Editorial
Measles elimination – a case definition to enhance surveillance

The end of the 20th century was greeted by a ground-swell of optimism that measles eradication was theoretically and technically feasible with the tools already available.[1,2]

This enthusiasm was galvanised by the reduction in measles incidence and mortality in many parts of the world, and progress towards elimination of indigenous measles transmission in the Americas.[3] The latter used the strategy of combining a single mass 'catchup' campaign for children 9 months to 15 years of age with high coverage through routine vaccination of infants, plus intensive surveillance and follow-up campaigns to prevent excessive build-up of susceptible individuals.[4]

In Australia, progress towards measles elimination has recently seen a shift from an 'outbreak control' phase to an 'elimination phase' with the publication of supportive comprehensive guidelines;[5] this is encouraging. Strategies accompanying this change in policy are similar to those adopted in the Americas and include modification of the vaccination schedule to improve coverage rates through earlier routine two-dose childhood vaccination, a once-off school-based mass campaign in 1998, protection of high-risk groups, and rapid response to outbreaks.

Improved surveillance is necessary to demonstrate the termination of wild virus circulation and to detect outbreaks.[6] This is of particular relevance to Australia as this country will remain vulnerable to importation of disease from countries where measles virus continues to circulate. The threat of measles outbreaks in people aged 16-30 years (the group not captured during mass vaccination campaigns and not enjoying immunity due to exposure to wild measles virus) has recently been recognised.[7,8] Thus the commitment to achieving measles elimination demands an intensified surveillance strategy so that outbreaks and importation of infection can be detected rapidly, and timely intervention initiated.

Enhanced surveillance has a number of components, including assuring the availability and accessibility of a laboratory network capable of serologically confirming all suspected measles cases.[9] This is important because, as elimination approaches, clinical 'measles-like' cases will more often be other rash-associated conditions, particularly those due to viruses, such as rubella, parvovirus B19, dengue, and human herpesvirus-6.[10,11,12] The value of the laboratory system is, however, dependent on the ability of the health system to detect all possible measles cases and submit appropriate specimens for confirmation. This demands a high level of awareness amongst health personnel of the need for immediate

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detection and reporting of measles-compatible clinical disease.

As the costs of missing a single measles case can be enormous, the phase of elimination demands a case-definition with a sensitivity approaching 100 per cent. The selected case-definition should also be standardised and unambiguous.[13]

The case-definition included in the current *Guidelines for the control of measles outbreaks in Australia* defines a suspected measles case as *'an illness with all of the following features: morbilliform rash, cough and fever present at the time of rash onset'*.[5] This case-definition has three potential attendant problems. Firstly, the description of the rash as 'morbilliform' is tautological since the term means 'measles-like'. However, this semantic irritation is not as important as the second potential consequence of describing the rash as 'morbilliform'. Historically, when measles was common, most clinicians would have recently seen the typical maculopapular skin rash and thus would have been able to recognise it reliably. As measles has become less common, many clinicians will fail to recall accurately features of the measles rash or may have never seen it; thus describing a rash as 'morbilliform' will be of dubious value.[14,15,16]

The third problem inherent in the present Australian case-definition is the mandatory inclusion of cough before clinical illness may be considered as '*possibly due to measles'*. This appears to be largely based on a study of 49 patients notified to the Eastern Sydney Public Health Unit with a clinical diagnosis of measles. In this study the Centers for Disease Control and Prevention (CDC) case-definition had a high sensitivity (92%) but a low specificity (24%) due to the false-positive rate of 51 per cent.[17] By including cough as a prerequisite for a modified measles case-definition, specificity was increased to 57 per cent but with no apparent change in sensitivity. However, the conclusion that there was no change may be invalid as their new proposed case-definition was applied to patients detected using the CDC definition and any statements on sensitivity should thus be guarded.[18]

The clinical picture of measles in older children and adults may differ from younger children, and measles is often more severe in older children.[19,20] With the changing epidemiology of measles susceptibility in Australia, to ensure a sensitive case-definition, attention needs to be paid to the clinical presentation in older individuals.

The precedent for evolving a measles case-definition towards greater sensitivity has already been set. Although the original measles case-definition for national surveillance in the United States of America was *'an illness characterised by all of the following features: a generalised maculopapular rash lasting three or more days; and a fever exceeding 38.3^{0}C; and cough or coryza or conjunctivitis'*, the Immunization Practices Advisory Committee has since recommended that all rash illness with fever should be investigated as possible measles.[21] Similarly, although the World Health Organization clinical measles case definition includes *'any person with: fever, and maculopapular (ie. non-vesicular) rash, and cough, coryza (ie. runny nose) or conjunctivitis (ie. red eyes*)', health care workers are instructed to suspect measles infection and respond accordingly in all patients presenting with fever and generalised maculopapular rash.[2,22] This definition is similar to that used in Mpumalanga Province, South Africa, where a public health response is catalysed by *'any patient with fever and a non-blistering generalized skin rash*'.[23]

Surveillance is rightly recognised as 'the key to eradication' of measles.[24] Thus, as elimination is neared in Australia, to prove the absence of indigenous measles and to contain transmission from any imported case, it may be necessary to change the current case-definition. This may indeed formalise the working approach already prevalent in certain States and Territories. Such a modified case-definition should have maximal sensitivity for detecting possible measles cases and be unambiguous. An enhanced case-definition that is unambiguous and captures all cases eg '*fever and generalised maculo-papular rash'* or *'fever and non-blistering generalised skin rash*', may be necessary to achieve this. We believe that such a case definition would increase the sensitivity for detecting cases of measles and that the decreased specificity due to such a case definition would be justified.

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Letter to the Editor

Guidelines for the control of measles outbreaks in Australia

Robert Hall

Acting Chair, Measles Elimination Advisory Committee

To the Editor: In response to Dr Christine Selvey's letter to the Editor[1] the Measles Elimination Advisory Committee (MEAC) would like to provide the following comment.

The definition of a susceptible person that was published in the Guidelines for the *Control of Measles Outbreaks in Australia* (2000) [2] could be interpreted to be inconsistent in the situation proposed by Dr Selvey. Therefore, MEAC agrees that practitioners should consider offering infants under 6 months of age immunoglobulin if:

- the exposure was to a confirmed case of measles, as defined in section 2.1 of the Guidelines; and
- the mother is retrospectively assessed to have been susceptible to measles at the infant's time of birth.

Such a situation is rare at present but might become more common in future.

Guidelines such as *Guidelines for the Control of Measles Outbreaks in Australia* cannot provide recommendations to cover every situation. Therefore, it is important for public health practitioners to consider recommendations in light of the clinical context and to be prepared to exercise a degree of clinical judgement in their implementation (refer to page xvii of the Guidelines).

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Enhanced surveillance for meningococcal disease in Queensland in 1999

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Abstract

Enhanced surveillance of invasive meningococcal disease commenced in Queensland in 1999. There were 93 cases, an incidence of 2.8/100,000 population. Most (87%) cases were laboratory confirmed, but 12 per cent were probable cases without laboratory confirmation. The highest age-specific attack rates were in the under 1, 1 to 4 and 15 to 24 year age groups. Most of the serologically characterised isolates were group B (70%), followed by group C (24%). There were 12 deaths, resulting in a case fatality rate of 13 per cent. Those who died were more likely to have group C than group B disease (OR 5.04, CI 1.05-25.14). Only 14 per cent of cases that saw a general practitioner (GP) prior to hospitalisation received parenteral antibiotics, 23 per cent of the 35 cases referred to hospital by a GP received pre-hospital parenteral antibiotics and 33 per cent of cases were notified to health authorities within 24 hours of hospital admission. Thirty per cent were notified two or more days after hospitalisation, delaying the start of public health action. Enhanced surveillance has demonstrated a need to promote the use of pre-hospital parenteral antibiotics by GPs and a need to encourage more timely reporting of cases to health authorities. *Commun Dis Intell* 2000;24:332-336.

