An outbreak of norwalk virus gastroenteritis following consumption of oysters

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Abstract

In August 1996, an outbreak of Norwalk virus gastroenteritis occurred among south-east Queensland and northern New South Wales residents over a four week period. Ninety-two of the 97 cases detected were confirmed as having consumed raw oysters within three days prior to developing the illness. No other food items or beverages were significantly associated with the illness. Environmental investigations indicated the Terranora Broadwater, Tweed Heads as the origin of the contaminated oysters. However, the primary source of Norwalk virus could not be verified. Oysters and other shellfish appear to be a common vehicle for transmission of this virus. This outbreak and the more recent hepatitis A outbreak associated with Wallis Lake oysters, highlight the susceptibility of oysters to environmental contamination and the urgent need for stricter quality control procedures. This report details the epidemiological, microbiological and environmental findings from an outbreak investigation conducted jointly by the Queensland and New South Wales health authorities. *Comm Dis Intell* 1997;21:317-320.

Introduction

Norwalk viruses are an important cause of both sporadic and epidemic gastroenteritis[1]. Norwalk virus was first identified in 1972 following an outbreak of gastroenteritis in Norwalk, Ohio[2]. Other viruses with similar features were subsequently described and designated as Norwalk-like viruses[3,4]. These were named after the places they were isolated from, for example, Hawaii agent, Snow Mountain agent, etc. These small round structured viruses (SRSV) have subsequently been classified as members of the family *Caliciviridae*[5].

In late August 1996, South Coast Environmental Health Services, a branch of the Southern Public Health Unit Network (SPHUN, Queensland) received a number of complaints from persons who

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ISSN 0725-3141
Volume 21
Number 21
30 October 1997

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had developed gastroenteritis following the consumption of seafood, in particular oysters. At the same time, the North Coast Public Health Unit (NCPHU, Lismore, New South Wales) were notified of similar complaints. A co-ordinated public health response was initiated by both public health units with the SPHUN investigating cases who were Queensland residents and the NCPHU investigating cases who resided in New South Wales.

Methods

Information on study participants was collected by questionnaire. This information included clinical details, demographic characteristics and food histories from cases and controls. These questionnaires were administered by telephone interview by the NCPHU, and by personal interview or posted questionnaire by the SPHUN. A case was defined as a person who developed either vomiting or diarrhoea, or nausea and one other symptom (abdominal cramps, fever, chills, joint pain, or headache) within 72 hours after eating a meal at which raw oysters were served, or visiting a restaurant, a community club or function where seafood was served. Controls were defined as persons nominated by a case as having attended the same event as the case without developing any symptoms of gastrointestinal illness within 72 hours of the event. Cases were either patients who presented directly to the public health units, or persons nominated by a case as having attended the same function and were known to develop a similar illness.

Stool samples were collected from persons who met the case definition. The samples were submitted for faecal microscopy, bacterial culture, and viral studies including electron microscopy and immune electron microscopy. As many of the cases identified oysters as the most likely cause of their illness, samples of raw oyster were collected from both retail and wholesale distributors, and also submitted for bacterial and viral testing. Bacteriological testing was performed by the respective State health laboratories. Specimens for viral studies were sent to the Institute of Clinical Pathology and Medical Research (ICPMR), Westmead, New South Wales. Four faecal samples and a composite oyster sample were forwarded to the Department of Veterinary Pathology, University of Sydney for detection of Norwalk and other viruses by reverse transcriptase polymerase chain reaction (RT-PCR). No serological testing was performed.

Initial environmental investigations included ascertaining the area where oysters were harvested, testing the quality of water where oyster leases were located, and examining oyster purification plant operations. Subsequent investigations focused on the likelihood of raw sewage contamination through reticulation failures, checking the timing of tidal discharge of treated sewage, and exploring the possible influence of recent nearby residential development.

Epi-Info version 6.03 was used for the analysis of the data[6]. Crude odds ratios with 95% confidence intervals were calculated to estimate measures of association between exposures and illness, and two-tailed chi-square or Fisher exact tests were used for statistical significance testing.

Results

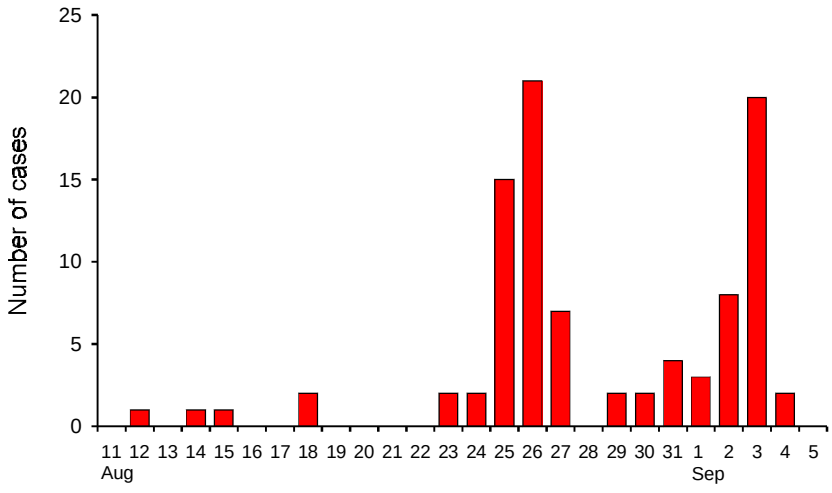
A total of 97 cases of gastroenteritis were identified by both public health units. Of the 97 cases, 93 (95.8%) responded to the questionnaires. There were 69 controls, with a response rate of 44.9%. The duration of the outbreak was approximately four weeks. The first reported date of onset of illness was 12 August (Day 1), and the last reported onset date was 4 September (Day 24) (Figure 1). Eighteen cases with onset dates on 25 or 26 August (Day 14 or Day 15) attended the same function. Approximately one week later, 24 cases with onset dates between 2 and 4 September (Day 22 and Day 24) were all guests of another large function.

Restaurants, sports and community clubs, and a private function were all involved in the outbreak. The consumption of raw oysters within three days prior to becoming ill was the common feature among 92 of the 93 cases. One case did not eat any seafood but admitted to handling raw oysters before consuming other food at a restaurant. The incubation period following consumption of oysters ranged from 5 to 60 hours (median 35 hours). However, 87% of cases had an incubation period of between 24 and 48 hours. Nausea and diarrhoea were the most common symptoms, followed by vomiting, stomach cramps, headache, fever/chills, and joint pain (Table 1). The duration of illness ranged from six hours to ten days (median 48 hours). The cases ages ranged from 13 to 83 years, with a male to female ratio of 1:1. Twenty-four cases (25.8%) consulted a medical

Table 1. Frequency of symptoms in cases

Symptoms	Per cent of cases
Nausea	95.7
Diarrhoea	90.2
Vomiting	80.4
Cramps	73.9
Headache	57.6
Fever/Chills	56.5
Joint pain	51.1

Figure 1 Number of cases of gastroenteritis following oyster consumption, by date of onset.



practitioner. There appeared to be no secondary cases among household contacts to suggest person to person spread. Two controls at one of the above functions ate oysters but did not develop any symptoms. All other controls did not consume oysters.

