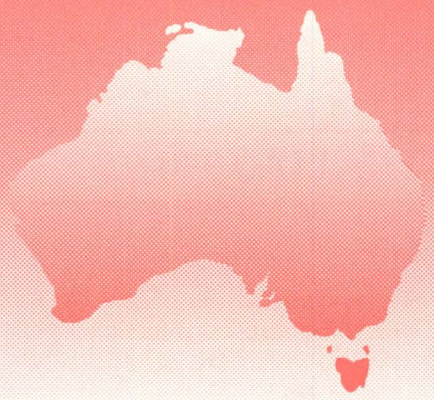

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DEPARTMENT OF HEALTH, HOUSING AND COMMUNITY SERVICES

COMMUNICABLE DISEASES NETWORK-AUSTRALIA
A National Network for Communicable Diseases Surveillance

REVIEW OF LEGIONELLOSIS IN SOUTH AUSTRALIA 1990-91

(Carolyn Walker and Phil Weinstein, Communicable Disease Control Unit, South Australian Health Commission)

Introduction

The first outbreak of legionellosis in South Australia occurred in 1986, and *Legionella pneumophila* serogroup 1 was found to be responsible[1]. Numerous sporadic cases of *L. pneumophila* infection followed, and these have been reviewed elsewhere[2,3]. *L. longbeachae* was first recorded in South Australia in 1987[4], and the 22 cases reported in 1988 remain the largest series of putatively soil-borne *Legionella* infections on record[3]. Both organisms are now responsible for a significant proportion of legionellosis in South Australia and cases notified during 1990 and 1991 are reviewed here.

Notification and Followup

Legionellosis has been a notifiable disease in South Australia since 1981, and both treating physicians and diagnostic laboratories report all cases to the Communicable Disease Control Unit (CDCU).

Cases are classified as either confirmed (organism isolated by culture) or presumptive (clinical presentation of legionellosis (Legionnaires' disease), a serological four-fold antibody titre rise or elevated illness titre with no other identified cause of illness). All notifications, whether confirmed or presumptive, are followed up by a nurse-epidemiologist to maintain consistency of data collection and to enhance awareness of changes in disease patterns. Personal contact is made with laboratory and medical staff and with the patient. The patient is interviewed in an attempt to identify risk factors, and environmental samples are collected by health surveyors if appropriate.

Results

From 1 January 1990 to 31 December 1991, 40 cases of legionellosis were notified to CDCU. Both *Legionella pneumophila* (several serotypes) and *L. longbeachae* have been responsible (Table 1). It is important to note that the nature of the non-productive cough in this disease makes culture of the organism difficult, and only 18% of serotypes and species recorded here could be confirmed by culture. Some recorded cases of *L. longbeachae* infection may therefore represent infection with antigenically related species which are serologically cross-reactive with *L. longbeachae*.

It can be seen that *L. pneumophila* and *L. longbeachae* account for exactly half of the cases each. The mean age of cases with *L. pneumophila* infection was 55.6 years (range 35-76 years), significantly lower than the mean age of 63.8 years (range 30-89 years) for cases of *L. longbeachae* infection ($t=2.018$, $df=38$, $p=0.05$).

The sex ratio of cases was similar for both organisms, and overall there were 2-3 males to every female. Analysis of disease incidence by week of onset and by postcode of residence revealed no temporal or geographic clustering of cases consistent with a point source outbreak. *L. pneumophila* infections occurred only during the summer in both years.

Given the population of 1.4 million in South Australia, 20 cases annually gives an incidence of legionellosis of 1.4 per 100,000. Deaths were attributed to both species (Table 1); there was a case fatality rate of 10% for both. The death rate from legionellosis in 1990-91 was therefore 0.14 per 100,000 population.

It was noted at interview that many people with *L. longbeachae* infection, although generally healthier, commented on preceding mental or physical ill health.

Soil samples were taken from the homes of six people with *L. longbeachae* infection and positive samples were found in four of these homes.

Discussion

Half the cases of legionellosis were attributable to *L. longbeachae*, which confirms the emergence of this species as a significant pathogen since its first reported occurrence in South Australia in 1987[4]. The absence of legionellosis in people less than 30 years of age, the mean age of cases being over 50 years, and a sex ratio of 2.3:1 are all consistent with the generally recognised epidemiology for the disease[5]. The higher mean age of

Table 1. Confirmed, presumptive and total cases of legionellosis, deaths and case fatality rates in South Australia, by causative organism, 1990-91

Species and Serotype	Confirmed Cases	Presumptive Cases	Total	Deaths	Case fatality rate
L. pneumophila 1	4	12	16	1	
L. pneumophila 2	0	3	3	0	10%
L. pneumophila 3	1	0	1	1	
L. longbeachae	2	18	20	2	10%
TOTAL	7	33	40	4	10%

cases with *L. longbeachae* infection may reflect the greater exposure of older people, such as retirees, to the organism during gardening activities.

All cases of legionellosis occurred sporadically, reflecting the ubiquity of *Legionella* species in the environment[6]. The summer incidence of *L. pneumophila* is consistent with higher summer water temperatures and the wider use of wet heat rejection plants (air conditioners). The occurrence of *L. longbeachae* in soil sampled from 4 of 6 homes is consistent with the proposed soil reservoir for this species[7].

Prior to 1990, the South Australian notification rate of sporadic cases in non-epidemic years was 0.5 per 100,000[3] and at 1.4 per 100,000 in 1990 and 1991 it more than doubled. This is not surprising when one considers the recent identification of *L. longbeachae* as a causative organism, coupled to an inevitable increase in awareness and ascertainment of legionellosis on the whole. By contrast, the case fatality rate has fallen from the 27% previously reported for the disease in South Australia[8] possibly reflecting earlier diagnosis and intervention, identification of milder cases and better management of the disease.

If there is to be a decrease in the number of cases of legionellosis, there must be increased public and business awareness of effective preventive measures. These include the regular maintenance of cooling towers and other industrial waters, and the handling of garden soil so as to minimise the risk of inhaling both dry and wet aerosols therefrom.

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LEGIONELLOSIS - NATIONAL NOTIFIABLE DISEASES DATA

(Marcus Hodge, Communicable Diseases Section, Department of Health, Housing and Community Services)

The Communicable Diseases Network Australia New Zealand (CDNANZ) collects data on legionellosis from two primary sources - National Notifiable Diseases reports and the *Communicable Diseases Intelligence* (*CDI*) Laboratory Reporting Schemes. Information from these databases is published in the Communicable Diseases Surveillance section of each issue of *CDI*.

Legionellosis (Legionnaires' disease) is now notifiable under the public health legislation of all States and Territories except the ACT. Notifications are collected into a National dataset by the Communicable Diseases Section, Department of Health, Housing and Community Services. The National case counts of legionnellosis over the past six years have ranged from 67 cases in 1988 to 112 cases in 1991 (Table 1).

Table 1. National legionnellosis notifications, 1986 to 1991, by year[1]

Year	Notifications
1986	68
1987	96
1988	67
1989	104
1990	90
1991	112

1. Not notifiable in Queensland until 1988 or in Tasmania until 1989; not notifiable in the ACT, but cases were notified in 1990 and 1991.

Table 2. Legionellosis - total notifications and rates per 100,000 population per year for 1990 and 1991, for Australia and by State and Territory

		Australia	NSW	Vic	Qld	SA	WA	Tas	NT	ACT
1990	Cases	90	27	13	24	19	4	0	0	3
	Rate	0.53	0.5	0.3	0.8	1.3	0.2	0	0	1.1
1991	Cases	112	24	22	29	21	15	0	0	1
	Rate	0.65	0.4	0.4	1.0	1.4	0.9	0	0	0.3