Keywords: Neisseria meningitidis*, diagnosis, meningococcus, invasive, enhanced surveillance, indigenous, parenteral antibiotics*

Introduction

Before 1999, only laboratory-confirmed cases of invasive meningococcal disease (IMD)* were routinely notified in Queensland. Because the probable clinical cases (with no laboratory confirmation) were not notified, an under-ascertainment of the true incidence of IMD resulted. Furthermore, important information (such as the indigenous status, the clinical and public health management, and the clinical outcome of each patient) was not routinely collected.

From the beginning of 1999, public health physicians implemented 'enhanced' surveillance of IMD in Queensland, which not only included probable clinical cases, but also extra details on each case. In this report we describe (i) the epidemiology of IMD in Queensland in 1999, (ii) the relevant details of the clinical and public health management of the cases, and (iii) the completeness of the information collected through the enhanced surveillance.

Methods

A standard surveillance report form for IMD, based upon that used in New Zealand[1] was developed for use by Public Health Units (PHUs) throughout Queensland. A case of IMD was defined as:-

- Confirmed, if there was a clinically compatible illness and at least one of: (i) isolation of *Neisseria meningitidis* from a normally sterile site, or (ii) detection of gram-negative diplococci in a specimen from a normally sterile site, or (iii) a positive meningococcal antigen test on cerebro-spinal fluid (CSF) or (iv) detection of meningococcal DNA in a specimen from a normally sterile site.
- Probable, if there was a clinically compatible illness and at least one of: (i) a petechial or purpuric rash, or (ii) isolation of *N. meningitidis* from a throat swab,[2] (iii) an epidemiological link to a confirmed case.

'Disease onset' for the calculation of timeliness of hospitalisation refers to this episode of illness, and includes the spectrum of disease from those who were mildly unwell initially to those with classic meningococcal disease.

Where possible the PHUs obtained the information detailed on the report form from the patient, the immediate contacts or the clinicians. Data from completed forms forwarded to the Communicable Disease Unit (CDU), Queensland Health, Brisbane, were entered on a computerised database.

Pathology laboratories throughout Queensland refer meningococcal isolates to Queensland Health Scientific Services (QHSS), Brisbane, for confirmation of the serogrouping and for further phenotypic characterisation. These laboratory data were also sent to the CDU for entry on its database.

The database was analysed using Epi Info version 6.04b and Windows Excel 97. Data from the 1996 national census were used to calculate incidence rates.

Results

Epidemiology and clusters

In all there were 93 cases of IMD in Queensland in 1999, an incidence of 2.8/100,000 population. The median age was 16.3 years (range 0.2 to 84.4 years) and the male:female

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ratio was 1:1.1. The highest number of cases occurred in the under 1 and 1 to 4 years age groups (11.8% and 22.6% of cases respectively). There was a secondary peak in the 15 to 19 years age group that extended into the 20 to 24 years age group (Figure 1).

A typical winter peak was exhibited in 1999 with 48.4 per cent of cases occurring between May and August. There was no clustering by town or suburb in 1999. Indigenous status was recorded in 81 (87%) of cases. Six of these cases (7.4%) were indigenous, all children under 15 years of age (four cases were under 5 years).

There were two recognised clusters in 1999. The first in May 1999 was of serogroup B disease among three people. Two had been passengers in the same train on the same date and the third case was a contact of one of the travellers. There was no known direct contact between the two train passengers but one was sitting behind the other for approximately 24 hours. The first two were co-primary cases; the third became ill 11 days after the primary cases. Analysis of the DNA of isolates using pulsed field gel electrophoresis (PFGE) confirmed that these cases were linked.

The second cluster in July 1999 was two cases of serogroup C disease, also confirmed by PFGE. The second case had illness onset 3 days after the first case and was a contact of a contact of the first case.

Laboratory diagnosis

Of the 93 cases, 81 (87%) were confirmed, 11 (12%) were probable and one could not be categorised due to missing data. Of the confirmed cases, 67 were culture-positive; 4 were confirmed by PCR alone, 6 by detection of gram-negative diplococci in blood or CSF alone and 4 by a combination of PCR, antigen detection or organism detection. All 11 probable cases were diagnosed by a clinically compatible illness in association with a rash.

All 67 isolates were serogrouped; 47 (70%) were group B, 16 (24%) were group C and there were 2 each of group Y and W135. Of the 11 isolates from fatal cases, 6 were group C and 5 were group B.

Forty (63.5%) of the 63 group B and C isolates could be further serotyped. No strain predominated among the group C isolates. The most commonly isolated strains among the group B isolates were 4:P1.4 (7) (n=5) and 15:P1.7 (n=5).

Serogroup B infections outnumbered serogroup C infections in all age groups, with the ratio being highest in the under one-year age group (Figure 2).

Clinical features, clinical management and outcomes

Twenty-one of the 93 cases (22.6%) were recorded as having both meningitis and septicaemia, 19 cases (20.4%) presented with septicaemia alone, 20 (21.5%) with meningitis alone and 2 (2.2%) with septic arthritis alone. A petechial or purpuric rash occurred in 57 (61%) of presentations. All 12 cases that died presented with septicaemia and 5 of these also had a meningitic component.

The 12 deaths from IMD represented a case fatality of 13 per cent, with the highest proportion of case fatalities in the 20 to 29 years (29%) and 1 to 4 years (19%) age groups. The median age of those dying was 19 years, compared with 14.9 years for those surviving.

Figure 1. Number of meningococcal cases, Queensland, 1999, by age group and sex

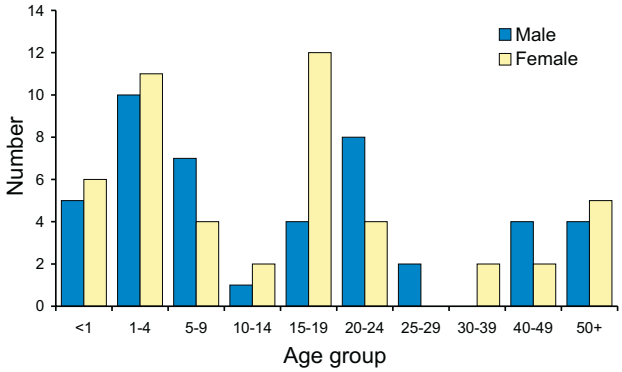
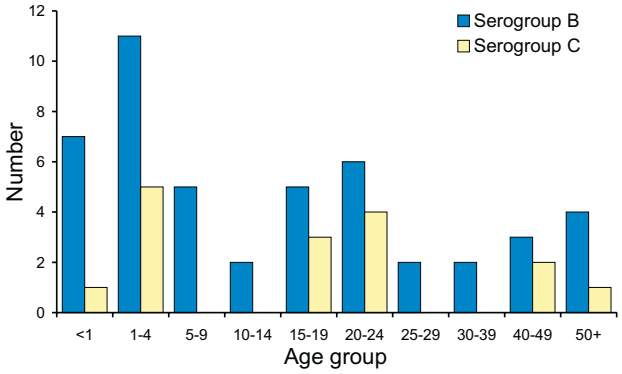


Figure 2. Number of meningococcal cases, Queensland, 1999, by age group and serogroup



Those with serogroup C disease were more likely to die than those with serogroup B disease (OR 5.04, 95% CI 1.05-25.14, p = 0.02) and those with a rash were more likely to die than those without (OR 5.96, 95% CI 0.7-133.2, p = 0.09). Those who saw a general practitioner (GP) prior to hospitalisation were less likely to die than those who did not (OR 0.24, 95% CI 0.05 -1.01, p = 0.05).