Analysis of the data showed a strong association between consumption of oysters and illness (odds ratio 1334, exact 95% CI 99-22026). No other food item or beverage listed on function or restaurant menus were significantly associated with illness. The number of oysters consumed by individuals was not consistently reported to enable the calculation of a dose-response effect.

Seven faecal samples, including two collected during the acute phase of illness, were submitted for laboratory analysis. All were negative for ova, cysts and parasites, and for bacterial pathogens including *Salmonella* spp., *Shigella* spp., *Campylobacter* spp., *Vibrio* spp., and *Yersinia* species. Faecal specimens examined by electron microscopy and immune electron microscopy failed to detect any virus particles. However, one of the four specimens subsequently tested by RT-PCR was positive for Norwalk virus (genotype 2). None of the above bacterial pathogens were detected in any of the raw oyster specimens. However, faecal coliform counts were elevated in some oyster samples (Australian Food Standards Code recommends a maximum *E. coli* level of 2.3/g). A composite oyster sample was positive for adenovirus by PCR. Norwalk virus was not detected in this oyster sample.

Environmental investigations indicated that the oysters associated with this food-poisoning outbreak were harvested from a common source, the Terranora Broadwater area at Tweed Heads. Water quality testing of numerous sampling sites near oyster harvest areas during a two week period showed faecal coliform counts varying between 4 per 100 ml and 340 per 100 ml (National Health and Medical Research Council, United States Food and Drug Authority and New South Wales Environmental Protection Agency guidelines recommend maximum faecal coliform levels of 14 per 100 ml)[7]. A number of possible sources of faecal contamination were identified during the investigation. These included leaking sewerage pipes, a leaking sub-marine sewerage pipe, 600 septic tanks in the residential development on one side of the Broadwater, and stormwater drain discharges. Any contamination would be exacerbated by the poor tidal exchange in the Broadwater area. This had previously been demonstrated with the use of dye testing by the New South Wales Environment Protection Agency (EPA) in 1985. The timing of tidal discharge (outgoing tide) by the sewage treatment plants was in accordance with regulations. Two out of five oyster purification plants on the Tweed River were currently in use and found to have unsatisfactory purification and sterilisation techniques, although the limitations of these in removing viruses is widely acknowledged.

Discussion

The findings from this investigation clearly implicated oysters as the likely vehicle of transmission for this outbreak. Although the wide confidence interval around the odds ratio indicates a small study sample, it is unlikely that any other food or beverage item would be causally associated with this illness given the strength of the association with oysters. Similarly, the poor questionnaire response rate of the controls and the different approaches to data collection by each public health unit may have introduced biases into the study. However, it is unlikely that these would change the findings of this investigation.

The number of cases related to this outbreak was probably considerably greater than the 97 cases identified, either because patients experienced only mild symptoms, or health authorities were not informed. However, the management of media releases assisted in the identification of unknown cases. The bimodal epidemic curve reflects two separate large functions held on consecutive weekends. Of the 94 persons known to eat oysters, all but two developed gastroenteritis. In contrast, only one out of 30 persons who were known not to consume oysters developed the illness. It is possible that the two subjects who did not become ill ate uncontaminated oysters, or both may have had asymptomatic infection.

The aetiological agent responsible for gastroenteritis appears to be Norwalk virus. This is a small round structured RNA virus (SRSV) which has previously been implicated as the aetiological agent in a number of acute non-bacterial gastroenteritis outbreaks[8-11]. Oysters and other shellfish appear to be a common vehicle for transmission of this virus. Oysters have also been shown to transmit bacterial and other viral pathogens including *Shigella* spp., *Salmonella* spp., *Vibrio* spp., and hepatitis A[10, 12-14].

The incubation period and clinical symptoms of cases were consistent with a Norwalk-like infection, according to 'Kaplan's criteria'[15]. Kaplan's criteria include; stools negative for bacterial pathogens, greater than 50% of cases with vomiting, a mean or median incubation period of 24 - 48 hours, and a mean or median duration of illness of between 12 - 60 hours. These criteria have been shown to have a relatively high specificity and sensitivity for a Norwalk-like infection during outbreaks of gastroenteritis. Although the gastroenteritis is usually self-limiting and not life-threatening, the likelihood of more severe disease may be increased for immunocompromised persons and the elderly.

Norwalk virus was not detected in seven faecal specimens tested by electron microscopy. However, the sensitivity of electron microscopy is limited if faecal specimens for viral examination are not collected within 48 hours of onset of illness. The recent development of RT-PCR as a diagnostic tool has improved detection rates, and the sensitivity of this test enables faecal specimens to be submitted up to seven days after the onset of illness[16]. The cost and time required to process specimens for Norwalk virus PCR testing currently limits its use as a routine diagnostic tool. The application of Kaplan's criteria as a screening tool would be helpful in determining whether faecal specimens should be sent for molecular diagnostic testing during outbreaks, particularly if there is a delay in the collection time of specimens rendering them unsuitable for electron microscopy.

Serological testing was not requested for any of the cases involved in this outbreak. Enzyme immunoassay (EIA) and radioimmunoassay (RIA) techniques have been used for the detection of Norwalk virus during foodborne outbreaks[10,17]. Serological testing should become more widely employed during suspected Norwalk virus outbreaks. This would complement faecal diagnostic testing,

and also address the difficulty in detecting Norwalk virus in stools, and the poor compliance with submitting stool specimens during gastroenteritis outbreaks.

The elevated faecal coliform counts in some of the oyster samples, and the identification of adenovirus in a composite oyster sample, indicate probable sewage contamination. These findings are supported by the results of the water quality testing. Despite these findings, improved indicators of viral contamination of both water and oysters are needed since faecal coliform levels often correlate poorly with the presence of viruses[18]. Environmental investigations identified a number of possible sources of sewage contamination. Although the oyster industry is not responsible for sewage pollution, these events highlight the susceptibility of the oyster industry to environmental factors and the need for the industry to implement strict quality control procedures. Quality testing should include continual environmental monitoring of both water and oysters, and increased laboratory testing during and after pollution incidents. In addition, oyster purification and processing plants need to be monitored regularly by food inspection services. The recent outbreak of hepatitis A (over 400 cases) associated with the consumption of raw oysters from the Wallis Lake area in New South Wales highlights the urgency of these requirements.

Terranora oysters were removed from retail and wholesale outlets following strong evidence that oysters were the most likely vehicle of transmission during the outbreak. No other cases were detected following this intervention. Harvesting of oysters was prohibited from the river for several months, until water quality met recommended standards and a comprehensive ongoing quality assurance program was in place. This investigation also highlighted the shortcomings of existing regulations designed to facilitate tracking of oysters from the consumer or retailer back to purification batches, oyster leases, or harvest dates. It was noted that record-keeping was inadequate and that mixing of oysters from different sources was widespread practice. This issue needs to be closely examined by government agencies and the oyster industry to enhance recall effectiveness while minimising the economic implications of the recall.

This outbreak of gastroenteritis has raised a number of significant issues regarding the oyster industry and public health safety. Effective quality assurance programs and more extensive collaboration and communication between oyster producers and local/State government agencies are urgently required.

Acknowledgements

Staff of the Institute of Clinical Pathology and Medical Research (ICPMR), Westmead, NSW.