Figure 1. Legionellosis notifications, 1991, by age group and sex

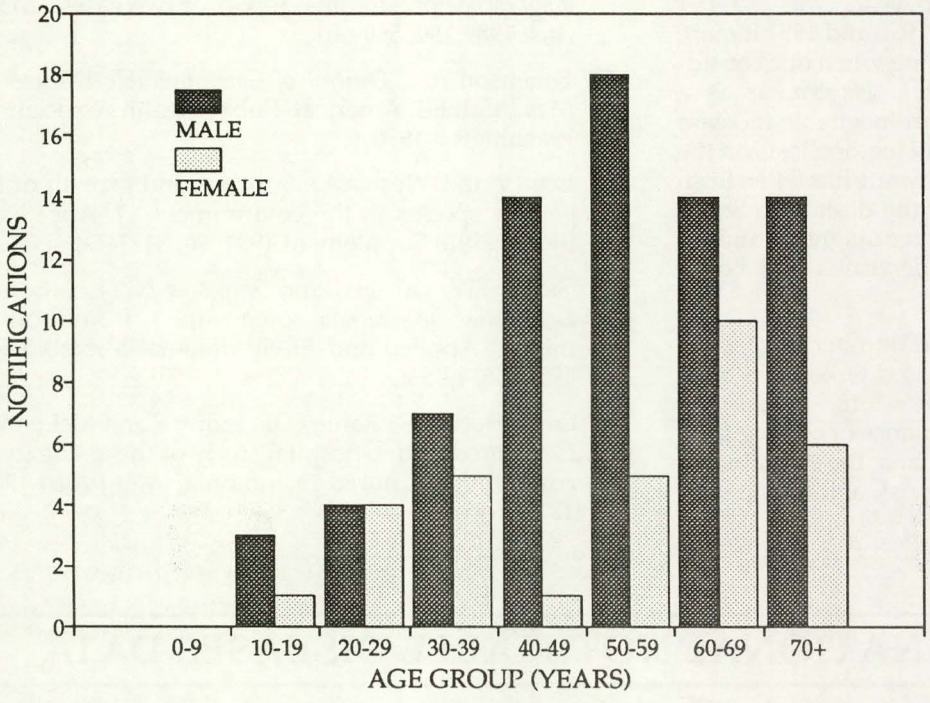
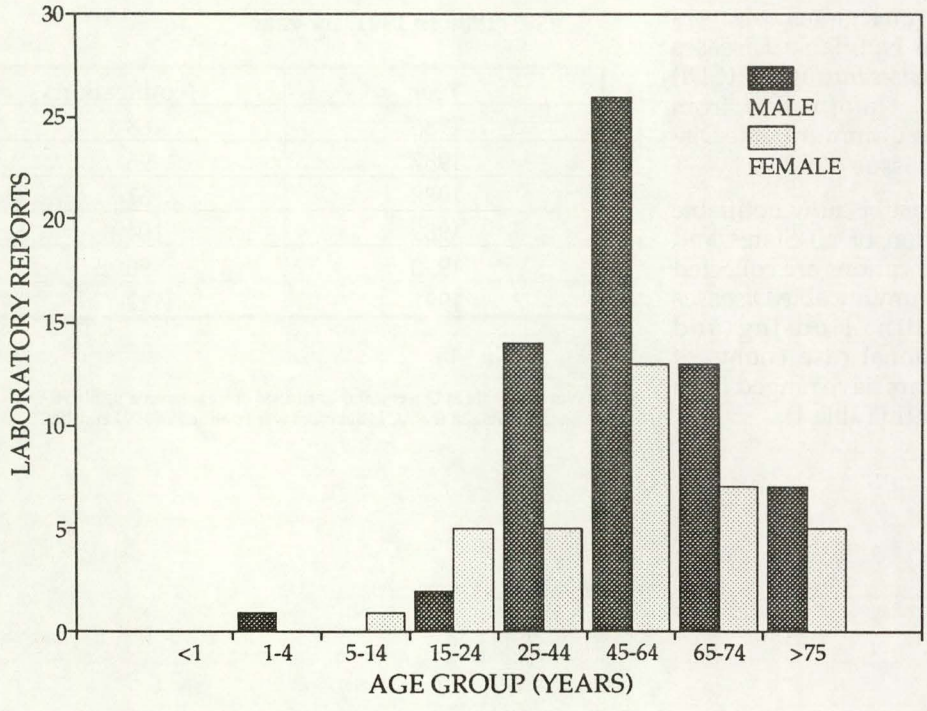


Figure 2. *Legionella* spp laboratory reports, 1986-1991, by age group and sex



In 1990 and 1991, the notification rates for Australia were 0.53 and 0.65 per 100,000 population, respectively. In both years, the highest rates were in South Australia (Table 2). (Rate calculations are based on the Australian Bureau of Statistics' estimated resident populations for June 1990 and the provisional estimated resident populations for June 1991).

Susceptibility to legionellosis is general but the disease is rare in those under 20 years of age[1]. The average age of infection for Notifiable Disease reports of legionellosis during 1991 was 53.6 years and the male to female ratio was 2.4:1 (usually reported to be about 2.5:1)(Figure 1).

The male to female ratio for the *CDI* laboratory reports since 1986 was 1.8:1 and shows a similar age distribution (Figure 2).

The *CDI* Laboratory Reporting Schemes data are based on reports from nine major sentinel laboratories and several smaller laboratories from the six States and the ACT. It has been collecting reports of *Legionella* since 1986. *L. pneumophila* cases (Figure 3) have occurred throughout the year and when considered alongside Notifiable Disease reports for 1991 there are no definite seasonal trends (Figure 4).

Fourteen serogroups of *L. pneumophila* are currently recognised and *L. pneumophila* serogroup 1 is most commonly associated with disease. The *CDI* Laboratory Reporting

Figure 3. *Legionella* **spp laboratory reports, by month, average for 1986 to 1991**

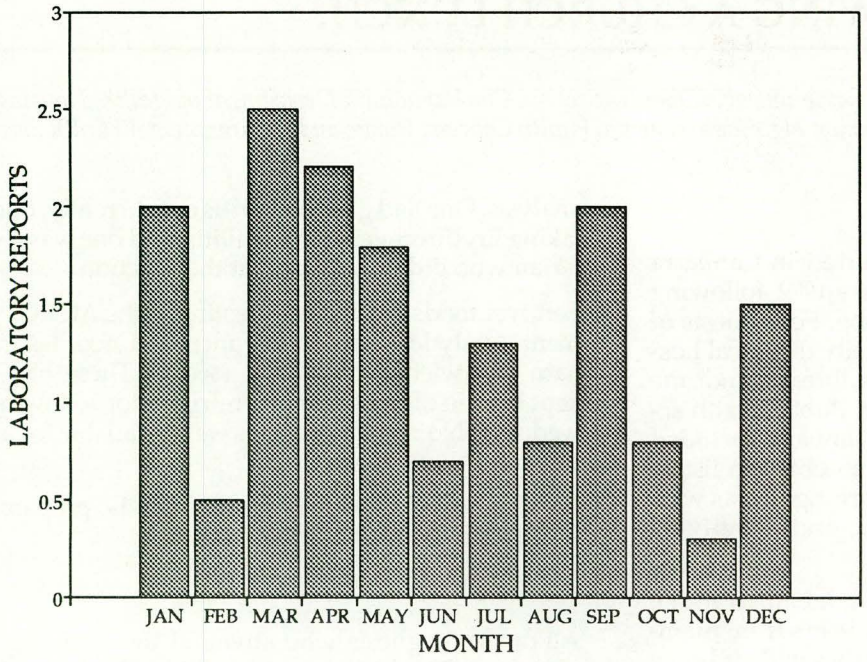


Figure 4. Legionellosis notifications, 1991, by month

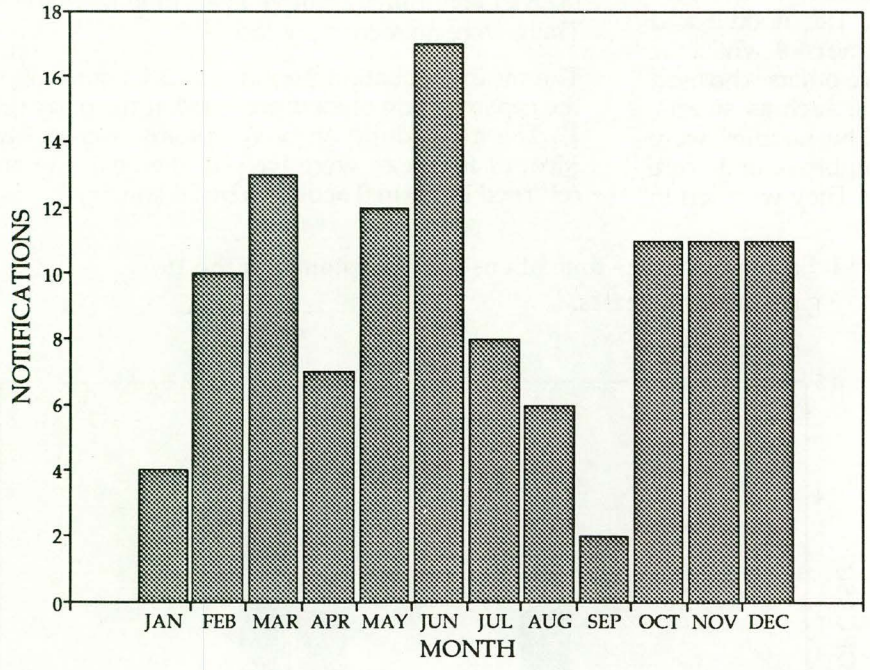


Table 3. Legionella spp laboratory reports 1986-1991[1]

Clinical category	Reports
Respiratory	68
Gastrointestinal	2
Pyrexia of unknown origin	3
Fever / malaise	3
Reticuloendothelial	1

1. Up to 2 clinical categories are recorded for each patient.

Schemes occasionally receive the serogroup of reports as supplementary information. Details of these reports have been published previously (*CDI* 1991;**16**:126,375,411). The Laboratory Reporting Schemes are currently being revised and it is proposed that the new system will collect serogroup information (if available) on each reported case.