Information on which cases saw a GP prior to hospitalisation was available in 89 cases; 56 (63%) saw a doctor, of these 8 (14%) were recorded as having received parenteral antibiotics. Information on which cases were referred to hospital by a GP was available in 86 cases; of these, 35 (41%) were referred to a hospital. If only those cases referred to hospital by the GP (ie. those in whom the GP probably suspected meningococcal disease) are used as the denominator, the percentage of cases receiving pre-hospital parenteral antibiotics increased to 23 per cent (8/35).

Meningococci were isolated from 2 (25%) of the 8 cases who received pre-hospital parenteral antibiotics (p = 0.05) and from 55 (67%) of the 82 cases who did not.

Table 1. Days from general practitioner visit and meningococcal disease onset to hospitalisation, and from hospitalisation to notification, Queensland 1999

	Days									
Time interval	<1	1	2	3	4	5	6	7	NR*	Total cases
From GP visit to hospital admission	36	12	5	0	1	0	0	0	2	56
From disease onset to hospital admission	23	35	14	5	2	2	2	2	8	93
From hospitalisation to notification (probable)	31(3)	27(5)	15(1)	5	1	3(1)	0	1	10(1)	93(11)

* Not recorded

Of the 56 cases who saw a GP prior to hospital admission, 36 (64%) were admitted within 24 hours of being seen (Table 1) while 18 (32%) took one day or more to be hospitalised. Of the 12 who died, 4 had been seen by a GP prior to hospitalisation, and of these 3 were referred to hospital; one had been treated pre-admission with parenteral antibiotics.

Fifty-eight (62%) of the cases were hospitalised in less than 48 hours from disease onset (Table 1). The median number of days between disease onset and hospitalisation was one day for both those who survived and those who died.

Eleven of 78 cases (14.1%) had a throat swab taken at the time of the first dose of antibiotics and 67 of 85 cases (78.8%) had blood cultures taken. Eighteen cases had neither blood cultures nor throat swabs taken before antibiotic administration.

Public health management

Information on time taken for notification was available in 83 (89%) of cases; in 25 (30%) of these it took two or more days for the local PHU to be notified of the hospitalisation (Table 1). In all but one of 40 cases for whom there was enough information to calculate response time this was one hour or less.

In most episodes, household contacts were the main type of contact (n = 77), followed by contact with oral secretions (n = 34), other close contacts (n = 26), institutional (n = 9) and childcare (n = 5) contacts. On average, 11.7 contacts per case were eligible for chemoprophylaxis; none was offered vaccine in 1999.

Table 2 lists the percentages of missing data by section (excluding the section on laboratory details for which data are obtained from reference laboratories). In general, fields that related to time (eg. time of initial response, time of onset, time seen by GP, time hospitalised) had the highest percentage of missing data. Indigenous status is an important field for which there were 12 entries missing.

Table 2. Mean percentages of data missing from surveillance report forms, by section

Section	Mean missing data (%)	Range (%)
Notifier details	21.4	2.2 - 45.2
Patient details	13.1	0 - 72.0
Clinical presentation	15.1	7.5 - 23.7
Status (confirmed/probable)	9.7	
Clinical course	13.1	0 - 46.2
Case management	13.2	0 - 16.1
Risk factors	10.4	0 - 50.0
Outbreak details (Yes/No)	11.8	

Discussion

At 2.8/100,000, the incidence of IMD in Queensland in 1999 was similar to that in Australia in 1998 (2.4/100,000).[3] This contrasts with the considerably greater incidence in England and Wales (5/100,000 in 1998), and New Zealand (13.9/100,000 in 1999).[4] The latter extremely high incidence reflects the sustained epidemic of serosubtype B:4:P1.4 disease in New Zealand.[4] This strain is also present in Queensland, although the numbers are small and have remained similar since 1996.[5]

The incidence of IMD by age group and season was typical of that generally described in developed countries. Most cases were sporadic, with only two confirmed clusters. At 7.4 per cent, the proportion of meningococcal disease among those identified as indigenous was high compared with the proportion of indigenous people in the general population (2.8%). Monitoring of IMD trends in the indigenous population needs to continue.

The diagnosis of meningococcal disease was confirmed in most cases by isolation of the organism from blood or CSF. The proportion of positive cultures was reduced in those who had received pre-hospital antibiotics. With the continued emphasis on the importance of pre-hospital antibiotics, and the increasing reluctance of clinicians to undertake lumbar puncture, non-culture diagnostic tests (in particular PCR) will increasingly be required to confirm a diagnosis of IMD. In addition to confirming the presence of meningococcal DNA, PCR testing has the capability to determine serogroups, serotypes and serosubtypes from clinical specimens.[6]

The hallmark of meningococcal septicaemia is a haemorrhagic rash that does not blanch under pressure.[7] Modelled on initiatives in England and New Zealand, for the first time in 1999 all GPs and emergency department physicians in Queensland were sent pictorial information depicting the typical rash of meningococcal disease. A rash was present in 57(66%) of cases, and all 12 patients dying

had septicaemia. GPs were urged to administer parenteral penicillin to anyone displaying the symptoms and signs of meningococcal septicaemia. Despite this, only 23 per cent of the cases referred to a hospital by a GP received pre-hospital parenteral antibiotics. The questionnaire did not record whether the GP suspected IMD, a question now included in the latest version.

Recent experience in New Zealand has documented a significantly lower case fatality in patients given antibiotics prior to hospitalisation.[4] Numbers reported here were too small to show this. Although other determinants of disease outcome, such as the severity at initial presentation, could influence the association between pre-hospital antibiotic use and case fatality, the early administration of antibiotics is generally considered the most important means to prevent IMD deaths. Further initiatives are needed to get GPs to administer parenteral penicillin to possible IMD cases, and monitoring of the proportion given pre-hospital antibiotics should continue.

Seventy per cent of isolates in Queensland in 1999 were serogroup B, not dissimilar to the proportion reported nationally in 1998 and 1999 (63%).[3,8] Queensland differed from Victoria and New South Wales in 1999 in that group C disease did not predominate in the 15-44 age group.[8] Although the small number of cases meant that the confidence intervals were wide, the group C case fatality rate (CFR) was significantly higher than that for group B disease. National data for 1999 were similar (CFR in group C disease was 14.9 per cent compared with 6.4 per cent in serogroup B disease).[8] As an effective vaccine against this serogroup is available, these deaths are potentially preventable and decisions about whether or not to introduce routine vaccination need to be reviewed continually.[9]

Because the risk of secondary cases is much greater in the days immediately following the onset of the index cases than later on, immediate notification of suspect cases of IMD to PHUs is crucial so that contacts can be identified, counselled and offered chemoprophylaxis if indicated.[10] That only 33 per cent were notified within 24 hours of hospital admission, and 30 per cent notified two or more days after hospitalisation, is thus of considerable concern. Late reporting may indicate some clinicians await laboratory confirmation before notification. Further efforts to get clinicians to notify cases upon clinical suspicion are required.

Public health physicians agreed unanimously that IMD be the first disease for enhanced surveillance in Queensland. Therefore the amount of missing information is both surprising and disappointing. Some fields, such as 'indigenous status' and 'outcome' were considered important enough to justify enhanced surveillance. High proportions of missing data in some fields (Table 2) may reflect a need to clarify the form, but PHUs should attempt to ensure that the enhanced surveillance report forms are completed with reliable detail. Further monitoring of the completeness of information collected by PHUs is necessary.