Dr G. Grohmann, Department of Veterinary Pathology, University of Sydney, NSW.

Dr J. Bates and staff of Public Health Microbiology, Queensland Health Pathology and Scientific Services.

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Commentary

An outbreak of Norwalk virus gastroenteritis following consumption of oysters

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This report of an outbreak of gastroenteritis associated with oyster consumption is an important reminder of the risks associated with consumption of raw or undercooked foods of animal origin, and the risks associated with the contamination of oyster harvesting areas with human sewage.

The authors should be congratulated for their cross-border collaboration in investigation of the outbreak and for their joint report. In the absence of a national outbreak database, such reports provide the basis for developing food safety policy and programs.

While the only laboratory evidence for Norwalk virus infection was a positive PCR result from a single stool, the symptom profile, incubation period, and duration of illness are entirely consistent with previous outbreaks of Norwalk virus infection[1]. In similar outbreaks where the symptoms, incubation period, and duration of illness suggest a viral aetiology, RT-PCR, as used in this investigation could be increasingly utilized. This technique is estimated to detect as little as 10 to 1,000 Norwalk virus particles per millilitre of stool[2] compared to a lower level of detection of 10^5 to10^6 particles per millilitre of stool by electron microscopy[3,4].

The authors acknowledge that the need to carry out a rapid field investigation may have introduced some biases. However, it is inconceivable that any plausible bias could account for such a strong association with oyster consumption. Alternative methods of analysis could have been employed to remove some of the biases - such as analysing all attendees at the two large functions that had 18 and 24 cases respectively. Instead they chose to use a nested case-control or case-cohort style methodology with a convenience sample of controls from various restaurants and functions. The fact that the prevalence of vomiting and diarrhoea were in the upper range of that reported in previous reviews, and the rate of medical consultation was so high, is probably due to more severe cases contacting the health departments. This bias could have been addressed through studying all well and ill persons in well defined cohorts, or random sampling of cases and/or controls within large well defined cohorts.

The subsequent outbreak of hepatitis A associated with oysters from Wallis Lake has focused consumer attention on the safety of oyster consumption[5]. Oyster associated outbreaks are likely to be increasingly recognised for the following reasons:

1. Many estuaries are subject to increasing urban development with associated overflows from sewage treatment plants, septic systems and storm water discharges. It may take years to provide the infrastructure required to protect these waterways.

2. Consumers are increasingly concerned about food safety[6] and may be more likely to report illness.

3. Health agencies are using more advanced epidemiological and laboratory investigation methods (e.g. PCR) that will increase the likelihood of similar outbreaks being detected.

In order to protect the health of oyster consumers viral monitoring of harvest areas should be introduced as a research program. However, this is a relatively new methodology requiring time to learn how to interpret the results, and therefore should not be considered a panacea. In this outbreak it was interesting to note that an adenovirus was detected in oyster material, but no Norwalk virus was detected. However, faecal coliforms were above the recommended level, and as an interim measure, compliance with existing guidelines should be a priority.

The New South Wales Health Department has previously recommended consumers should be made aware of the risks associated with the consumption of raw seafood. In particular, persons at increased risk of death due to oyster associated infections should be aware of that risk[7]. This includes people with liver disease who are at risk of complications due to *Vibrio vulnificus* and *V. parahaemolyticus,* and hepatitis A infection (*Vibrio* are not associated with sewage contamination). In addition, persons with immune-compromising conditions such as cancer and AIDS, and the elderly, are at increased risk of fulminant infections associated with raw oyster consumption. It would be worthwhile evaluating to what extent these high risk groups are aware of these warnings. In the state of Florida in the United States of America the following notice is required at all points of raw oyster sale:

Consumer information: There is risk associated with consuming raw oysters. If you have chronic illness of the liver, stomach, or blood or have immune disorders, you are at greater risk of serious illness from raw oysters and should eat oysters fully cooked. If unsure of your risk, consult a physician.

Some groups object to such warnings and hold the view that food is either ‘safe for everyone’ or ‘not safe enough for anyone’. This is based on the premise that food is either ‘safe’ or ‘unsafe’ without qualification. Perhaps consumers are too sophisticated to be given blanket reassurances of safety, and now expect agencies to provide them with information that allows them to come to their own conclusions. It may benefit industry if those at greatest risk of disease are not consuming higher risk food products, and may lessen public outrage if consumers suffer illness after making an informed decision to eat a higher risk food.

Such selective warnings are not without precedent in Australia. The Australian New Zealand Food Authority, and many State health departments issue brochures for pregnant women advising of the dangers of eating foods associated with listeriosis, such as pate and soft cheeses. The New South Wales Health Department has recently issued warnings advising against the consumption of undercooked hamburger mince. It is consistent to give similar advice in relation to raw seafood.

While not providing a guarantee, there is evidence that cooking oysters can lessen the risk of illness, and specific information on cooking methods, times, and temperatures should be provided to consumers[8,9].

In summary, we are very likely to see similar reports in the future. It will take many years to provide the infrastructure required to mitigate the effect of urban development on the many vulnerable oyster harvesting areas, and it will take time to validate viral monitoring programs. In the interim, compliance with existing water quality guidelines and consumer and patient education efforts, may be the best way to protect public health and the oyster industry.

The views expressed in this commentary are those of the author and do not necessarily represent official New South Wales Health Department policy.

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Meningococcal disease in New South Wales

In early October two separate outbreaks of meningococcal disease (serogroup C) were reported to the New South Wales Health Department. The first of these involved two university students who had attended the same intervarsity sporting event in New South Wales. One student, from Western Australia, died. In the second outbreak three cases were reported at a high school.

It is usual to observe an increase in the number of reports of meningococcal disease at this time of year[1] . Meningococcal meningitis is caused by the bacterium, *Neisseria meningitidis*. This organism is common in the community and exists harmlessly in the throats of many adults and children. It is spread by respiratory droplets from the nose and throat of an infected person. In a small proportion of individuals infection progresses to an acute invasive disease. Symptoms include high temperature, fever, sore neck, headache, vomiting, rash and joint pain. Treatment is successful in the majority of cases if administered promptly. In cases of suspected meningococcal disease benzylpenicillin is the drug of choice. Where other causes of bacterial meningitis could be involved ceftriaxone should be used where available[2].

The National Health and Medical Research Council recommends rifampicin chemoprophylaxis for contacts of a case of invasive meningococcal infection[2,3]. Vaccination is only recommended in special circumstances. Most cases of disease in Australia are due to serogroup B, for which no effective vaccine is available[3,4]. In the case of an outbreak due to a vaccine preventable serogroup the National Health and Medical Research Council recommends that a vaccination program should be considered if the population at risk can be clearly identified, such as in a day-care centre, school or university. Routine vaccination is not recommeded as the risk of meningococcal disease in Australia is low.

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Communicable Diseases Surveillance

Q fever

Q fever is a zoonotic disease caused by the rickettsia, *Coxiella burnetii*. The disease in humans has an incubation period ranging from two to four weeks and is most often characterised by an acute onset of chills, fever, sweating, headache, malaise and myalgia, and less commonly cough, nausea, vomiting and arthralgia. Transient mild rash occurs in a minority of cases for two to five days. Serious chronic complications are uncommon, although a relapsing debilitating syndrome may occur.