The *CDI* Laboratory Reporting Schemes also collect limited clinical information. For *L. pneumophila* reports between 1986 and 1991, respiratory symptoms were the most commonly reported (Table 3).

No reports of mortality due to legionellosis have been received by the Laboratory Reporting Schemes.

Organisms related to *L. pneumophila*, including *L. longbeachae*, have been isolated predominantly from immunosuppressed patients with pneumonia. Cases of infection due to *L. longbeachae* have recently been reported in association with the use of potting mixtures in Queensland. One case of *L. longbeachae* infection was reported to the *CDI* Laboratory Reporting Schemes in October 1990 (male, 65-74 years). A further report was received in February 1991 (female, 45-64 years) and two were received in March 1991 (females, 45-65 years). All of these cases were reported by the Queensland State Health Laboratory.

One of the advantages of examining communicable diseases data from a national perspective is that significant outbreaks of rare conditions affecting more than one State or Territory, such as infection with *L. longbeachae*, may be more readily recognised and the risk factors for infection more easily identified.

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STAPHYLOCOCCUS GASTROENTERITIS OUTBREAK IN CANBERRA FOLLOWING A CHURCH LUNCH.

(David Cheah, Epidemiology Registrar, Communicable Diseases Section, Commonwealth Department of Health, Housing and Community Services; Peter Kong and Simon Ng, Environmental Health Officers, Public and Environmental Health Service, ACT Board of Health)

Introduction

An outbreak of gastroenteritis occurred in Canberra over the weekend of 18 and 19 January 1992, following a church lunch for a Chinese wedding. Four guests of the wedding who attended the casualty of a local hospital alerted authorities to the outbreak, and the investigation commenced that night. Public health action taken on the evening of 18 January included contacting the wedding organisers to obtain a list of guests for subsequent follow up, warning guests with leftover foods to avoid eating them, and identifying food samples for analysis.

The wedding reception took place in a church in the northern suburbs of Canberra. Volunteers from members of the church cooked various dishes in their home kitchens. Food items presented for consumption included spring rolls, curry pastries, gelatinous rice, noodles, sweets, drumsticks, sushi, ham and cheese sandwiches, and egg sandwiches. The noodle and sushi dishes were prepared by the same cook whilst the egg sandwiches were prepared by two others who used the same kitchen. Some food items, such as sweets, were bought from a local store. The noodles were boiled on the evening before the outbreak and were cooked on the morning of the party. They were left in the open overnight as there was insufficient room in the refrigerator for storage. The egg sandwiches were prepared on the morning of the party. Both noodles and egg sandwiches were carried to the church in cars, unrefrigerated, and allowed to stand at room temperature for several hours in the church before consumption.

Methods

We constructed a questionnaire to record data, including demographic characteristics of the guests, food histories, and basic clinical details. We defined a case of gastroenteritis as anyone who had eaten food at the wedding party and had developed vomiting, diarrhoea or abdominal cramps within 6 hours. We excluded three guests from analysis. One had gastroenteritis concurrently, one was taking Erythromycin for tonsillitis, and one was a vegetarian who did not eat food at the function.

Leftover foods available for testing in the ACT Government Analytical Laboratory included noodles, sushi, ham sandwiches, cakes and sweets. These had been kept by one of the cases in a refrigerator following the wedding. No egg sandwiches were available for analysis.

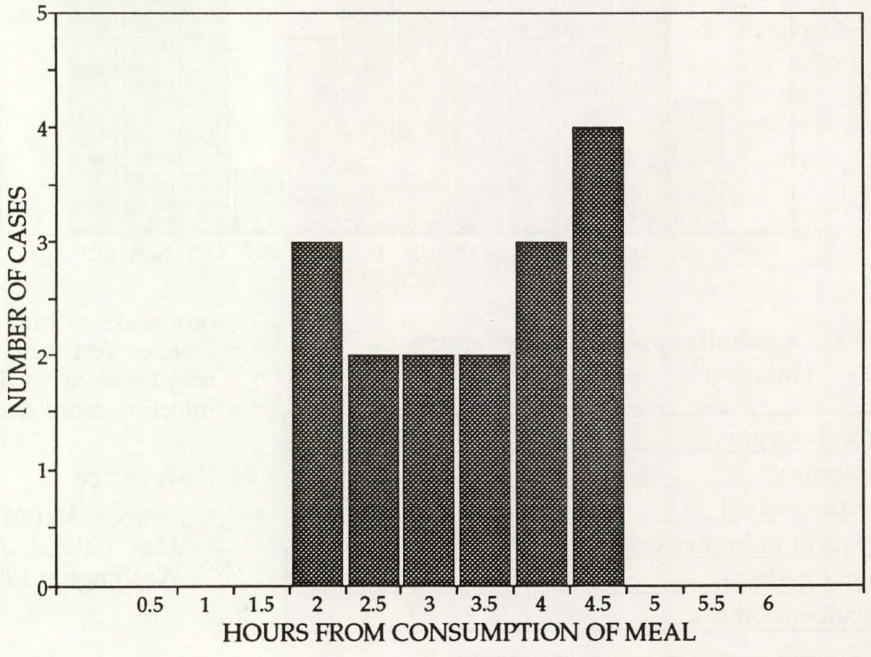
We also interviewed the three cooks who prepared the foods with the highest attack rates.

Results

All of the 52 guests who attended the wedding were contacted, 50 within forty eight hours of the outbreak. Predominant symptoms in the 16 guests who developed gastroenteritis were diarrhoea (87.5 %), vomiting (87.5%), abdominal cramps (43.8%) and nausea (18.7%). There were no secondary cases.

The mean incubation period was 3.4 hours following the consumption of food provided at the party (Figure 1). The mean duration of symptoms was 14.9 hours. Most of the cases were well by the next day and all returned to normal activities by 20 January.

Figure 1. Epidemic curve - time of onset of symptoms for the 16 gastroenteritis cases.



Males and females were affected equally (M:F ratio = 1:1). Fifty percent of those who were affected needed medical attention, either in the casualty of the local hospital or by general practitioner attendance.

We analysed food item specific attack rates using Epi Info. Relative risks were not able to be calculated because of small numbers in the contingency table. The three foods with the highest attack rates were egg sandwiches, sushi and noodles (Table 1). Those who ate egg sandwiches had the highest attack rate (41%), however, they account for only seven cases and all of them also ate noodles.

Microbiological results

The ACT Government Analytical Laboratory isolated *Staphylococcus aureus* from the noodles in significant amounts (4.0×10^7 per gram), and *Bacillus cereus* in smaller amounts (3.2×10^3 per gram). The significance of the presence of *Bacillus cereus* is not certain as the amount may be insufficient to cause disease.

A swab from the nostril of the cook who prepared the noodle dish, taken three days after the outbreak, grew *Staphlococcus aureus*. Phage typing of the organism is currently under way from both these isolates. Swabs taken from the two cooks who made the egg sandwiches did not yield *Staphylococcus aureus* or enteric pathogens. Stool samples collected from 8 cases nearly 72 hours following the outbreak were negative for *Staphylococcus aureus* and *Bacillus cereus*. No other pathogens were isolated from these samples.