In conclusion, the first year of enhanced surveillance in Queensland has already demonstrated the need to improve (i) GP awareness of the diagnosis of IMD, and the effectiveness of parenteral administration of antibiotics prior to the urgent referral to hospital, (ii) the timeliness of notification of IMD by clinicians, and (iii) the completeness of the information collected by PHUs on each case of IMD. Extra costs beyond those normally involved in collecting data for case management were minimal and mainly related to data entry and analysis.

Continued enhanced surveillance will enable detection of trends. However these may need to be interpreted with caution, given that increasing identification of cases by non-culture based methods means that increasing numbers of milder cases are likely to be uncovered.[8] Data pooled over several years will allow more meaningful analysis of risk factors and management practices, as would adoption of enhanced surveillance nationally.

*** Abbreviations:**

CDU, Communicable Disease Unit; CFR, case fatality rate; CSF, cerebrospinal fluid; GP, general practitioner; IMD, invasive meningococcal disease; PCR, polymerase chain reaction; PHU, Public Health Unit; PFGE, pulsed field gel electrophoresis; QHSS, Queensland Health Scientific Services.

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A waterborne outbreak of *Salmonella* Saintpaul

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Abstract

Contamination of a tank water supply system led to an outbreak of *Salmonella* Saintpaul with 28 cases of gastroenteritis amongst over 200 workers at a large construction site. The outbreak was identified following notification of two salmonellosis cases by general practitioners from different towns during March 1999. The source of infection, contaminated drinking water, was identified through environmental sampling and confirmed by epidemiological investigations. Frogs and/or mice may have been the original source of the contamination. This report details control measures, the results of investigations and recommendations for future research. *Commun Dis Intell* 2000;24:336-340.

Keywords: salmonellosis, waterborne, surveillance, frogs, mice

Introduction

Outbreaks due to *Salmonellae* are usually foodborne, though waterborne outbreaks have been reported. In the USA between 1993 and 1997,[1] 357 (54.5%) of the 655 foodborne outbreaks known to be due to bacteria were due to *Salmonellae*. Conversely, in the United States from 1993 to 1998, only one of fifteen (6.6%) of the reported gastroenteritis outbreaks involving drinking water due to bacteria was caused by *Salmonella*;[2,3,4] the likely source of that outbreak, due to *S*. Typhimurium, was one of a pair of storage towers inadequately protected against bird droppings.[2]

S. Saintpaul has been associated with foodborne outbreaks including one due to contaminated paprika.[5] *S.* Saintpaul usually accounts for about 12 per cent of all typed human salmonella isolates from the central Queensland area. According to National Salmonella Surveillance Scheme (NSSS) reports, it is consistently amongst the top 10 serovars in Australia, with Queensland accounting for around two thirds of the nation's total reported cases.

Lizards (including geckos) and other reptiles have been well documented as sources of salmonella,[6-8] and there are reports that amphibians such as frogs and toads are potential salmonella sources.[9-13] NSSS data on *S*. Saintpaul isolates from non-human sources in Queensland January 1990 to April 1999 show a wide range of animal sources, including reptilian, bovine, ovine, porcine, equine, canine, avian and marsupial species. (Personal communication, D Lightfoot, National Salmonella Surveillance Scheme, Microbiological Diagnostic Unit, University of Melbourne).

In this paper we report two laboratory-proven cases of salmonellosis due to *S*. Saintpaul in workers from an isolated construction site in Central Queensland. It was concluded that the source of the outbreak, which involved 28 cases in all, was probably a contaminated water supply in which live green tree frogs were found.

Methods and Results

Clinical investigation

Two cases of salmonellosis were notified by General Practitioners from towns 250 km apart, on 15 March and 24 March 1999. Both worked at the same site and both were faeces culture-positive for salmonella, later typed as *S*. Saintpaul. Telephone follow-up of the first suggested no associated cases, but follow-up of the second did, prompting an immediate site visit and further investigation.

Enhanced surveillance was carried out by asking general practitioners and hospital staff in the nearby town to sample and notify any further cases of gastroenteritis amongst construction site workers. However no further cases were notified by this means.

Environmental investigation

Although numbers fluctuated with the various stages of construction, at the time of this outbreak 212 workers were employed at a construction site in central Queensland. Many were accommodated in temporary living quarters about 2 kilometres away, where they were catered for by a camp kitchen.

The initial site visit (conducted on 25 March 1999) included environmental health assessments, interviews with identified cases and examination of absenteeism records supplied by three main subcontractors (A, B and C). These records highlighted an unusual number of employees off work during March with gastrointestinal illness. Preliminary assessment identified several potential sources of exposure. These included septic tanks that had overflowed in recent weeks, sharing of drinks (particularly 5 litre water bottles, from which workers drank directly), manual handling of ice for water bottles, tank-sourced drinking water, a mouse infestation, and sub-optimal lunch box storage facilities.

On 29 March, samples for bacteriological analysis were collected from two ice machines, three water fountains

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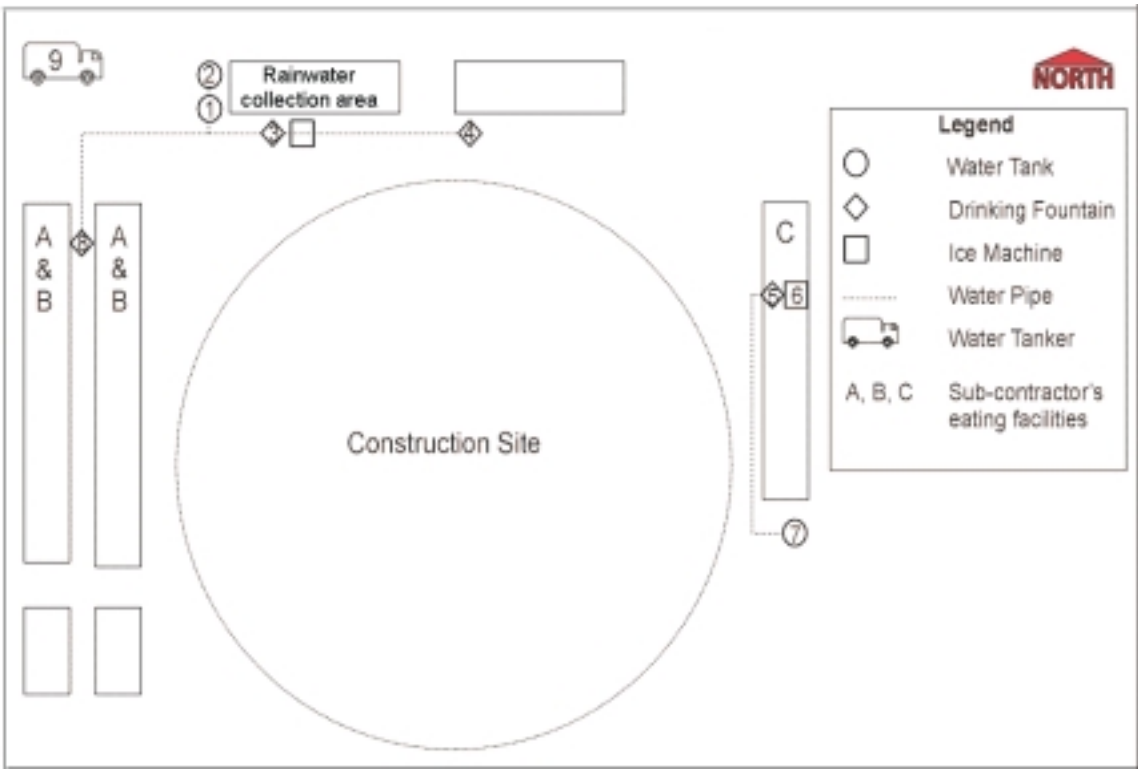
Figure 1. Site diagram showing water sampling locations, 8 April 1999



serving most on-site drinking water needs and food sold from the mobile food van servicing the site daily. More targeted water sampling was conducted, including specifically for *Salmonella* culture, on 8 April, 27 April and 26 May.