Inapparent infection with this organism occurs in a wide range of wild and domestic animals. Infection in wild animals is largely maintained by vectors (especially ticks) that also provide a source of infection for domestic animals. By contrast with other rickettsiae, infection in humans is usually acquired by inhalation, rather than by contact with infected vectors. Transmission most often occurs via inhalation of aerosols or dust contaminated with *C. burnetii* from infected ruminants. This frequently occurs in or near abattoirs or establishments handling animal by-products, and also by contact with infected animals and contaminated articles such as straw, wool, hair and hides. Products of conception, particularly the placenta, are the most potent source of *C. burnetii*, with milk and faeces of minor importance. Direct transmission from person-to-person rarely occurs.

In Australia, Q fever is essentially an occupational disease. This is reflected by the age - sex distribution of notifications for 1996 (Figure 1), with 62% of cases being reported in males aged 20 to 44 years. Cases of Q fever occur throughout the year with no apparent seasonal distribution (Figure 2). The majority of notifications of Q fever are reported from Queensland and New South Wales, and these two States represented 83% to 93% of the total reports for the years 1992 to 1996.

Preventative measures for Q fever include educating the public on the sources of infection and appropriate disinfection procedures. Other important measures include disposal of animal products of conception, pasteurisation of milk, the use of special isolation rooms within abattoirs for the incision of gravid uteri, and vaccination of occupationally exposed people such as abattoir workers, veterinarians, and laboratory personnel.

Figure 1. Q fever notifications, 1996, by age group and sex

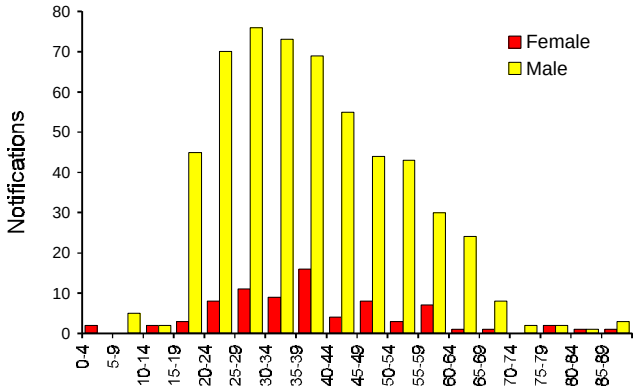
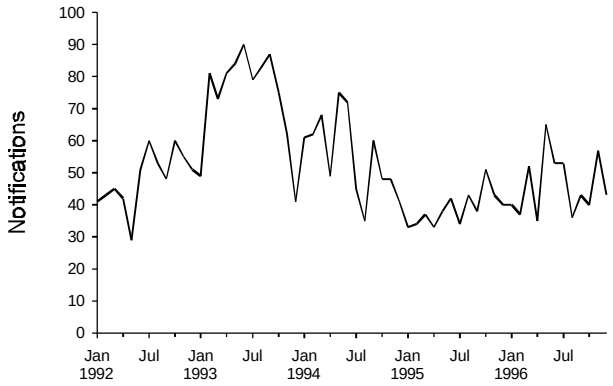


Figure 2. Q fever notifications, 1992 to 1996, by month of onset



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 more than 40 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 legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1997;21:5.

Reporting period 17 September to 14 October 1997

There were 4103 notifications received for this four week period (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 3).

The number of reports for hepatitis A is 35% higher than for the previous four-week period, but remains low by comparison with numbers reported earlier in the year. Most of the current notifications were received from New South Wales and Queensland.

There were 34 notifications of meningococcal infection for the current period, bringing the total number of cases for the year so far to 378. This represents 50 more notifications than for the corresponding year to date number last year. Cases have been reported during the last month from all jurisdictions except the Australian Capital Territory and Tasmania. In each of the last 6 years, the number of reports of cases has declined after October. The number of notifications received for measles has increased markedly in

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 September to 14 October 1997

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type B infection	0	0	0	3	0	0	0	0	3	2	41	47
Measles	15	6	2	61	1	0	16	1	102	39	535	374
Mumps	2	1	1	2	6	0	3	1	16	6	161	97
Pertussis	11	174	3	244	90	6	107	105	740	287	6468	2326
Rubella	1	0	0	64	31	5	62	3	166	214	1085	2014
Tetanus	0	0	0	0	0	0	0	0	0	0	7	1

NN. Not Notifiable

1. No notifications of poliomyelitis have been reported since 1986.
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.

Table 2. Notifications of other diseases received by State and Territory health authorities in the period 17 September to 14 October 1997

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
Arbovirus infection (NEC)[3]	0	1	4	0	0	0	1	0	6	2	114	43
Barmah Forest virus infection	0	6	-	17	0	0	3	-	29	37	563	752
Campylobacteriosis[4]	14	-	15	314	132	30	244	72	821	810	8956	9218
Chlamydial infection (NEC)[5]	5	NN	43	191	0	17	78	105	439	530	6308	6554
Dengue	0	1	0	0	0	0	0	0	1	1	196	30
Donovanosis	0	NN	1	0	NN	0	0	1	2	0	26	37
Gonococcal infection[6]	5	38	73	56	0	0	6	74	252	271	3457	3270
Hepatitis A	2	34	4	85	9	0	15	6	155	109	2498	1815
Hepatitis B incident	0	1	2	2	0	0	12	0	17	25	196	186
Hepatitis C incident	0	0	0	-	0	0	-	-	0	6	12	49
Hepatitis C unspecified	25	NN	15	254	NN	39	276	31	640	608	7531	7599
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	1	14	15
Legionellosis	0	1	0	0	3	0	2	0	6	12	116	149
Leptospirosis	0	2	0	2	0	0	1	0	5	8	96	176
Listeriosis	0	2	0	1	1	0	0	0	4	14	63	55
Malaria	0	5	0	39	1	0	6	3	54	40	663	688
Meningococcal infection	0	9	2	6	5	0	8	4	34	41	378	328
Ornithosis	0	NN	0	0	0	0	2	0	2	2	41	64
Q Fever	0	13	0	26	0	0	4	0	43	32	470	431
Ross River virus infection	0	13	3	19	0	0	2	6	43	43	6396	7512
Salmonellosis (NEC)	3	71	19	92	23	8	63	23	302	265	5502	4517
Shigellosis[4]	1	-	16	14	6	0	8	5	50	36	644	526
Syphilis	1	28	26	28	0	1	0	2	86	75	955	1214
Tuberculosis	2	16	6	8	8	3	16	6	65	95	756	846
Typhoid[7]	0	0	0	0	0	0	2	0	2	7	55	74
Yersiniosis (NEC)[4]	1	-	1	6	1	0	0	0	9	19	199	201

