kilometres south-east of Darwin. In March, one chicken seroconverted to both Murray Valley encephalitis and Kunjin viruses and one to Murray Valley encephalitis. In April, two more chickens seroconverted to Murray Valley encephalitis.

There were no seroconversions to flaviviruses in the sentinel chicken flocks in Victoria during March and April 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. Pathcentre, Perth
6. Medical Entomology Branch, Department of Health and Community Services, Darwin, NT.

Virology and Serology Reporting Scheme

The Virology and Serology Reporting Scheme, LabVISE, is a sentinel reporting scheme. Twenty-three laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in *Communicable Diseases Intelligence* each fortnight. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see *CDI* 20 1996, pages 9-12.

There were 780 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 7 and 8).

Ross River virus was reported for 146 patients this fortnight all diagnosed by IgM detection. Seventy-seven percent of the patients were between the ages of 25 and 64 years. The number of reports has continued to drop since the peak in February (Figure 12).

Figure 12. Ross River virus laboratory reports, 1995 and 1996, by state and month of specimen collection

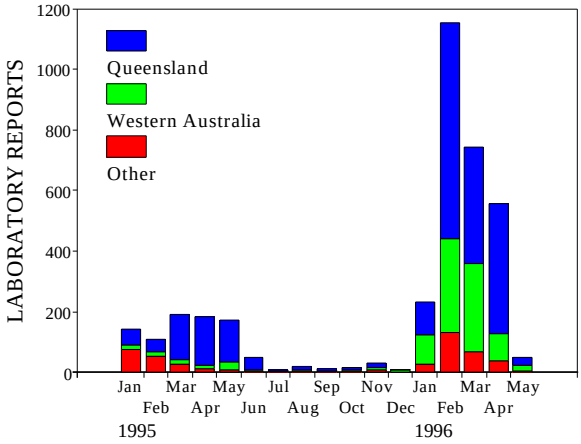
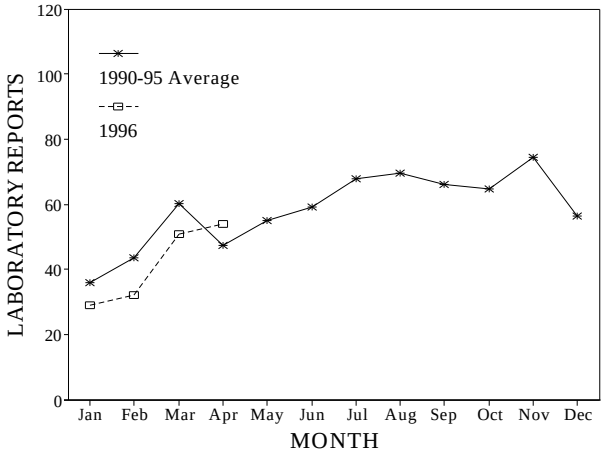


Figure 13. Rhinovirus laboratory reports, 1990 to 1995 average and 1996, by month of specimen collection



Parainfluenza virus type 1 was reported for 19 patients this period. Diagnosis was by antigen detection (17) and virus isolation (2). Eighteen patients were below the age of 4 years.

Repiratory syncytial virus was reported for 72 patients this period. Diagnosis was by antigen detection (48) and virus isolation (24). Reports came from New South Wales (35), Western Australia (25), Victoria (23), the Northern Territory (11) and South Australia (one). Sixty-nine of the patients (96%) were below 4 years of age.

Twenty two reports of **rhinovirus** were received this fortnight all being diagnosed by virus isolation. All age groups between 1 month and 75 years were represented. Reports have continued to increase in recent months which is usual for the time of year (Figure 13).

Rotavirus was reported for 80 patients this period all diagnosed by antigen detection. Seventy-nine of the reports were for patients below the age of 4 years. Reports are below average for the time of year (Figure 14).

Ten reports of **parvovirus** were received this fortnight. Diagnosis was by IgM detection (8) and nucleic acid detection (2). Reports came from Victoria (8) and Western Australia (2). For 1996, 26 patients (58%) have been between the ages of 25 and 44 years (Figure 15). Included were 21 females.

Figure 14. Rotavirus laboratory reports, 1990 to 1995 average and 1996, by month of specimen collection