An assessment of water carted to the site was also conducted. The carter servicing the site obtained water from a reticulated water supply for which there were no indications of recent problems. Supplementary chlorine was added to it prior to delivery. Samples taken from the carter's tankers complied with NHMRC *Australian Drinking Water Guidelines*[14]. On delivery water was stored without further chlorination in six new 5,000-litre tanks located at three points around the construction site (Figure 1). Two of the six tanks (1 and 2) were interconnected and supplemented with rainwater collected from a workshop roof. One of these tanks had an uncovered inlet.

All ice and water samples taken from the site on 29 March (results available 8 April) failed to comply with NHMRC guidelines in terms of coliform counts. Some samples contained *E. coli* counts of up to 47 organisms/100 mL.

Repeat water samples taken on 8 April (results available 19 April) also showed contamination (Table 1). Other than site 9 (plate count <25 cfu) all sites had Standard Plate Counts greater than 500 cfu. *Salmonella* was cultured from samples taken from tank 2 and from two water fountains sourced from this tank. Subsequent typing identified the serovar as *S*. Saintpaul, the same as the human isolates. Water samples taken on 27 April demonstrated improved water quality whilst those taken 26 May showed no coliforms.

Food safety assessments were conducted on site and at the accommodation camp. These found the accommodation camp kitchen and mobile food van practices to be satisfactory. All food samples were of satisfactory bacterial quality.

Epidemiological investigations

A case-control study was initiated, with most controls being interviewed on 29 March using a modified Queensland Health Foodborne Illness questionnaire. Information was also sought about consumption and source of ice in water bottles, and usual sources of food.

A case was defined as any construction site employee who had suffered diarrhoea, or vomiting and abdominal cramps, or vomiting and fever, during the month of March. Controls were taken from unaffected construction site employees who worked at the site during this period. Data were analysed using Epi Info version 6.04c.

Table 1. Results of water sampling, 8 April 1999

Sample location (refer Figure 1)	Coliform count[1]	*E. coli* count[1]	*Salmonella* culture
1	> 80	23	- ve
2	+ ve[2]	> 48	+ ve
3	> 80	7	Not tested
4	> 80	16	+ ve
5	7	- ve	Not tested
6	5	1	Not tested
7	5	- ve	- ve
8	> 80	19	+ ve
9	- ve	- ve	- ve

1. Colony forming units (cfu) per plate.
2. Detected, but no count due to confluent growth.

Twenty-eight cases and 88 controls were interviewed. Most (if not all) cases were ascertained, so the attack rate was approximately 13 per cent. Illness occurred more frequently amongst employees of two of the three main subcontractor groups (firms 'A' and 'B' had a combined attack rate of 21 per cent, compared with an attack rate of 9 per cent in firm 'C'). Although some employees of all firms stayed at the accommodation camp, there was little on-site contact between employees of firms A and B with those of firm C, either during work or rest breaks. Firms A and B shared lunch areas, ablution facilities and sources of ice that were separate from those of firm 'C' (Figure 1). Gastroenteritis had occurred in both accommodation camp residents and workers living off-site in the nearby town. There were no reported cases in the families of employees and no evidence of secondary spread.

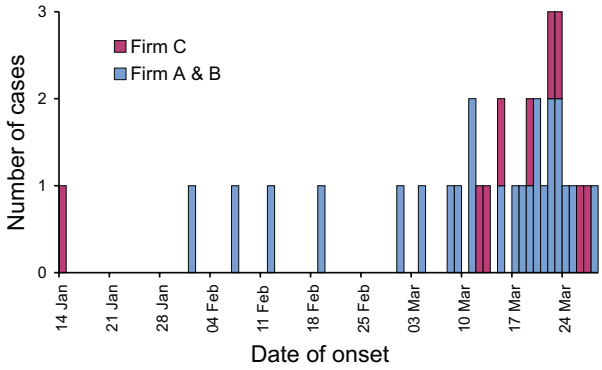
The association between illness and the use of an ice machine was striking, pointing to the ice (or water used to make it) as a source of infection. The association with employer firm was biologically plausible as *Salmonella* contaminated both the ice machine and water tanks used by firms A and B (Figure 1). For each firm separately it appeared the distribution of cases to controls was unlikely to be random, with $p = 0.03$. (Table 2). The same analysis, this time allocating the firms according to which tanks they used, shows that the risk of illness to employees in firms A and B (sharing the same water tanks) was threefold that of employees of firm C, who used the uncontaminated tanks ($p = 0.02$).

Whilst eating at the pievan initially appeared to be a significant risk factor (Table 2), the questions asked of where the employees 'usually' ate were not rigorously defined, nor mutually exclusive, and the interpretation of results based on their responses is uncertain. Stratified analysis of the variables (illness, eating at the pievan, employee firm, sharing of water bottles and using an ice machine) was performed. A much higher proportion of employees in firms A and B (75%) usually consumed food from the pievan than did firm C employees (30%).

Stratified analysis of firm, illness and use of pievan showed crude and summary odd ratios to be different (4.02 versus 2.15) indicating confounding. There was a reduced risk of illness amongst firm C employees whether they ate at the pievan (odds ratio 0.15) or not (odds ratio 0.26).

The epidemic curve (Figure 2) shows a series of sporadic cases in the 6 weeks from the start of February and then a clustering of cases peaking around 24 March.

Figure 2. Gastrointestinal illness amongst construction site workers, 1 February to 31 March 1999, by date of onset and construction firm



Control measures

Following initial environmental health assessments on 25 March, site-managers were advised to ensure all staff used their own water bottles and that ice machine scoops be appropriately provided, stored and cleansed. It was requested that hand washbasins, soap and disposable paper towelling be placed close to meal facilities.

On 31 March 1999, after preliminary analysis of the epidemiological data, the construction site safety coordinator was requested to empty and sanitise the ice machines. On the same day a scheduled shutdown of the entire plant for 8 days over Easter enabled a convenient break in further exposure for workers whilst further results were awaited. On receipt of unsatisfactory water sample results during this period, the tanks and distribution system were re-sampled before being emptied, disinfected, flushed and refilled, measures not initially recommended because

Table 2. Exposures and characteristics of cases and controls, and results of statistical analysis

Exposure category	Cases (n=28)	Controls (n=88)	Odds ratio (OR)	Cornfield's 95% confidence intervals	Probability (P)
Sex	3F:25M*	4F:84M	2.52	0.41< OR <14.83	0.22
Firm (A, B or C)	12A:8B:8C	20A:19B:49C			0.03
Firms sharing water tanks (A&B, C)	20A&B:8C	39A&B:49C	3.10	1.2 < OR < 8.8	0.02
Used an ice machine	25Y:1N*	61Y:20N	8.20	1.05 < OR <175.5	0.01
Shared water bottle	13Y:10N	23Y:58N	3.28	1.13 < OR <9.60	0.01
Usually ate at pievan†	13Y:5N	22Y:34N	4.02	1.10 < OR <15.38	0.01
Usually ate at canteen†	13Y:2N	37Y:18N	3.12	0.42 < OR <4.93	0.35
Usually ate at home†	13Y:2N	37Y:18N	3.16	0.57 < OR <23.06	0.12

* F = female, M = male, Y = yes, N = no

† These categories are not mutually exclusive

obtaining drinking water for the site was difficult. However, during this remediation and whilst awaiting further water sampling results to assess its efficacy, a temporary alternative supply of bottled water was arranged.