Food Handling

The kitchen of where the noodle dish and the egg sandwiches were prepared was visited by Environmental Health Officers from the Public Health Branch of the ACT Board of Health on 21 January and 30 January. The kitchens were physically clean but the method of food handling, storage and transport to the church on the day of the outbreak were not according to standards. The practice of handling boiled noodles with bare fingers and leaving them unrefrigerated overnight may have been a factor contributing to the contamination and colonisation by *Staphylococcus aureus*. This was further compounded by carrying the cooked noodles in a car to the church and allowing them to stand at room temperature in the hall for several hours before consumption. Similar practices were noted for the egg sandwiches. The ambient temperature on the day of the outbreak was 26°C which is an ideal temperature for bacterial growth. Examination of the food handlers did not reveal any infected lesions on their hands.

Discussion

This is the first known report of a foodborne outbreak in the ACT in which a causative agent has been isolated.

Staphylococcal food poisoning is a common cause of gastroenteritis. The classical symptoms are those exhibited in this outbreak, with vomiting the main feature. Typically, the incubation period is short, and patients do not present with a fever[1]. The Centers for Disease Control estimate that 23% of all foodborne outbreaks are caused by *Staphylococcus*[2]. The duration of the illness is short, so the investigation must be performed at the earliest possible time, and the suspect food isolated, refrigerated and kept for analysis in the appropriate manner[3]. Stool or vomitus from affected individuals should also be collected as soon as possible from affected individuals. Since a significant number of people are *Staphylococcus* carriers, it would be impossible to completely eradicate nasal carriers of this organism. Outbreaks of staphylococcal food poisoning are typically associated with improper storage of food and poor hygiene of food handlers. Storage of foods at refrigeration temperatures would prevent most outbreaks.

Acknowledgments

The following persons and organisations are acknowledged for their help in the investigation: Dr Robert Scott, Chief Health Officer, ACT Board of Health; Mr Alec Percival, Director, Public and Environmental Health Service, Public Health Branch, ACT Board of Health; Ms Gay Priest, Barry Moran's Pathology, Canberra; Dr Simon Rockliff and Mr Brad Duck, ACT Government Analytical Laboratory.

Table 1. Food specific attack rates among 49 guests at a wedding party, Canberra, 1992

Food Items	Number who ate	Number ill	Attack Rate (%)
Spring roll	40	10	25
Curry pastries	43	13	30
Gelatinous rice	36	12	33
Noodles	42	16	38
Sweets	32	10	31
Drumstick	37	12	32
Sushi	37	15	41
Ham/Cheese sandwich	14	4	27
Egg sandwich	17	7	41
Total	49	16	33

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EARLY DETECTION OF HERPES SIMPLEX ENCEPHALITIS BY POLYMERASE CHAIN REACTION

(Jacqueline Montanaro, Department of Microbiology, Royal Children's Hospital, Parkville, Victoria. Reprinted with acknowledgment from Infectious Diseases Bulletin No 67, August-October 1991, Editor Geoff Hogg)

Case Report

A ten year old girl who was previously well, presented with a nine day illness, characterised by fevers and headache. On the day prior to her admission her temperature was noted to be 'very high'. She had rigors, seemed delirious with abnormal speech and was becoming increasingly drowsy. By the day of admission, she was agitated, abusive and incomprehensible. She was admitted to a Base Hospital where investigations were all normal except for CT scan which showed left uncal haemorrhage.

The CSF taken on the day of admission contained $100x10^6$/L red blood cells, $400x10^6$/L lymphocytes, $100x10^6$/L polymorphs, 3.7 mmol/L glucose, 0.88g/L protein, intracranial pressure 13-15cm. Polymerase Chain Reaction (PCR) for herpes simplex virus (HSV) DNA was positive. Serum taken on day two of admission was negative for HSV IgM and low positive for IgG. A second CSF was taken on day 3 of admission, containing $420x10^6$/L red blood cells, $550x10^6$/L lymphocytes. $24x10^6$/L polymorphs, 2.8mmol/L glucose, 0.8g/L protein. The CSF was sterile for both bacteria and viruses, was negative for HSV IgM and IgG and was again positive by PCR. A third CSF taken eight days after admission had borderline HSV IgM and positive HSV IgG, but was negative by PCR.

When the PCR results were known, ceftriaxone therapy was ceased and acyclovir treatment was continued for a total of 14 days. Ten days after admission she was well, eating and drinking. Three weeks after admission she was discharged with residual left facial palsy and global language difficulties. At follow-up, she continues to have significant speech impairment and learning difficulties.

Detection of Herpes Simplex Virus in CSF by PCR

In 1991, a retrospective PCR study was performed on CSFs from 15 patients. All patients were confirmed to have herpes simplex encephalitis (HSE) by detecting specific antibody in the CSF or by finding viral antigen in brain biopsy. The primers for PCR chosen from a published method, used a 141 base-pair glycoprotein D segment of the herpes simplex us6 (unique short) region[1,2]. Each PCR cycle of denaturing, annealing and extension was carried out automatically at 94°C, 60°C and 72°C respectively in a DNA thermal cycler for 35 cycles.

The 'serology' results were as follows: 3/11 CSFs showed positive serology less than four days after onset of encephalitis, 11/12 CSFs showed positive serology more than four days after onset of symptoms.

The PCR results were as follows: 6/9 CSFs taken during the first three days after onset of encephalitis were positive, 1/1 CSF taken between four and ten days after onset was positive, only 1/9 CSFs taken more than ten days after onset of symptoms was positive.

Sensitivity: PCR was shown to be 100 fold more sensitive than tissue culture isolation for HSV types 1 and 2.

Specificity: No reaction was noted with specimens containing cytomegalovirus (3 urine samples) or varicella-zoster virus (2 vesicle samples) nor in CSF specimens from non-HSE patients (20 samples).

The results of this study indicate that PCR for HSV on CSF is most useful during the first 4 days after the onset of encephalitis, when 'serology' may be negative. PCR on CSFs with suspected HSE is much less useful after 10 days when serology detects more positives. The two methods may be considered complementary. Another point to note, however, is that although a positive PCR result may be considered diagnostic, the significance of a negative result remains uncertain. Following the retrospective study, 10 CSFs from patients with a suspected HSE have been examined. Only one of these was positive by PCR.

PCR is a technique used to amplify very small amounts of a particular piece of DNA into detectable amounts. The technique depends upon targeting a segment of DNA that lies between two regions of a known DNA sequence. Two oligonucleotides are used as 'primers', or starting points, for a series of synthetic reactions that are catalysed by a heat stable DNA polymerase. (It was the discovery of this heat stable DNA polymerase from a thermophilic bacteria called *Thermus aquaticus (TAQ)* that has enabled the PCR technique to be automated and thus readily accessible[3,4].) After one cycle of denaturing, annealing and extension, the amount of target DNA is the sample will have doubled. After the next

cycle, there will be four copies of target DNA produced. This process may be repeated over and over again, increasing the amount of target DNA exponentially. Thus from a single copy of target DNA, we can produce 10^6 copies of particular DNA sequence after approximately 20 cycles. Other methods are then used to check that the size of the PCR product is as expected[3]. Further testing of the PCR product with DNA probes is a necessary confirmatory test for diagnostic testing.

The amount and concentration of each solution in the PCR reaction tube is critical to the success PCR. The selection of primers that are unique and specific for the DNA we are seeking is also essential. In addition, the annealing temperature and the time for each step must be 'customised' for each reaction using different sets of primers[4].

The most significant limitation to the routine use of PCR for diagnosis is the risk of false-positive results caused by contamination. the exquisite sensitivity of the PCR enables a single piece of target DNA from a previous PCR (amplicon) to contaminate a PCR tube containing a patient's specimen. Elaborate protocols to limit this risk include physically isolating PCR products and preparation mixtures (for example by setting up reactions in a different room from that used for running visualising gels), autoclaving all solutions, using disposable gloves, 'premixing' reagents, and using positive displacement pipettes to aliquot samples. If there is any doubt about a result, the entire test must be repeated[5].

PCR shows great promise for detection of organisms that require special handling, for example *Mycobacterium tuberculosis*, and other *Mycobacterium spp., Mycoplasma, Legionella* and *Chlamydia* species, where culture is expensive, slow or difficult[4]. In addition, fixed or museum specimens and archaeological specimens may be reinvestigated with this new technology. Given enough time and money, the usefulness of PCR to microbiology is limited only be our ingenuity and imagination. We cannot, however, lose sight of the fact that more traditional methods such as culture and serology should not be abandoned in favour of the 'boutique' PCR detection method. These traditional methods are well characterised and the diagnostic implication of a positive or negative result are known. Another advantage of traditional methods is that culture systems for both bacteria and viruses are broad-based and can detect multiple organisms with one test. PCR by comparison can only give a yes/no answer to a single question. Thus, although PCR has opened up an exciting new area of microbiology its use does not allow us to abandon more traditional methods of microbiological diagnoses.