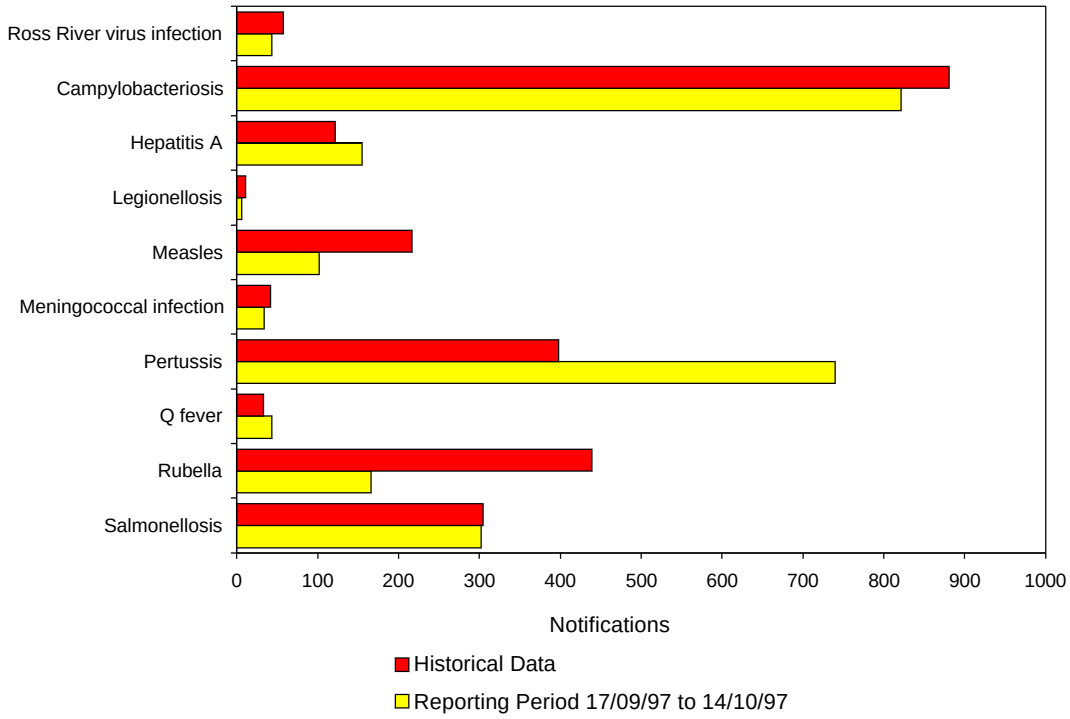
1. For HIV and AIDS, see Tables 4 and 5. For rarely notified diseases, see Table 3.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative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

NN Not Notifiable.

NEC Not Elsewhere Classified

- Elsewhere Classified.

Figure 3. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in the corresponding 4 week periods of the last 3 years and the 2 week periods immediately preceding and following those.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 17 September to 14 October 1997

Disease[2]	Total this period	Reporting States or Territories	Total notifications 1997
Brucellosis	4	Qld, WA	32
Chancroid			1
Cholera			2
Hydatid infection	5	Vic, WA	44
Leprosy			10

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1996.
2. No notifications have been received during 1997 for the following rare diseases: botulism, lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.

recent weeks. The total of 102 cases for the current four-week period is nearly three times the number of reports for the same period last year. The total for the year to date is also greater than 40% higher than last year, but much lower than the numbers of cases reported in 1994 and 1995 (Figure 4). Most reported cases are in young children (Figure 5). During 1997, 81 cases (15%) were reported in infants less than 1 year old, and a further 95 (18%) in children less than 2 years old.

The number of notifications of pertussis has continued to increase (Figure 6), 740 reports being received for the current four-week period. Recent increases have been seen in all jurisdictions except the Northern Territory. For 1997, the highest numbers of cases have been in children 8 and 9 years old, who together account for 14% of the total, although all age groups are affected (Figure 7). Infants under 1 year of age account for about 5% of cases.

A slight increase in notifications of rubella has been noted for the current period, compared with the previous four weeks, though the incidence remains lower than in 1996 (Figure 8). Of the current 166 cases, 76 (46%) were in males 15 - 24 years of age; this is consistent with data for 1997 as a whole (Figure 9).

Correction: In the last issue of CDI (21:298) the rate of pertussis for children less than 1 year old in 1996 should read 74.7 per 100,000 population.

Figure 4. Notifications of measles, 1991-1997, by month of onset

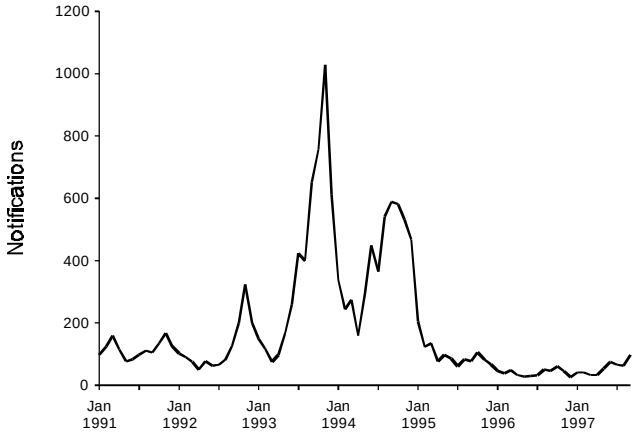
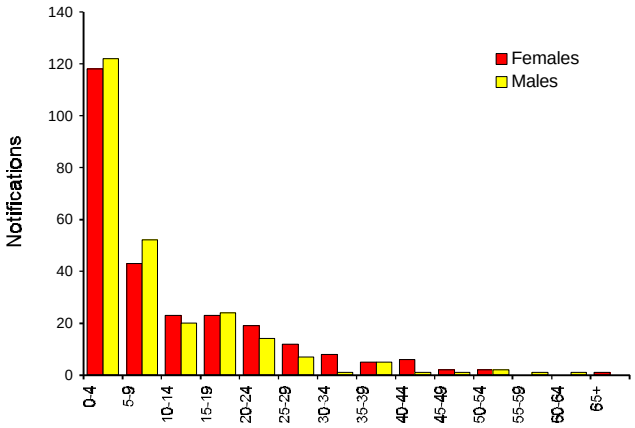


Figure 5. Notifications of measles, 1997, by age group and sex



National Influenza Surveillance, 1997

Three types of data are included in National Influenza Surveillance, 1997. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network, Department of Human Services, Victoria, Department of Health, New South Wales and Department of Health and Community Services, Northern Territory; laboratory surveillance data from the Communicable Diseases Intelligence Virology and Serology Laboratory Reporting Scheme, LabVISE, and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. For further information about these schemes, see CDI 1997;21:126.