Further recommendations included discontinuation of rainwater collection, regular chlorination of stored water, and disinfection of water bottles when not in use. The upsurge in cases ceased abruptly following institution of control measures. Follow-up water samples complied with NHMRC guidelines, and no further cases of gastroenteritis were reported after the end of March.

Exposures in relation to illness were analysed by contingency tables (Table 2).

Discussion

The epidemic curve for this outbreak (Figure 2) is neither consistent with person-to-person spread of a gastrointestinal infection with a relatively short incubation period, nor the 'classic' picture of a point-source outbreak, such as a meal at a function. It could be consistent with a point source of infection with an initial relatively low level of risk, which then increased due to either significantly increased levels of contamination or exposure.

Only two cases had faeces sampled owing to a time lag between onset and recognition of the outbreak, combined with the fact that few cases presented to a medical practitioner and faeces sampling was either not offered or was declined. These cases had no connection with each other apart from their workplace, yet both cultured *S*. Saintpaul. This fact, taken in context of the water sample results, makes it highly likely that the other cases were caused by the same organism. Their onset times also span the period during which the outbreak was at its peak in March, consistent with the other cases being caused by an ongoing (waterborne) source rather than from a single exposure at one point in time.

Since it is unclear exactly when this outbreak began, it is possible our case definition either excluded some earlier cases in February or included unrelated cases early in March (Figure 2). It is also possible that case ascertainment was incomplete to a minor degree if some staff developed their illness after leaving the area upon completion of contracts.

The observed association between illness and sharing water bottles is likely to be explained by confounding rather than person-to-person spread. Firms A and B did not supply individual water bottles. Therefore these employees were more likely to share water bottles; 58 per cent of employees from A and B did this, compared with 16 per cent from firm C. In the case of firms A and B, the shared water was sourced from contaminated tanks.

Owing to constraints in access to on-site staff, we were unable to carry out a cohort study or ask more detailed questions about exposures in a lengthy questionnaire. However, results of microbiological sampling supported by epidemiological findings provide strong evidence that this outbreak was caused by contamination of parts of the worksite reticulation system with *Salmonella* Saintpaul. The tanks with proven salmonella contamination (which collected rainwater as well as being topped up by tanker deliveries) supplied water to the taps, drinking water fountains, ice machine and kitchen/eating areas used by the most-affected work groups.

The most significant rain during February/March was a fall of 125 mm on 6 March, and it is possible this marked the actual beginning of the outbreak. Although the original source of contamination of the water tanks is uncertain, there are at least two plausible explanations.

Salmonellae may have been introduced via mice and/or their excreta washed into the tanks from the roof collection area. Mice were evident during site inspections despite rodent control measures. On cleaning, no dead mice were found inside the salmonella-contaminated interconnected tanks, but a number of live green tree frogs were and these may have introduced contamination directly. Alternatively, salmonella from animal excreta flushed into the tank by the heavy rainfall event multiplied in the frogs and were effectively 'amplified'.

O'Shea et al reported various salmonellae in 19 of 150 cane toads (*Bufo marinus*), but not *S*. Saintpaul.[15] NSSS data for 1985 - 1999 support this finding. The NSSS database records only one isolation of salmonella from a green tree frog (a *Salmonella* Onderstepoort). However, based upon overseas experience, a greater number of isolates and range of salmonellae are likely to be found if frogs were sampled more extensively.

Local Parks and Wildlife staff confirm that inquiries from the public about removal of frogs from water tanks are common. Maintenance of barriers across water tank inspection ports is the obvious (but frequently neglected) intervention to prevent ingress of frogs. Further research may be warranted to investigate the potential role of frogs as vectors in human salmonella infection.

Acknowledgments

Thanks to John Bates and staff at QHSS Microbiology Laboratory for water testing and advice, and to Diane Lightfoot and staff of the National Salmonella Surveillance Scheme based in the Microbiological Diagnostic Unit, Melbourne University. Mark Crome (Communicable Diseases Unit, Queensland Health) provided helpful comments on the manuscript. Jack Hunt (workplace health and safety coordinator at the construction site) and Banana Shire environmental health staff, including Jan Proposch, provided excellent assistance. The general practitioners concerned are particularly acknowledged for their role in bringing to our attention an outbreak that may otherwise have been missed.

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Antimicrobial resistance in *Streptococcus pneumoniae*: a decade of results from south-western Sydney

Iain B Gosbell,[1,2] Stephen A Neville[1]

Abstract

We report the emerging drug-resistance in *Streptococcus pneumoniae* seen by the South Western Area Pathology Service (SWAPS) from 1 January 1990 to 31 July 2000. SWAPS performs all the pathology testing for the public hospitals in the South Western Sydney Area Health Service, which serves a population of 700,000; 120,000 separations occur at these hospitals annually. In all, 2,265 patients submitted specimens yielding *S. pneumoniae*. These included respiratory tract specimens, blood cultures, eye swabs and cerebrospinal fluid (CSF). Resistance to penicillin, cefotaxime, and non-β-lactam antibiotics, especially cotrimoxazole, has emerged over the 1990s. From 1997 onwards, around 10 per cent of CSF and blood culture isolates demonstrated penicillin-resistance and 5 per cent showed cefotaxime-resistance. In 2000, 35 per cent of pneumococci from sites other than CSF and blood exhibited resistance to penicillin and 15 per cent showed resistance to cefotaxime. Resistance to other agents also increased over the decade. In 2000, 76 per cent of all isolates were resistant to cotrimoxazole, 26 per cent to erythromycin and 24 per cent to tetracyclines. Rifampicin-resistance was negligible over the decade, and vancomycin-resistance was absent. Antibiotics currently used for empirical treatment of certain *S. pneumoniae* infections may now need to be reviewed. *Commun Dis Intell* 2000;24:340-343.

Keywords: Streptococcus pneumoniae*, antimicrobial resistance, blood cultures, cerebrospinal fluid, sputum*

Introduction

Streptococcus pneumoniae is a major bacterial pathogen. The emergence of resistance in the drugs used to treat infections with this organism is of major public health significance.

Penicillin-resistant *S. pneumoniae* (PRSP) was first isolated clinically in Australia in 1967.[1] In the two decades after this, PRSP isolates were reported from Papua New Guinea,[2] South Africa,[3] southern Europe,[4] and the United States.[5] PRSP has become common in Australia in the 1990s,[6-11] but there is considerable geographic variation.[8] PRSP isolates are often resistant to multiple classes of antibiotics, such as cephalosporins, macrolides, tetracyclines and folate antagonists.[8,12]

Treatment failures with penicillin and third generation cephalosporins are well described in meningitis due to PRSP.[13] The significance of PRSP in respiratory tract infections is less clear; perhaps only isolates with high level resistance are associated with treatment failure.[5] The outcome in pneumonia due to PRSP with intermediate resistance to penicillin is not significantly different if penicillin or third generation cephalosporin is used in treatment.[14] The presence of penicillin-resistance is associated with a poorer outcome in children with invasive pneumococcal disease.[11]

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We report the emergence of drug-resistance in *S. pneumoniae* isolated from 1990 to the present from patients serviced by South Western Area Pathology Service.