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OVERSEAS BRIEFS

In the last two weeks, the following information regarding cholera and influenza has been supplied by the World Health Organization, Australian diplomatic missions and the Institut Pasteur, Paris.

Cholera Update

The government of **Argentina** has confirmed an outbreak of cholera in the far north-east region of the northern Province of Salta. Ten fatalities have been reported, all Mataco Indians who live in the remote jungle area along the Argentine-Bolivian border. The disease is believed to have entered Argentina through waterways shared by the two countries, contaminated fish and the continual border crossings by inhabitants of the region. It is anticipated that the disease will probably reach Buenos Aires in the not too distant future.

Cholera has also recently spread to **Belize**, with the Toledo District recently being declared infected. Cases occurred in the country in January.

Reports of cases in January were also received from **Chile**, which had cases early in 1991 but has not had any in recent months. The Tarapaca province has been declared infected.

Honduras, Colombia, Venezuela, Brazil, Burkino Faso and **Tanzania** also reported cases in January and **Iraq** has reported cases for December and January.

Influenza in the Northern Hemisphere Update

Influenza A(H3N2) has been dominant in Europe since November 1991, but A(H1N1) is now appearing more often (15 to 20% of isolates so far). Influenza B remains exceptional, but has recently been reported from China.

Activity continues to decrease in most of Europe, but **Belgium, Czechoslovakia, Hungary, Norway** and **Yugoslavia** report increased activity since the middle of January. In Hungary, a high level of morbidity has been reported, with 172,000 cases of influenza-like illness reported in one week, and in Yugoslavia, 145,500 cases of influenza-like illness have been reported. Outbreaks continue to be reported from **Switzerland** and the **United Kingdom**.

By 10 February, there had been 267 influenza isolations in the United Kingdom: 228 influenza A(H3N2), 38 influenza A(H1N1) and one influenza B. In **France** by 6 February, there had been 328 strains isolated and 84% were influenza A(H3N2).

In North America, influenza continues to decrease. In the **USA**, 257 influenza A(H3N2), 123 influenza A(H1N1) and 3 influenza B strains had been identified by 12 February. There has been influenza A(H3N2) activity in **Canada**.

China has reported outbreaks of influenza in schools and families in the Beijing area and influenza B strains have been isolated.

COMMUNICABLE DISEASES SURVEILLANCE

- There were 32 reports of **Ross River virus** infection received this fortnight. Two were from New South Wales (one from Cobar), 8 were from Western Australia (Busselton, Dawesville, Glen Forest, Leeming, Maidavale, Bayswater and Carlisle) and there were 28 from Queensland (Beenleigh, Brisbane, Cairns, Redcliff, Roma, Rockhampton, Townsville, Toowoomba and Warwick). The specimen collection dates ranged from November to January.

  Most of the Ross River virus patients had muscle/joint symptoms, skin disease or general malaise, but for a 17 year old male from Townsville, encephalitis was the reported syndrome.

  Encephalitis has only rarely been reported in the *CDI* Laboratory Reporting Schemes. In a total of 8993 Ross River virus infection reports, only 13 reports have listed encephalitis as the presenting syndrome: 2 in 1982, one in 1984, 8 in 1987, one in 1989 and this one in 1992. Nine of these patients were males and 4 were females. Their ages ranged from 17 to 76 years. One of the patients in 1982 was diagnosed by demonstration of a single high titre, but all the others, including the patient this fortnight, were diagnosed on the basis of demonstration of IgM to the virus.

- **Dengue** was reported in two 29 year old male patients who had recently been in South-east Asia. One had **dengue-1** and the other had an untyped **dengue** infection. **Dengue-2** was reported in a 56 year old male who had recently been overseas and who presented with severe fever and hepatic symptoms.

- **Kokobera virus** infection has been reported to the Laboratory Reporting Schemes for the first time. The patient was a 65 year old male from the Rockhampton area. The diagnosis was made by the State Health Laboratory, Brisbane, by demonstration of IgM in an assay for the arbovirus which they have recently developed.

- There were two reports of **Stratford** virus infection, making a total of 3 reports of this virus for the season. One case was a 39 year old male from the Cairns area (no reported clinical information), and the other was a 38 year old male from New South Wales who had general malaise/mild fever. The diagnoses were made by the State Health Laboratory, Brisbane, and in both cases was by demonstration of IgM.

- There were 35 reports of *Mycoplasma pnuemoniae* this fortnight, with 11 from Western Australia and 10 From Victoria. Unusual symptoms were reported for 3 patients: a 28 year old male presented with cardiac symptoms, a 10 year old male presented with muscle/joint disease and one patient had skin/mucous membrane disease.

- Twenty-eight cases of untyped **adenovirus** were reported. Symptoms reported for most patients were respiratory or gastrointestinal. For one 3 year old male patient, however, cardiac symptoms were reported. For two patients, the infection was reported to be nosocomial. The patients, a one year old male and a 6 month old female, had been in the same hospital room and both had respiratory symptoms.

- There was one further case of **echovirus type 4** infection reported. As for the 2 cases reported last fortnight, this one was from Victoria. The patient was a 29 year old male with meningitis.

- Victorian laboratories also reported 4 cases of **echovirus type 16** infection, bringing the total for 1991 to 9 cases. Meningitis was the reported syndrome for one of the patients, an 18 year old female.

- **Rubella** was reported in 10 patients. Included were females aged 25 years and 30 years.

- There were 10 reports of **hepatitis A** this fortnight. Age and sex details were provided for 9 of the patients, and 8 were adult males.

- Seventy-eight reports of **hepatitis C** were received. Amongst them was one patient with a history of injecting drug use, and two haemophiliac brothers, aged 5 and 6 years.

- There were 83 reports of **cytomegalovirus** infection. Guillain-Barré Syndrome was the reported

syndrome for one patient, a 44 year old female. Congenital infection was reported in a 4 day old male and in a one year old female. Further isolates were from urine samples of a 1 day old male with jaundice, a 2 year old girl with Kawasaki's disease (mucocutaneous lymph node syndrome) and post-mortem tissue of an 8 month old male who had suffered SIDS. Three pregnant women were also diagnosed with this virus this fortnight.

- There were 26 reports of **varicella-zoster virus** this fortnight. Included was one case of encephalitis, in a 22 year old male.
- Thirteen reports of **ornithosis** were received, with 12 from Victoria. One patient had had a cockatoo which had died and another ran an aviary.
- One case of **brucellosis** was reported. The patient was a male (age group 25 to 44 years) who was a pig shooter. The species of *Brucella* was not determined, but feral pig exposure has recently been associated with *Brucella suis* infection (*CDI* **15**:376-378 and 378-381).
- Four cases of **syphilis** were reported in pregnant women.
- A further report of endocarditis caused by non-toxigenic ***Corynebacterium diphtheriae*** has been received. The patient was a 9 year old Aboriginal girl from the Mackay area of Queensland. The organism was isolated from blood culture, and toxigenicity studies were done by the State Health Laboratory, Brisbane.
(Peter Lowe, Central Queensland Pathology Laboratory, Mackay)
- There were 19 reports of **Q fever** during this reporting period: 10 from New South Wales, 2 from Victoria, and 7 from Queensland. Three patients were described as meatworkers. 'Q fever in a nearby meatworks' was the comment recorded for another patient.

Australian Sentinel Practice Research Network

ASPREN reports published this fortnight include the first 2 reports of gastroenteritis for the scheme (Table 1). Influenza reports remain at low levels, as expected for this time of year, however, the few reports received are consistent with the small number of influenza reports still being received by the Laboratory Reporting Schemes (2 influenza A and 6 influenza B this fortnight). The number of encounters for weeks 4, 5 and 6 were 3940, 6206 and 5914 respectively.

Australian Encephalitis: Sentinel Chicken Surveillance Programme - Serological Results for January 1992

Sentinel chicken serology results from chickens tested in Victoria and New South Wales during January 1992 showed no evidence of flavivirus activity. Results from Western Australia indicated one additional seroconversion to Murray Valley encephalitis (MVE) virus at Kununurra at the end of January. Six chickens which had previously seroconverted to MVE at Kununurra were replaced at the end of January.