Figure 6. Notifications of pertussis, 1993-1997, by month of onset

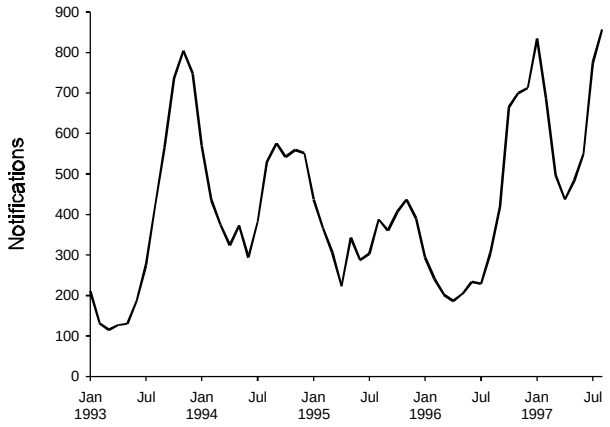


Figure 7. Notifications of pertussis, 1997, by age group and sex

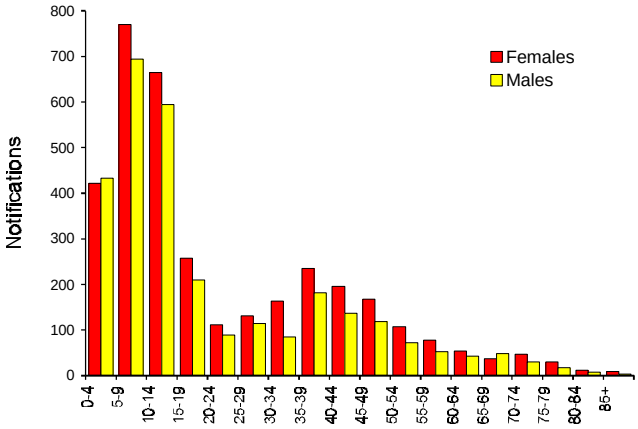


Figure 8. Notifications of rubella, 1991-1997, by month of onset

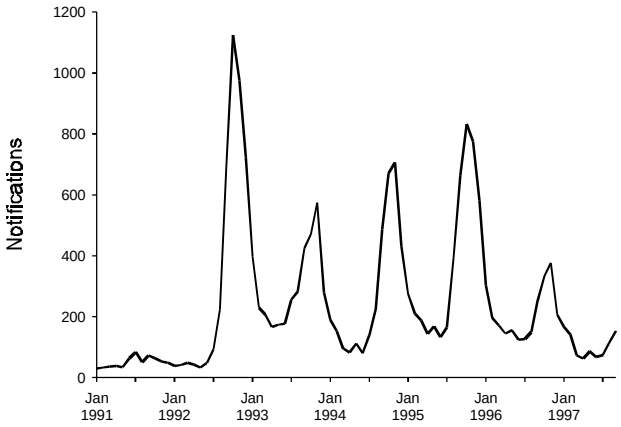


Figure 9. Notifications of rubella, 1997, by age group and sex

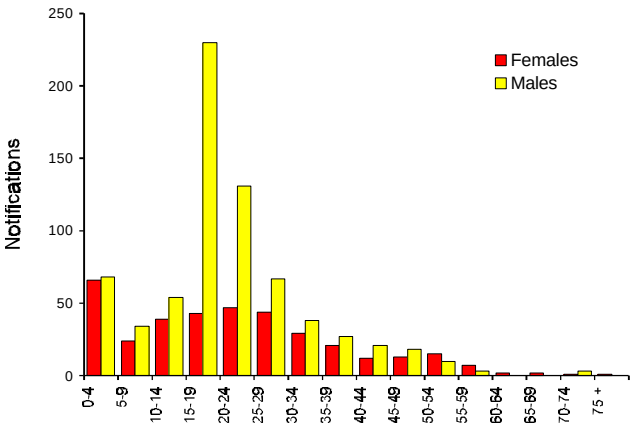


Figure 10. Laboratory reports of influenza, 1997, by type and week of specimen collection

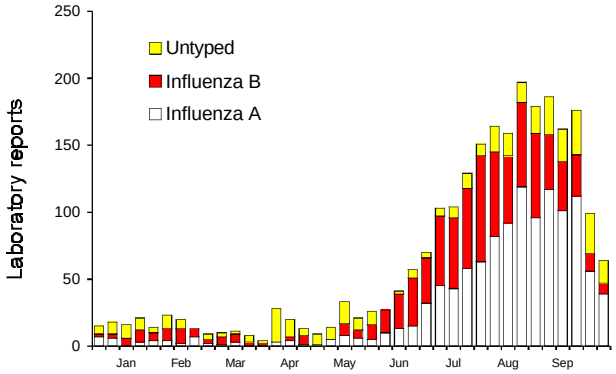
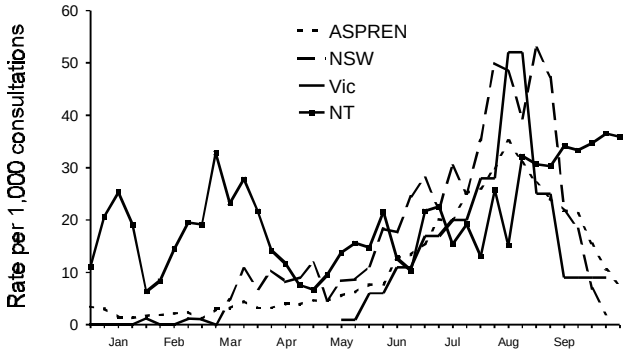


Figure 11. Sentinel general practitioner influenza consultation rates, 1997, by week and scheme



This is the final report of the National Influenza Surveillance for this season. Influenza activity continued to decline throughout most of Australia during September. However, the Northern Territory continued to show high activity throughout the month. The majority of laboratory reports for this period were for influenza A.

Laboratory Surveillance

A total of 318 reports of influenza virus were recorded by the LabVISE scheme this month. Of these, 191 were for influenza A, 68 for influenza B and 59 were untyped (Figure 10). Most reports during September were for influenza A. Reports for influenza A increased through July and August and have been decreasing since early September.

Sentinel General Practitioner Surveillance

Reports of consultation rates for influenza-like illness from the New South Wales scheme, the Department of Human Services Victoria, and the ASPREN scheme continued to decline throughout September, having reached a peak rate in late July to early August (Figure 11). The Northern Territory scheme, however, continued to show a high level of influenza activity throughout September.

HIV and AIDS Surveillance

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

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 June to 30 June 1997, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Totals for Australia: This period 1997	This period 1996	Year to date 1997	Year to date 1996
HIV diagnoses	Female	0	0	1	0	0	0	1	0	2	2	34	37
	Male	2	13	0	5	4	0	15	0	39	78	335	405
	Sex not reported	0	2	0	0	0	0	0	0	2	0	13	3
	Total[1]	0	24	0	3	3	0	14	5	49	77	340	365
AIDS diagnoses	Female	0	0	0	0	0	0	0	1	1	3	13	14
	Male	0	8	0	1	2	0	1	1	13	48	106	325
	Total[1]	0	8	0	1	2	0	1	2	14	51	119	339
AIDS deaths	Female	0	1	0	0	0	0	0	0	1	2	6	13
	Male	0	3	0	2	1	0	1	1	8	47	84	251
	Total[1]	0	4	0	2	1	0	1	1	9	49	90	264

1. Persons whose sex was reported as transgender 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 30 June 1997, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	21	491	6	110	46	4	186	77	941
	Male	180	10,521	91	1,759	616	78	3,586	820	17,651
	Sex not reported	0	2,056	0	0	0	0	28	0	2,084
	Total[1]	201	13,081	97	1,874	662	82	3,809	900	20,706
AIDS diagnoses	Female	7	152	0	37	19	2	59	23	299
	Male	80	4,159	28	718	308	40	1,478	328	7,139
	Total[1]	87	4,322	28	757	327	42	1,544	353	7,460
AIDS deaths	Female	2	109	0	27	14	2	40	14	208
	Male	52	2,940	22	503	208	26	1,161	235	5,147
	Total[1]	54	3,055	22	532	222	28	1,207	250	5,370

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

reporting interval indicated, to allow for reporting delay and to incorporate newly available information. 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) 9332 4648 Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for June 1997, as reported to 30 September 1997, are included in this issue of *CDI* (Tables 4 and 5).