Methods

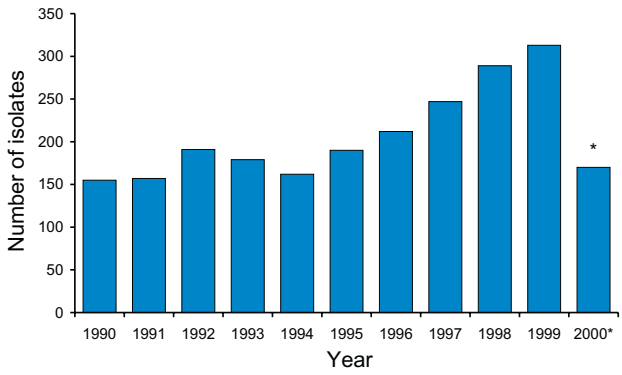
The South Western Area Pathology Service performs all the pathology testing for public hospitals in the South Western Sydney Area Health Service, which serves a population of 700,000. All specimens from which *S. pneumoniae* was isolated for the period 1 January to 31 July 2000 were included. Specimens with duplicate hospital record numbers were deleted. Isolates were identified as *S. pneumoniae* on the basis of typical colonial morphology and sensitivity to optochin; atypical isolates were confirmed with the API 20 Strep biochemical strip and the Slidex Pneumo-Kit (bioMérieux Vitek, Australia, Pty Ltd).

Susceptibility testing was performed using the ATB Strep microbroth dilution strip (bioMérieux Vitek, Australia, Pty Ltd), using NCCLS interpretive criteria.[15] Oxacillin was used to screen for penicillin-resistance, in which case penicillin and cefotaxime Minimal Inhibitory Concentrations (MICs) were determined using the E-test (AB BIODISK Solna, Sweden). For penicillin, isolates with an MIC 0.12-1.0 mg/L were considered to show intermediate resistance, and isolates with an MIC 2 mg/L to exhibit high level resistance; for cefotaxime, isolates with an MIC = 1.0 mg/L were considered to show intermediate resistance, and isolates with an MIC 2 mg/L to exhibit high level resistance.[15]

Results

The number of non-duplicated specimens yielding *S. pneumoniae* per year is shown in Figure 1. Between 1 January 1999 and 31 July 2000, a total of 2,265 unique specimens yielded *S. pneumoniae.* The sites sampled were: respiratory tract n = 1,312 (expectorated sputum n = 908, ear swabs n = 131, endotracheal aspirates n = 124, bronchoscopy specimens n = 76, nose swabs n = 42, throat swabs n = 31), blood cultures n = 628, eye swabs n = 172, CSFs n = 16, and other types n = 138.

Figure 1. Total isolates of *Streptococcus pneumoniae*, South Western Area Pathology Service, 1 January 1990 to 31 July 2000, by year



* I January to 31 July only

The increase in the proportions of isolates of *S. pneumoniae* demonstrating intermediate and high-level resistance to penicillin is depicted in Figure 2 for blood culture and CSF isolates, and for other types of specimen in Figure 3. Around 10 per cent of blood culture and CSF isolates have demonstrated penicillin-resistance from 1997 onwards. Resistance to penicillin has steadily increased since the mid-1990s, and in 2000 just over 35 per cent of isolates from sites other than blood and CSF demonstrated resistance.

Corresponding data for cefotaxime are shown in Figures 4 and 5. Resistance was more evident in isolates from sites other than blood and CSF, and has risen since the mid-1999s to 15 per cent in 2000. A lesser degree of resistance was seen in blood and CSF isolates.

Increases in resistance to cotrimoxazole, erythromycin, and tetracycline are shown in Figure 6. Resistance to cotrimoxazole has risen sharply, to 76 per cent in 2000. Only seven isolates resistant to rifampicin were detected, none in 1999 or 2000. No isolate was resistant to vancomycin.

Figure 2. Penicillin-sensitivity of *Streptococcus pneumoniae* isolated from blood cultures and CSFs

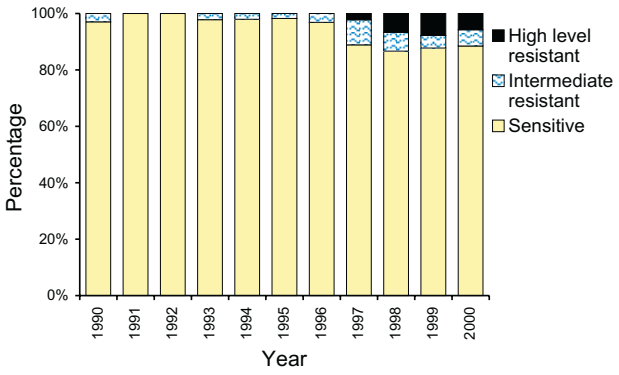
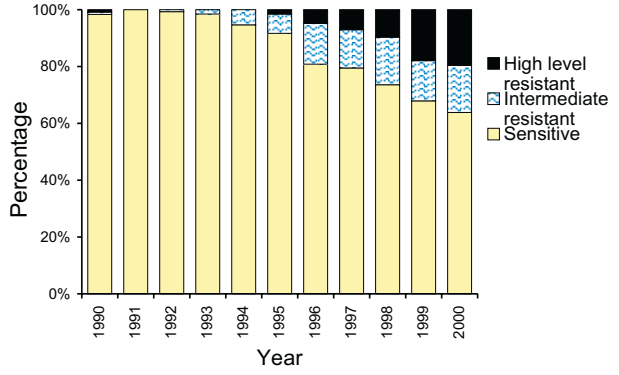


Figure 3. Penicillin-sensitivity of *Streptococcus pneumoniae* isolated from sites other than blood cultures and CSFs



Tables 1 and 2 indicate the overall numbers and proportions of penicillin-sensitive, and intermediate and high-level penicillin-resistant, *S. pneumoniae* isolates which were resistant to one or more other antimicrobial agents.

Figure 4. Cefotaxime-sensitivity of *Streptococcus pneumoniae* isolated from blood cultures and CSFs

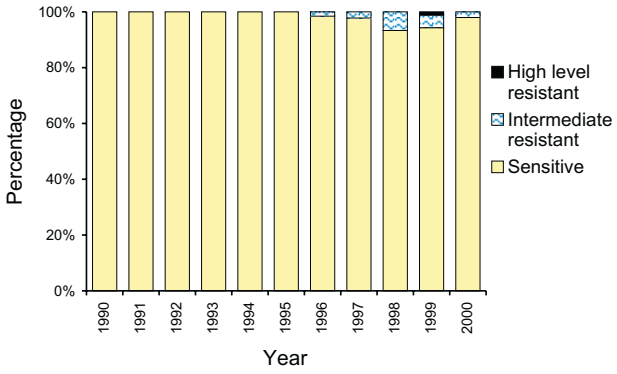


Figure 5. Cefotaxime-sensitivity of *Streptococcus pneumoniae* isolated from sites other than blood cultures and CSFs

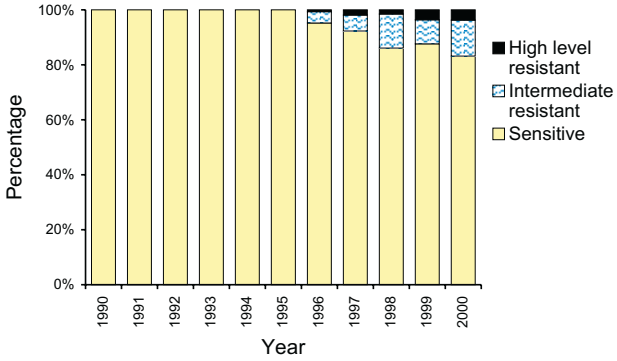
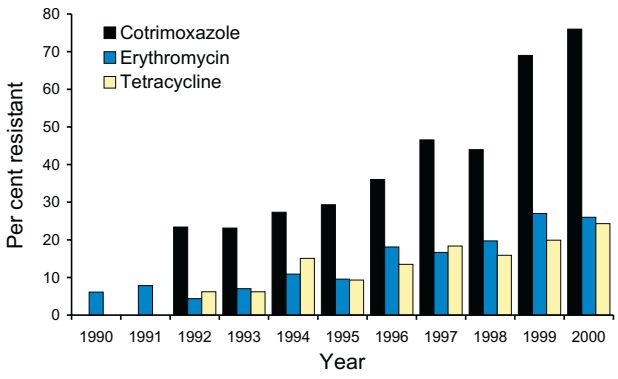


Figure 6. Resistance of *Streptococcus pneumoniae* isolates to non-β-lactam antibiotics



Discussion

Antimicrobial resistance in *S. pneumoniae* is rapidly emerging in Australia[6-8] and our data show that over the 1990s there has been a substantial increase in resistance in isolates from all body sites. 'Invasive' (primarily blood culture and CSF) isolates are less likely to be resistant to penicillin than 'non-invasive' (primarily sputum) isolates,[8] as we report here (Figures 2 and 3). *S. pneumoniae* isolates that are resistant to penicillin are more likely to be resistant to other types of antimicrobials such as cefaclor, cefotaxime, macrolides, tetracyclines, and folate antagonists (Tables 1 and 2), a finding mirrored elsewhere.[5,8] The rise in resistance to cotrimoxazole was particularly striking (Figure 6). Rifampicin is rarely used in the community, so it is not surprising that resistance to this agent has been minimal.