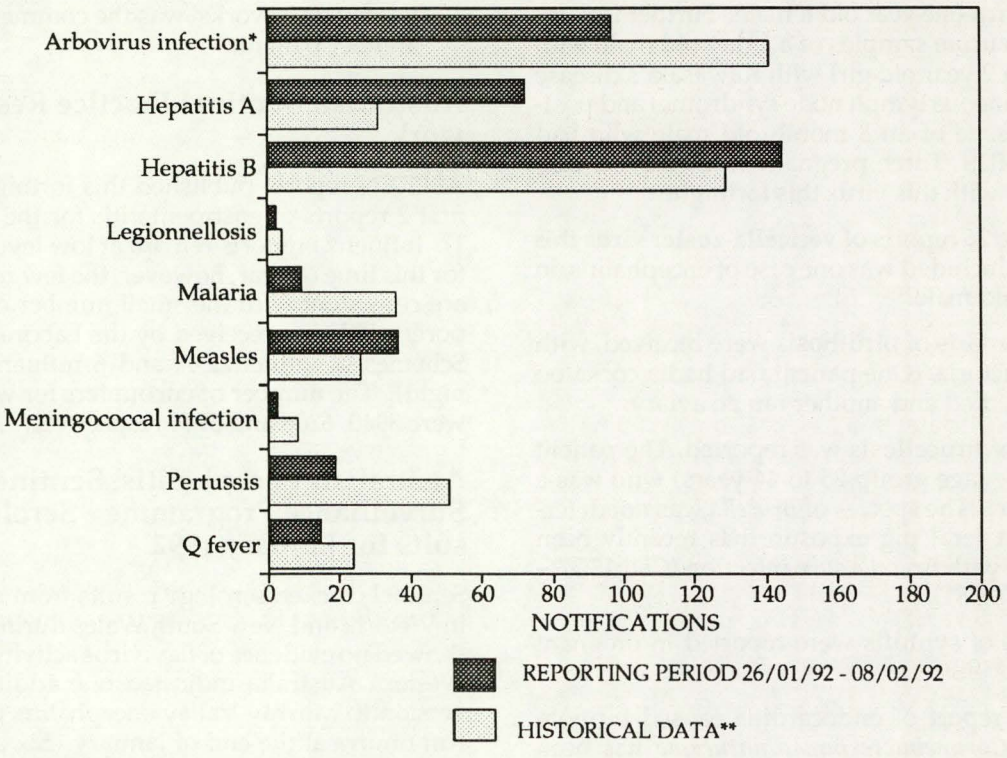
Information on the location of sentinel chicken flocks was presented in *CDI* **16**:55-57.
(J Aldred, Victorian Institute of Animal Science; L Hueston, Virology Department, Westmead Hospital, NSW; AK Broomand JS Mackenzie, Department of Microbiology, the University of Western Australia)

Table 1. Australian Sentinel Practice Research Network, Weeks 4-6 1992[1]

Condition	Week 4, to 26 January 1992		Week 5, to 2 February 1992		Week 6, to 9 February 1992	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	4	1.02	18	2.90	4	0.68
Measles	0	0	1	0.16	0	0
Mumps	0	0	3	0.48	2	0.34
Rubella	0	0	6	0.97	3	0.51
Pertussis	2	0.51	0	0	1	0.17
Genital herpes	0	0	4	0.16	1	0.17
Gastroenteritis	2	0.51	0	0	0	0

1. ASPREN data may be subject to revision

Figure. National Notifiable Diseases Reports, 26 January 1992 to 8 February 1992 and historical data**



*Includes Ross River virus and Dengue

**The Historical data are the averages of the number of notifications in 6 previous 2-weeks reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

LABDOSS (Laboratory Database of Organisms from Sterile Sites)

LabDOSS is a new *CDI* laboratory reporting scheme which has been set up to monitor significant isolates from sterile sites. A pilot for the scheme has been operating in several Sydney laboratories for the last six months, and it is hoped that eventually it will be expanded throughout Australia.

Regular reports of the Scheme will be published in the Communicable Diseases Surveillance section of *CDI*, starting with this issue.

Aims of the *CDI* LabDOSS Scheme

Laboratory reports collected in the scheme will supplement the National Notifiable Diseases reports and the limited number of reports of significant isolates collected in the 'Pathogens Reporting Scheme'. (It is planned that LabDOSS will eventually take over the collection of sterile site data from the 'Pathogens Reporting Scheme,' and that the 'Viruses Reporting Scheme' will incorporate the serological reports from the pathogens scheme to become a more general virus and serology reporting scheme.)

The scheme will collect information which is not normally collected or compiled for cases reported in the National Notifiable Diseases Reports. For *Haemophilus influenzae* for example, it will collect type information, and the clinical characteristics (meningitis, bacteraemia, cellulitis) of each infection. This type of information is currently unavailable Australia-wide, and will prove invaluable in any future assessment of the impact of *Haemophilus influenzae* type b vaccination of children.

Reports of organisms which cause diseases which are not notifiable will also be collected, enabling the surveillance of meningitis and other invasive disease caused by organisms such as *Streptococcus pneumoniae*, *Cryptococcus neoformans* and Group B *Streptococcus*. When the Scheme is fully operational, epidemiological patterns of these organisms will be able to be characterised for the first time on an Australia-wide basis.

LabDOSS sterile sites surveillance also has the potential to enable recognition of outbreaks of unusual organisms causing bacteraemia or other invasive disease. For example, the recent cases of non-toxigenic *Corynebacterium diphtheriae* endocarditis (*CDI* 15:277) were brought to the attention of *CDI* through this scheme.

The system will also provide research opportunities through the provision of data, published in *CDI* and otherwise supplied.

LabDOSS is thus similar to the Bacteraemia and Meningitis Surveillance scheme of the Victorian Hospital Pathogens Surveillance Scheme of the Standing Committee on Infection Control of the Health Department of Victoria. This scheme publishes reports of Victorian blood and CSF isolates in its regular report, *VICBUG*.

Data Collected in the *CDI* LabDOSS Scheme

Reports in the LabDOSS scheme are of significant isolates cultured from normally sterile sites. One report is made for each episode of infection and comprises three types of data:

1. Clerical data: laboratory patient identification, the first two letters of the patient's first two names (to allow for checking for duplicates), date of specimen collection, sex, date of birth or age, and patient location information (postcode);
2. Clinical data: for example endocarditis, meningitis or cellulitis, risk factors such as recent surgery or an immunocompromised state, and whether or not the patient died as a result of the infection;
3. Microbiological data: source specimen, method of diagnosis (isolation, antigen detection, antibody detection) and an identification of the organism which is as full as the laboratory has determined.

LabDOSS reports can be provided to *CDI* on paper forms, or data can be entered into a computer using the LabDOSS program and then sent on floppy disk.

The LabDOSS program has been written in EpiInfo, a program which was developed at the Centers for Disease Control in Atlanta, USA. It combines word processing, database management, statistical analysis and graphics into a package which can be used by those with minimal experience with computers. The EpiInfo software, LabDOSS programs, EpiInfo manual and LabDOSS manual are all provided free of charge to laboratories that may wish to contribute to the LabDOSS scheme. (An IBM compatible personal computer is required.)

An important feature of the system is that the LabDOSS programs can be tailored to the individual needs of the laboratory whilst retaining the ability to generate reports for *CDI*. Laboratories can record data other than those required for *CDI* reports, for example, full names and addresses of patients, referring practitioners, and details of 'contaminants'. They can then use the programs to store and analyse both the data that are sent to *CDI* and their supplementary data.

Enquiries from laboratories wishing to join the LabDOSS Scheme, or general enquiries on the system are welcome. The contact person is Dr Leslee Roberts, phone (06) 289 7217.

Tabulated LabDOSS data will be published regularly in *CDI* as will assessments and reviews of the data. Data for January 1992 from the pilot scheme is presented in this edition of *CDI* in three groups; bacteraemia reports (Table 2), meningitis and CSF reports (Table 3) and other sterile site isolate reports (Table 4).

Contribution laboratories for Janaury were the Institute of Clinical Pathology and Medical Research, Sydney (47 reports), the South-west Area Pathology Service, Liverpool (26 reports), and the Royal Prince Alfred Hospital, Camperdown (54 reports).