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) currently comprises 107 general practitioners from throughout the country. Up to 9,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance. Of these, CDI reports the consultation rates for chickenpox, gastroenteritis, HIV testing (doctor initiated), HIV testing (patient initiated), influenza, measles, pertussis, Ross River virus infection and rubella. For further information, including case definitions, see CDI 1997;21:6.

Data for weeks 38 to 41 ending 21 and 28 September, and 5 and 12 October are included in this issue of *CDI* (Table 6). During the current reporting period, the consultation rate for pertussis has continued to rise, and is higher than previously seen this year. The consultation rate for influenza-like illness has declined markedly over the last six weeks. The gastroenteritis consultation rate has remained at a low level since the beginning of June. The consultation rate for chickenpox has remained steady since June. Measles, rubella and Ross River virus infection consultation rates have remained low for several months. The consultation rates associated with HIV testing have remained at moderate levels throughout the year.

Table 6. Australian Sentinel Practice Research Network reports, weeks 38, 39, 40 and 41, 1997

	Week 38, to 21 September 1997		*Week 39, to 28 September 1997*		*Week 40, to 5 October 1997*		*Week 41, to 12 October 1997*	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Chickenpox	12	1.8	5	0.9	19	3.2	7	1.4
Gastroenteritis	64	9.8	64	11.8	71	12.0	53	10.3
HIV testing (doctor initiated)	7	1.1	5	0.9	3	0.5	3	0.6
HIV testing (patient initiated)	7	1.1	12	2.2	7	1.2	11	2.1
Influenza	65	9.9	40	7.4	44	7.5	26	5.1
Measles	1	0.2	1	0.2	0	0.0	0	0.0
Pertussis	4	0.6	2	0.4	3	0.5	5	1.0
Ross River virus infection	2	0.3	1	0.2	0	0.0	1	0.2
Rubella	3	0.5	0	0.0	2	0.3	0	0.0

Figure 12. Rubella virus laboratory reports, 1995 to 1997, by month of specimen collection

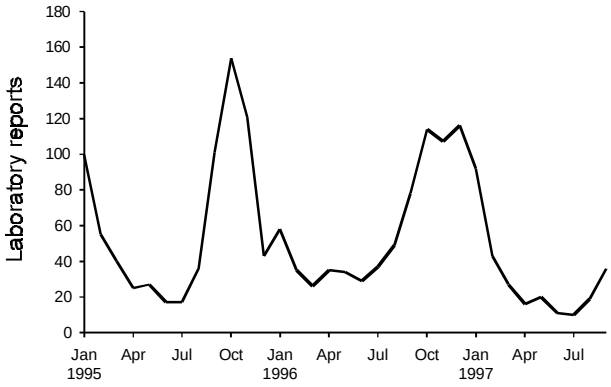
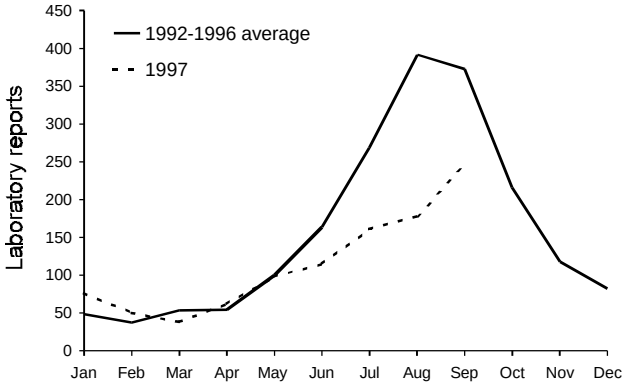


Figure 13. Rotavirus laboratory reports, 1992 to 1996 average and 1997, by month of specimen collection



Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease Australian encephalitis in humans. Currently 24 flocks are maintained in the north of Western Australia, ten in the Northern Territory, ten in New South Wales and ten in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information see CDI 1997;21:6

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5. Department of Health and Community Services, Darwin

Sentinel chicken serology was carried out for 20 of the 24 flocks in Western Australia in August and September 1997. There were 4 seroconversions to flaviviruses in the Derby flock from Western Australia in August. Three of the seroconversions were to MVE virus and one to a flavivirus that does not react with the MVE and Kunjin monoclonal antibodies. Most of the chicken flocks in Western Australian have now been replaced in preparation for the next wet season. Four new flocks have been added in the Gascoyne region of Western Australian. This will allow determination of the southerly limit of MVE and Kunjin virus activity in the state.

Five flocks of sentinel chickens from the Northern Territory were tested in August and September 1997. During this period there were 2 seroconversions to flaviviruses. One chicken from the Coastal Plains flock serconverted to Kunjin virus in August, and one from the Leanyer flock (in Darwin) seroconverted to both MVE and Kunjin viruses in September. The Leanyer seroconversion has not yet been confirmed.

LabVISE

The Virology and Serology Laboratory Reporting Scheme, LabVISE, is a sentinel reporting scheme. Twenty-one 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 1997;21:8-9.

There were 2,220 reports received in the *CDI* Virology and Serology Laboratory Reporting Scheme this 4-week period (Tables 7 and 8).

Laboratory reports of rubella virus have increased as expected for this time of year (Figure 12). There were 38 reports for this period, 24 (66%) were from Queensland. Eight reports were for females aged from 15 to 31 years, the remainder were males.

Figure 14. Parainfluenza virus type 2 and type 3 laboratory reports, 1995 to 1997, by month of specimen collection

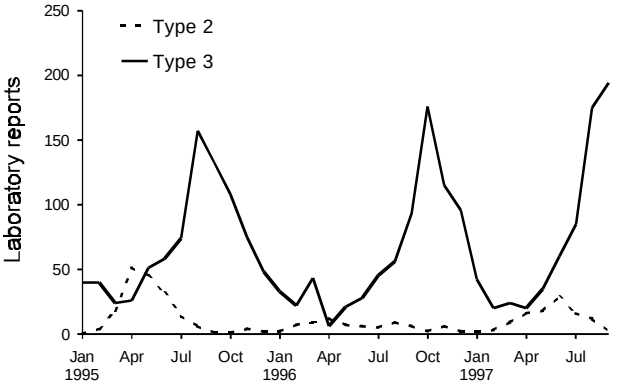

Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 11 September to 8 October 1997, and total reports for the year