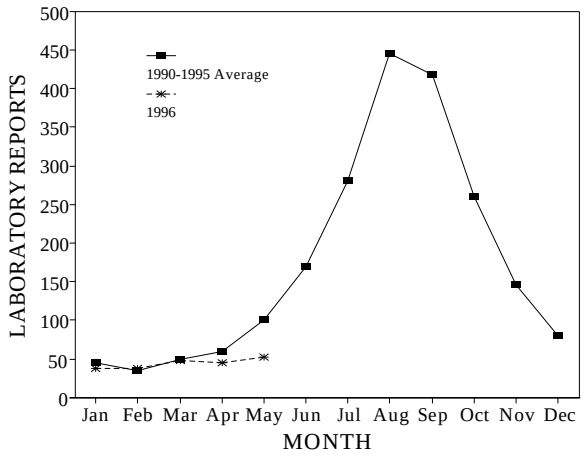


Figure 15. Parvovirus laboratory reports, 1996, by sex and age group

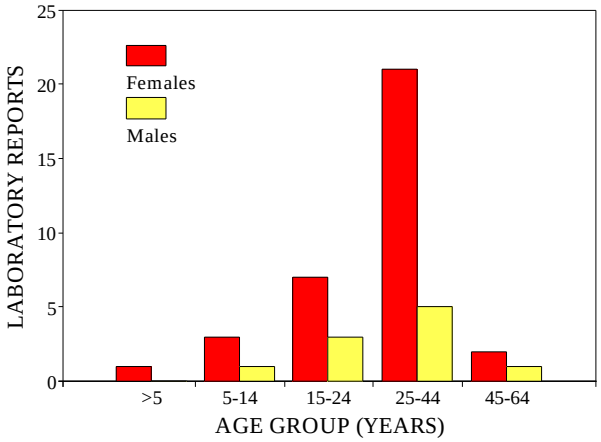


Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 16 to 29 May 1996, historical data[2], and total reports for the year

	State or Territory[1]							Total this fortnight	Historical data[2]	Total reported this year
	NSW	NT	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA										
Measles virus							1	1	18.7	28
Mumps virus							1	1	4.5	20
Rubella virus			1			2	2	5	13.2	261
HEPATITIS VIRUSES										
Hepatitis A virus		4				3	11	18	16.8	241
ARBOVIRUSES										
Ross River virus	1	3	113			1	28	146	101.7	2,779
Barmah Forest virus		1	2					3	15.7	128
Dengue not typed		1	1					2	.2	8
Kunjin virus							1	1	.0	5
ADENOVIRUSES										
Adenovirus type 1						1		1	1.7	9
Adenovirus type 3						1		1	3.2	56

Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 16 to 29 May 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	SA	Tas	Vic	WA			
Adenovirus type 37						1		1	.0	4
Adenovirus type 40							5	5	.0	16
Adenovirus not typed / pending	3		5			8	18	34	46.5	646
HERPES VIRUSES										
Cytomegalovirus	1		1		1	10	30	43	73.2	735
Varicella-zoster virus						10	5	15	46.3	578
Epstein-Barr virus		3	2			9	28	42	64.7	913
OTHER DNA VIRUSES										
Molluscum contagiosum							1	1	.2	2
Parvovirus	1					7	2	10	1.3	64
PICORNA VIRUS FAMILY										
Coxsackievirus A16						2		2	1.2	3
Poliovirus type 1 (uncharacterised)						1		1	3.3	7
Rhinovirus (all types)	6			1		9	6	22	34.7	291
Enterovirus not typed / pending						3	15	18	38.0	408
ORTHO / PARAMYXOVIRUSES										
Influenza A virus			2					2	24.8	80
Influenza B virus			1					1	5.5	26
Parainfluenza virus type 1						14	5	19	34.3	125
Parainfluenza virus type 2			1				3	4	9.8	36
Parainfluenza virus type 3						8		8	21.2	284
Parainfluenza virus typing pending						2	1	3	2.8	9
Respiratory syncytial virus	19	11		1		23	18	72	168.7	658
OTHER RNA VIRUSES										
Rotavirus	1	34		3		9	33	80	40.5	411
OTHER										
Chlamydia trachomatis not typed	2	83				10	58	153	89.3	1,665
Chlamydia psittaci						2		2	2.5	60
Chlamydia species			3					3	7.3	63
Mycoplasma pneumoniae			3			7	2	12	20.8	245
Coxiella burnetii (Q fever)						1		1	16.2	60
Rickettsia tsutsugamushi			1					1	.0	3
Rickettsia spp - other							1	1	.2	2
Bordetella pertussis						17	2	19	15.2	234
Legionella pneumophila			1					1	.0	5
Leptospira pomona			1					1	.0	1
Leptospira grippotyphosa	1		1					2	.0	2
Leptospira hardjo			6					6	.8	13
Leptospira australis			1					1	.2	4
Leptospira species			6					6	.8	25
Schistosoma species		1			1	6	1	9	2.2	160
TOTAL	35	141	152	5	2	167	278	780	948.0	11373

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 8. Virology and serology laboratory reports by contributing laboratories for the reporting period 16 to 29 May 1996

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Royal Alexandra Hospital for Children, Camperdown	27
	Royal Prince Alfred Hospital, Camperdown	5
Northern Territory	Alice Springs Hospital	49
Queensland	State Health Laboratory, Brisbane	153
Victoria	Microbiological Diagnostic Unit, University of Melbourne	4
	Monash Medical Centre, Melbourne	34
	Royal Children's Hospital, Melbourne	71
	Unipath Laboratories	6
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	57
Western Australia	PathCentre Virology, Perth	114
	Princess Margaret Hospital, Perth	65
	Royal Perth Hospital	21
	Western Diagnostic Pathology	174
TOTAL		780

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