Table 2. LabDOSS reports of blood isolates for January 1992[1]

Organism	Number of Isolates	Clinical Information	Risk Factors
GRAM POSITIVE BACTERIA			
Staphylococcus aureus	14	Gastrointestinal 1	Diabetes 2, IV central line 2, Trauma 1, Preterm neonate 1, IV peripheral line 2, Malignancy 1, Neutropaenia 1, Other vascular prosthesis 1, Urinary tract surgery 1
Staphylococcus epidermidis	12	LRTI 1	Malignancy 4, Preterm neonate 1, IV central line 3, Neurological surgery 1
Staphylococcus coagulase negative	7	LRTI 3, Osteomyletitis 1	Other immunocomprosmised 1
Streptococcus pneumoniae	2	LRTI 1	Neutropaenia 1
Streptococcus milleri	4		Neutropaenia 1
Streptococcus bovis	1	Endocarditis (native valve) 1	Abdominal surgery 1
Streptococcus viridans	1		IV central line 1
Streptococcus alpha-haemolytic	1		
Streptococcus sp.	1		Other Immunocompromised 1
Enterococcus faecalis	1	Endocarditis (prosthetic valve) 1	Other vascular prosthesis 1, Preterm neonate 3
Bacillus sp.	1		Postnatal 1
Corynebacterium JK	1		Neutropaenia 1
Corynebacterium sp	1		

Table 2. LabDOSS reports of blood isolates for January 1992[1], continued

Organism	Number of Isolates	Clinical Information	Risk Factors
GRAM NEGATIVE BACTERIA			
Escherichia coli	22	LRT11Gastrointestinal 3, UTI 6,	Diabetes 1, IV central line 1, Malignancy 2, Transplant 1, HIV infection 1
Klebsiella oxytoca	1	Gastrointestinal 1	
Klebsiella pneumoniae	4	UT11	Trauma 1
Proteus mirabilis	2	LRTI 1	
Proteus vulgaris	1		Urinary tract surgery 1
Gitrobacter sp.	2		Malignancy 1
Enterobacter cloacae	2		Transplant 1
Enterobacter sp.	5	Endocarditis (prosthetic valve) 1	IV central line 1, Preterm neonate 1, Other immunocompromised 1
Morganella morganii	1	Gastrointestinal 1	Abdominal surgery 1
Serratia liquefaciens	1		Malignancy
Pseudomonas aeruginosa	4	UTI (catheter, instrumentation) 1	Neutropaenia, Abdominal surgery 1
Pseudomonas fluorescens	1		Neutropaenia 1
Xanthomonas maltophilia	2	Gastrointestinal 1	IV central line 1
Pseudomonas sp.	1		Neutropaenia 1
Salmonella typhi	1		
Aeromonas hydrophila	1		Preterm neonate 1
Haemophilus influenzae (not typable)	2	LRTI 2	
Haemophilus influenzae type b	2	Skin/cellulitis/wound 1	Diabetes 1
Neisseria meningitidis group B	1		
Neisseria sicca /subflava	1	Thyroglossal cyst 1	
Acinetobacter sp	1		Preterm neonate 1
Flavobacterium sp	1	Gastrointestinal 1	Other vascular prosthesis 1
Cardiobacterium hominis	1	Endocarditis (native value) 1	
ANAEROBES			
Bacteroides fragilis	1	Gastrointestinal 1	Malignancy 1
Peptostreptococcus sp.	1		
Clostridium perfringens	1		
FUNGI			
Candida albicans	1		
Torulopsis glabrata	1		Transplant 1
Total	119		

1. LRTI - Lower respiratory tract infection/pneumonia
UTI - Urinary tract infection

Table 3. LabDOSS CSF isolates and meningitis reports for January 1992

Organism	Number of Isolates	Age (years)	Source Specimen	Risk Factors
Cryptococcus neoformans	1	31	Blood	HIV infection
Staphylococcus epidermidis	1	25	CSF	Neurological surgery
Klebsiella oxytoca	1	25	CSF	Neurological surgery
Acinetobacter calcoaceticus	1	34	CSF	Neurological surgery

Table 4. LabDOSS reports from other sterile sites for January 1992

Site	Organism	
Peritoneal dialysate	*Pseudomonas aeroginosa*	1
	Staphylococcus epidermidis	1
	Candida tropicalis	1
Peritoneal fluid	*Staphylococcus aureus*	1

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Table 5. Diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation

DISEASES	ACT	NSW[4]	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
									This Period 1992	This Period 1991	Year to Date 1992[1,3]	Year to Date 1991
Diphtheria	0	0	1	0	0	0	0	0	1	1	1	1
Measles	2	27	0	2	2	0	4	0	37	37	90	79
Mumps	NN	2	NN	NN	NN	NN	0	NN	2	NN	2	NN
Pertussis	NN	3	0	6	3	0	3	4	19	18	45	48
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0
Rubella[2]	0	1	0	4	1	0	2	0	8	12	31	33
Tetanus	0	0	0	NN	0	0	0	0	0	1	0	1

1. 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
2. NT, Tas, WA: CRS only; ACT, NSW, Qld: rubella only; SA, Vic: rubella and CRS
3. NSW data only upto 31 January 1992.
4. Data for January 1992.

NN Not Notifiable.

Table 6. Other Notifiable Diseases[1]

DISEASES	ACT	NSW[10]	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
									This Period 1992	This Period 1991	Year to Date 1992[2,11]	Year to Date 1991
Arbovirus infection (NEC)[3]	0	0	NN	6	0	0	0	0	6	34	13	61
Ross River virus infection	NN	2	1	69	-	NN	5	14	91	185	264	297
Dengue	NN	0	0	0	-	NN	0	NN	0	0	1	0
Campylobacteriosis[4]	NN	-	12	124	41	24	48	3	252	227	948	615
Chlamydial infection (NEC)[5]	0	NN	2	124	0	13	28	0	167	130	541	376
Donovanosis	0	NN	0	2	NN	NN	0	0	2	0	2	4
Gonococcal infection[6]	0	11	10	15	0	0	9	18	63	100	202	199
Haemophilus influenzae type b[7]	NN	4	NN	3	0	2	0	NN	9	5	36	21
Hepatitis A	0	47	1	13	0	0	9	3	73	40	169	98
Hepatitis B	0	74	0	26	0	1	36	8	145	118	385	319
Hepatitis C	NN	46	NN	52	NN	1	28	NN	127	74	731	220
Hepatitis (NEC)	NN	2	0	19	0	0	0	NN	21	1	61	6
HIV infection[8]	0	0	0	0	0	0	0	0	0	0	7	1
Legionellosis	NN	2	0	0	0	0	1	0	3	4	4	6
Leptospirosis	0	3	0	1	0	0	1	0	5	2	19	4
Listeriosis	NN	0	NN	0	1	0	0	0	1	1	2	4
Malaria	0	1	0	6	0	0	1	2	10	40	58	80
Meningococcal infection	0	2	0	1	0	0	0	0	3	10	18	33
Ornithosis	0	NN	0	0	1	0	0	0	1	2	5	3
Q fever	0	1	0	14	0	0	0	0	15	12	30	31
Salmonellosis (NEC)	0	52	17	96	7	6	17	9	204	225	553	525
Shigellosis[4]	0	0	4	1	1	0	0	6	12	34	62	77
Syphilis	0	14	7	30	0	0	2	4	57	78	177	206
Tuberculosis	0	8	0	0	3	0	0	1	12	18	31	42
Typhoid[9]	1	4	0	0	1	0	0	1	7	1	8	2
Yersiniosis[4]	NN	-	0	16	7	0	0	0	23	11	66	41

1. For rarely notified diseases, see Table 7.
2. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of notifications and the increment in the cumulative figure from the previous period.
3. NSW and SA: includes Ross River virus and dengue.
4. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
5. ACT: trachoma only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. SA: only as 'bacterial meningitis'; meningococcal infection is separately notified; Tas: only as 'non-meningococcal meningitis'; Vic: eppiglottitis and meningitis only.
8. More complete data on new diagnoses of HIV infections are presented in the monthly *Australian HIV Surveillance Report*. ACT: AIDS only.
9. NSW, SA and Vic: includes paratyphoid.
10. Data for January 1992.
11. NSW data only upto 31 January 1992.

NN Not Notifiable.

NEC Not Elswhere Classified.

\- Elsewhere Classified.