	State or Territory [1]								Total this period	Total reported in *CDI* in 1997
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles, mumps, rubella										
Measles virus		1	1		2		1	2	7	53
Mumps virus							1	2	3	41
Rubella virus		1		25	11			1	38	481
Hepatitis viruses										
Hepatitis A virus		1	1	26	3			8	39	628
Hepatitis D virus					1				1	18
Arboviruses										
Ross River virus			2	13	1		2	3	21	2,050
Barmah Forest virus		2	2	10				1	15	213
Dengue type 2				1					1	49
Dengue not typed								1	1	58
Adenoviruses										
Adenovirus type 1					1				1	21
Adenovirus type 2					3				3	31
Adenovirus type 40								4	4	16
Adenovirus not typed/pending		17		30	29		9	18	103	848
Herpes viruses										
Herpes virus type 6								1	1	5
Cytomegalovirus		24		18	3	1	14	5	65	964
Varicella-zoster virus		2	2	23	9	1	24	23	84	1,131
Epstein-Barr virus		25	3	20	35		12	28	123	2,057
Other DNA viruses										
Contagious pustular dermatitis (Orf virus)								1	1	3
Poxvirus group not typed							1		1	3
Parvovirus				4			20	2	26	302
Picorna virus family										
Poliovirus type 2 (uncharacterised)					1				1	11
Rhinovirus (all types)		15		20			5	14	54	541
Enterovirus not typed/pending				14				30	44	538
Ortho/paramyxoviruses										
Influenza A virus		6		40	29		41	70	186	1,244
Influenza A virus H3N2				5					5	96
Influenza B virus		4		24	10		19	11	68	889
Influenza virus - typing pending					59				59	431
Parainfluenza virus type 1		1		1	1			4	7	60
Parainfluenza virus type 2				3	1				4	116
Parainfluenza virus type 3		52		25	8		16	74	175	935
Parainfluenza virus typing pending					18				18	229
Respiratory syncytial virus		26	1	47	83	3	26	96	282	4,282
Paramyxovirus (unspecified)							1		1	22
Other RNA viruses										
Rotavirus		136		1	22	3	22	35	219	1,240
Norwalk agent							7		7	77
Other										
Chlamydia trachomatis not typed		19	45	33	20	2	14	101	234	3,885
Chlamydia pneumoniae		1							1	3

Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 11 September to 8 October 1997, and total reports for the year, continued

	State or Territory [1]								Total this period	Total reported in *CDI* in 1997
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Chlamydia psittaci							3		3	54
Chlamydia species		2		1					3	28
Mycoplasma pneumoniae		16	2	37	8	2	27	15	107	1,469
Coxiella burnetii (Q fever)		4		5			3	1	13	269
Rickettsia tsutsugamushi				2					2	26
Salmonella species								1	1	1
Bordetella pertussis			1	61			53	68	183	1,485
Bordetella species							1		1	23
Legionella longbeachae								1	1	24
Cryptococcus species				1					1	18
Leptospira pomona				1					1	13
Leptospira australis				1					1	5
TOTAL		355	60	492	358	12	322	621	2,220	26,986

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.

Compared to the previous five years, the number of laboratory reports for rotavirus has been well below average (Figure 13). There were 219 reports this reporting period, 136 (62%) were from New South Wales. Most reports (196) were for children aged less than five years, of these 61 (28% of total) were aged less than one year.

Laboratory reports of parainfluenza virus type 2 have declined, but reports of type 3 are the highest recorded in recent years (Figure 14). The number of reports of parainfluenza virus type 3 is high, as expected for this time of year. There were 4 reports of parainfluenza virus type 2 this period and 175 reports of type 3.

Table 8. Virology and serology laboratory reports by contributing laboratories for the reporting period 11 September to 8 October 1997

State or Territory	Laboratory	Reports
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	54
	New Children's Hospital, Westmead	133
	Royal Prince Alfred Hospital, Camperdown	41
	South West Area Pathology Service, Liverpool	114
Queensland	Queensland Medical Laboratory, West End	303
	State Health Laboratory, Brisbane	205
South Australia	Institute of Medical and Veterinary Science, Adelaide	356
Tasmania	Northern Tasmanian Pathology Service, Launceston	10
Victoria	Microbiological Diagnostic Unit, University of Melbourne	11
	Royal Children's Hospital, Melbourne	144
	Victorian Infectious Diseases Reference Laboratory, Fairfield	172
Western Australia	PathCentre Virology, Perth	399
	Princess Margaret Hospital, Perth	171
	Western Diagnostic Pathology	107
TOTAL		2,220

Reminder

CDI is now a 4-weekly publication

Overseas briefs

Source: World Health Organization (WHO)

Cholera in Kenya

Health authorities have reported an outbreak of cholera in Migori District, Nyanza Province. The outbreak began mid-June and 555 cases with 29 deaths had been notified up to 24 August. Control measures instituted by the Ministry of Health, including health education and case management, are continuing. The area affected is in south western Kenya bordering on the United Republic of Tanzania.

Encephalitis in Nepal

The number of cases of encephalitis in Nepal increased markedly in September. The increase was mainly in the Nepalgunj and Dhangadi sentinel sites where, in the period 30 August to 26 September, two zonal hospitals reported 578 and 297 cases respectively. The total of 992 cases and 52 deaths for all sentinel sites reported during this period, brings the cumulative total since mid-April 1997 to 1,364 cases and 84 deaths.

Hantavirus Pulmonary Syndrome in the Americas

The number of cases, and the geographical distribution, of hantavirus pulmonary syndrome (HPS) has increased in the Americas since the syndrome was first identified in 1993 in the United States of America. It has now also been reported in Argentina, Bolivia, Brazil, Canada, Chile, Paraguay, Peru and Uruguay. Facing the possibility of further spread of HPS, the Ministers of Health in all countries in the Americas joined in a resolution to intensify surveillance for, and the fight against, hantavirus infection. Around 350 to 400 HPS cases have been confirmed in the Americas, most of them in Argentina and the United States of America. About 45% of the reported cases were fatal. The high fatality rate is associated with the sudden onset of pulmonary oedema and respiratory distress. There is no specific treatment for HPS, although prompt diagnosis is important for appropriate management of respiratory distress. Control measures in endemic areas focus on rodent control. Many infections have resulted from cleaning rodent-infested areas, where the use of a disinfectant such as chlorine bleach is recommended. If available, respiratory protection should be used during this high risk activity.

Notice to readers

Composition of Australian Influenza Vaccine for the 1998 Winter

A meeting of members of the Australian Influenza Vaccine Committee (AIVC) held on 10 October 1997 agreed that the composition of the Australian Influenza Vaccine for the 1998 winter season will be:

A (H1N1):
An A/Bayern/7/95 (H1N1) - like strain, 15 micrograms haemagglutinin per dose. The NIB 39, a reassortant of A/Johannesburg/82/96 is a suitable vaccine strain.

A (H3N2):
An A/Sydney/5/97 (H3N2) - like strain, 15 micrograms haemagglutinin per dose. Reassortants of A/Sydney/5/97 and A/Auckland/20/97 (equivalent to A/Sydney/5/97) are suitable vaccine strains.

B:
A B/Beijing/184/93 - like strain, 15 micrograms haemagglutinin per dose. The strain B/Harbin/7/94 currently used by vaccine manufacturers is a suitable vaccine strain.

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Contributions covering any aspects of communicable diseases are invited. Instructions to authors can be found in *CDI* 1997;21:9.

CDI is produced fortnightly by the National Centre for Disease Control, Department of Health and Family Services, GPO Box 9848 Canberra ACT 2601; fax: (02) 6289 7791, phone: (02) 6289 6895. For subscriptions or change of address please fax (02) 6269 1212 or write to PO Box 462, Fyshwick ACT 2609.

Electronic editions of *CDI* and data from the National Notifiable Diseases Surveillance Scheme (NNDSS) are available on the Department of Health and Family Services Internet web site. The address is'http://www.health.gov.au/hfs/pubs/cdi/cdihtml.htm'.