Table 7. Rarely Notified Diseases[1]

DISEASES	Total this period	Reporting States or Territories	Total for 1992 to Date
Botulism			0
Brucellosis			0
Cholera			0
Chancroid			0
Hydatid infection			0
Leprosy			2
Lymphogranuloma venereum			1
Plague			0
Rabies			0
Yellow fever			0
Other viral haemorrhagic fevers			0

1. Fewer than 50 cases of each of these diseases were notified each year during the period 1986 to 1991.

Table 8. Laboratory reports by State or Territory of reporting laboratory for the reporting period (29 January to 11 February 1992), historical data[1] and total reports for the year

	STATE OR TERRITORY OR REPORTING LABORATORY						Total This Period	Historical Data[1]	Total Reported This Year
	ACT	NSW	Qld	SA	Vic	WA			
MEASLES, MUMPS, RUBELLA									
Measles virus	3	1	2	3	1		10	9.8	48
Mumps virus		2			1		3	1.8	7
Rubella virus		1	3	1	1	4	10	16.7	52
HEPATITIS VIRUSES									
Hepatitis A virus	1	6	1			2	10	11.5	56
Hepatitis B virus	3	19	16	8	12	13	71	105.8	364
Hepatitis C virus	6	1		38		33	78	2.8	309
Hepatitis D virus						1	1	.2	4
ARBOVIRUSES									
Ross River virus		1	23			8	32	17.0	73
Barmah Forest virus			5				5	.0	6
Dengue type 1			1				1	.0	2
Dengue type 2						1	1	.0	1
Dengue not typed			1				1	.2	3
Kunjin virus			3				3	.0	3
Kokobera virus			1				1	.0	1
Stratford virus			2				2	.0	3
Flavivirus (unspecified)			2				2	.8	3
ADENOVIRUSES									
Adenovirus type 1		4					4	6.7	23
Adenovirus type 2		2			1		3	7.0	24
Adenovirus type 4					1		1	3.7	2
Adenovirus type 8					1		1	1.0	6
Adenovirus type 22					1		1	.0	2
Adenovirus type 24		1					1	.0	1
Adenovirus type 28					1		1	.0	3
Adenovirus not typed/pending		5	10	3	9	9	36	24.3	165
HERPES VIRUSES									
Herpes simplex virus type 1		3	39	10	43	28	123	105.8	649
Herpes simplex virus type 2		13	27	5	28	42	115	139.8	757
Herpes simplex not typed/pending	5	8	4	2		5	24	72.3	131
Cytomegalovirus		15	30	1	29	8	83	45.2	368
Varicella-zoster virus		9	3	2	6	6	26	14.0	120
Epstein-Barr virus		9	20	8	14	8	59	35.8	273
Herpes virus group - not typed					2		2	8.2	17
OTHER DNA VIRUSES									
Parvovirus					8		8	.0	30
PICORNA VIRUS FAMILY									
Coxsackievirus B4						1	1	4.0	5
Echovirus type 4					1		1	.2	3
Echovirus type 11		1			1		2	2.7	4
Echovirus type 16					4		4	.0	6
Echovirus type 22					1		1	.7	1
Poliovirus not typed/pending		8					8	2.3	14
Enterovirus not typed/pending		9	15		1	4	29	21.5	160
Rhinovirus (all types)		3		1	12	1	17	17.2	131

Table 8. Laboratory reports by State or Territory of reporting laboratory for the reporting period (29 January to 11 February 1992), historical data[1] and total reports for the year, continued

	STATE OR TERRITORY OR REPORTING LABORATORY						Total This Period	Historical Data[1]	Total Reported This Year
	ACT	NSW	Qld	SA	Vic	WA			
ORTHO/PARAMYXO VIRUSES									
Influenza A virus			1			1	2	2.3	18
Influenza B virus			3	1	1	1	6	1.5	25
Parainfluenza virus type 1					6	5	11	3.0	25
Parainfluenza virus type 2		1		1			2	1.5	9
Parainfluenza virus type 3		1	2	1	14	1	19	14.5	95
Parainfluenza virus typing pending					2		2	1.2	13
Respiratory syncytial virus			2	1	4	1	8	6.3	92
OTHER RNA VIRUSES									
Rotavirus		3		2	4	1	10	15.8	230
Coronavirus		1					1	.0	8
Calici virus		2					2	.3	7
Small virus (like) particle		2					2	1.3	7
HIV-1						1	1	3.0	3
OTHER									
Chlamydia trachomatis (unspecified)	3	13	51	13	24	39	143	142.8	519
Chlamydia psittaci (ornithosis)			1		12		13	2.7	30
Mycoplasma pneumoniae		4	5	5	10	11	35	16.3	115
Coxiella burnetii (Q fever)		10	7		2		19	4.3	42
TOTAL	21	158	280	106	258	235	1,058	896.0	5,068

1. 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 preceeding and following thoses.

Table 9. Laboratory reports by clinical information for the reporting period 29 January to 11 Feburary 1992

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/Unknown	Total
MEASLES, MUMPS, RUBELLA													
Measles virus								7				3	10
Mumps virus		1										2	3
Rubella virus								4				6	10
HEPATITIS VIRUSES													
Hepatitis A virus							7					3	10
Hepatitis B virus							24					47	71
Hepatitis C virus							9					69	78
Hepatitis D virus							1						1
ARBOVIRUSES													
Ross River virus	1		1					5		13		12	32
Barmah Forest virus												5	5
Dengue type 1												1	1
Dengue type 2												1	1
Dengue not typed												1	1

Table 9. Laboratory reports by clinical information for the reporting period 29 January to 11 Feburary 1992 continued

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/Unknown	Total
Kunjin virus												3	3
Kokobera virus												1	1
Stratford virus												2	2
Flavivirus (unspecified)												2	2
ADENOVIRUSES													
Adenovirus type 1					3							1	4
Adenovirus type 2					1							2	3
Adenovirus type 4									1				1
Adenovirus type 8									1				1
Adenovirus type 22												1	1
Adenovirus type 24												1	1
Adenovirus type 28						1							1
Adenovirus not typed/pending	1				14	8			1			12	36
HERPES VIRUSES													
Herpes simplex virus type 1					11			79	5		25	3	123
Herpes simplex virus type 2								52			58	5	115
Herpes simplex not typed/pending					3			17			2	2	24
Cytomegalovirus			1	2	15	2	4	1	5	1	2	50	83
Varicella-zoster virus	1							21				4	26
Epstein-Barr virus					8	1	4	1		1		44	59
Herpes virus group - not typed								1				1	2
OTHER DNA VIRUSES													
Parvovirus								3		2		3	8
PICORNA VIRUS FAMILY													
Coxsackievirus B4			1										1
Echovirus type 4		1											1
Echovirus type 11					1							1	2
Echovirus type 16		1			1	1						1	4
Echovirus type 22												1	1
Poliovirus not typed/pending						7						1	8
Enterovirus not typed/pending		3	3		6	10		1				6	29
Rhinovirus (all types)					15							2	17
ORTHO/PARAMYXOVIRUSES													
Influenza A virus												2	2
Influenza B virus					3							3	6
Parainfluenza virus type 1					9							2	11
Parainfluenza virus type 2					2								2
Parainfluenza virus type 3					14	1						4	19
Parainfluenza virus typing pending					2								2
Respiratory syncytial virus					8								8
OTHER RNA VIRUSES													
Rotavirus					1	7						2	10
Coronavirus						1							1
Calici virus						2							2
Small virus (like) particle						2							2

Table 9. Laboratory reports by clinical information for the reporting period 29 January to 11 Feburary 1992, continued

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/Unknown	Total
HIV-1												1	1
OTHER													
Chlamydia trachomatis (unspecified)									6		114	23	143
Chlamydia psittaci (ornithosis)					12							1	13
Mycoplasma pneumoniae					26			1		1		7	35
Coxiella burnetii					1		1					17	19
TOTAL	3	6	6	2	156	43	50	193	19	18	201	361	1,058

Table 10. Laboratory reports by contributing laboratories - total reports received for the reporting period 29 January to 11 Feburary 1992

STATE	LABORATORY	Reports
Australian Capital Territory	Woden Valley Hospital, Garran	21
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	113
	Prince Henry/Prince of Wales Hospitals, Sydney	32
	Royal Alexandra Hospital for Children, Camperdown	13
Queensland	State Health Laboratory, Brisbane	280
South Australia	Institute of Medical & Veterinary Science, Adelaide	106
Victoria	Fairfield Hospital, Melbourne	189
	Microbiological Diagnostic Unit, University of Melbourne	24
	Royal Childrens Hospital, Melbourne	45
Western Australia	Princess Margaret Hospital, Perth	24
	State Health Laboratory Services, Perth	211
TOTAL		1058