What are the implications of the above? In the case of meningitis, treatment failures are well described, even with isolates of intermediate resistance.[13] For the empiric treatment of meningitis, the decision to add additional drugs (such as vancomycin) depends on the likelihood of penicillin resistance.[8] In our laboratory, 11 per cent of blood and CSF isolates now demonstrate resistance to penicillin. Smaller percentages are also resistant to cefotaxime. In this situation, it may be reasonable to add vancomycin to the empiric treatment. If PRSP is isolated from a patient with meningitis, modification of the drug regimen is not defined and specialist advice should be sought. A recent Australian paper reported that children with invasive penicillin-resistant pneumococcal infections, particularly meningitis, required longer hospitalisation, and it took longer for their fever to abate.[11] Several regimens have been suggested for treating meningitis with penicillin-resistant pneumococci; these are vancomycin (possibly with rifampicin), very high doses of a third generation cephalosporin, or meropenem.[8]

The treatment of the much more common respiratory infections is more difficult. Treatment of pneumococcal respiratory infections with -lactams is unlikely to fail unless the isolate has high level resistance (MIC 2 mg/L).[16] In one study of pneumonia due to *S. pneumoniae*, treatment of PRSP (mainly of intermediate resistance) with penicillin or third generation cephalosporin did not result in a

Table 1. Number and proportions of penicillin-sensitive and penicillin-resistant *Streptococcus pneumoniae* resistant to other antimicrobial agents

Other antimicrobial	Sensitive n = 1966 %	Intermediate resistant n = 180 %	High level resistant n = 119 %
Cotrimoxazole	37.0	88	95
Cefotaxime			
Intermediate 1mg/L	0.0	26	63
High 2mg/L	0.0	0	21
Erythromycin	8.8	45	59
Tetracycline	7.6	45	58
Rifampicin	0.5	0	0

Table 2. Number and proportions of β-lactam-sensitive and β-lactam-resistant pneumococcal isolates resistant to no, or up to three, other classes of antimicrobial agent

		Resistance to non- -lactam antimicrobials*				
Penicillin susceptibility	n	0 (%)	1 (%)	2 (%)	3 (%)	4 (%)
Sensitive	1966	62	25	4	3	0
Intermediate	180	17	31	23	28	0
High level resistant	119	9	28	22	41	0

* Cotrimoxazole, erythromycin, tetracycline, and rifampicin.

significantly different mortality.[14] Many strains of PRSP are multi-drug resistant,[8,16] as was the case in our study, which makes the choice of a non- -lactam antimicrobial agent problematic. However, newer quinolones may be useful.[17]

It is essential that medical practitioners be informed of developing patterns of resistance in common organisms.[8] This information can influence their prescribing patterns, both in terms of prevention of the emergence of resistant organisms and treatment of infections by them. Most respiratory infections are viral, and many prescriptions for antimicrobials are therefore superfluous. The emergence of PRSP is driven partially by selection pressure of antimicrobial use.[18] When patients present with infections which may be due to PRSP, diagnostic specimens should be obtained for culture whenever possible.[8,19]

Penicillins may paradoxically remain the drugs of choice to treat PRSP infections, as resistance to penicillin is only relative and is less than to the other classes of antimicrobials, in particular oral cephalosporins.[8,20] In addition penicillins (amoxycillin in particular) will reach levels in respiratory secretions above the MIC of many strains of PRSP, which is not the case with oral cephalosporins.[16,19,20]

PRSP isolates are usually of one of the polysaccharide types incorporated into the available 23-valent pneumococcal vaccine.[5] Adults at risk of invasive pneumococcal infection (those over age 65 years, Aboriginals and Torres Strait Islanders over 50 years of age, asplenic or immunocompromised individuals, or those with chronic medical disease or CSF leaks) should be vaccinated as per the Australian Standard Immunisation Schedule.[21] Unfortunately, children under 2 years of age, who are at high risk of pneumococcal disease and are also more likely to harbour PRSP, do not respond to the currently available vaccine.[22] Conjugated pneumococcal vaccines will probably confer a higher response, particularly in children, and with the emergence of PRSP are now urgently needed.

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An outbreak of ciguatera fish poisoning in Victoria

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Abstract

An outbreak of ciguatera fish poisoning in outer Melbourne in September 1997 was traced to a 16.2 kg Maori Wrasse fish imported into Victoria from Trunk Reef in Queensland. The outbreak involved 46 individuals attending a banquet at an Asian restaurant at which four different dishes prepared from the flesh and viscera of the fish were offered. In all 30 individuals consumed at least one of these dishes and all reported one or more symptoms, in the main gastrointestinal and/or in 18 cases neurological. Seventeen cases were seen in four different hospitals and nine were treated with parenteral mannitol therapy. Nine of 18 cases were still symptomatic 10 weeks after the episode. Education of Asian restaurateurs and the wider community about the risks of ciguatera fish poisoning was undertaken. *Commun Dis Intell* 2000;24:344-346.

Keywords: reef fish, ciguatera, gastroenteritis, neurological symptoms, food poisoning.

Introduction

Ciguatera fish poisoning is caused by the consumption of fish contaminated by naturally occurring toxins produced by the algal dinoflagellate *Gambierdiscus toxicus* associated with coral reefs. Ciguatera fish poisoning is widespread, affecting mainly tropical and sub-tropical areas of the world.[1] Most reported outbreaks in Australia have involved Spanish mackerel, but numerous other species of fish, including red bass, the chinaman and coral trout, have been implicated.[1, 2]

The outbreaks are generally restricted to certain areas around the coastline of the Northern Territory and Queensland where 175 outbreaks involving 527 people were reported between 1965 and 1984.[1] However with increasing popularity of fresh reef fish (and their export to other States) cases of ciguatera fish poisoning can present anywhere in Australia.

On 16 September 1997, following a report of a suspected case seen at the Emergency Department of a Melbourne hospital, the Infectious Diseases Unit, Department of Human Services, Victoria, commenced investigations into a suspected outbreak of ciguatera fish poisoning. The same day a general practitioner also reported seeing three patients with symptoms consistent with ciguatera poisoning. Both doctors advised that their patients had been part of a large group that had eaten at an Asian restaurant in the eastern suburbs of Melbourne the day before. Initial information was that approximately 18 of 36 people had become ill after eating fish as part of a banquet meal.

Methods

A case was defined as a person who, having eaten the suspect fish, experienced neurological symptoms with or without gastrointestinal symptoms. With the assistance of the notifying doctor and the organiser of the group, active case finding was commenced and it was determined that 46 people (29 adults, 17 children) had eaten at the banquet. Children ate from a different menu, with only one child eating any of the suspect fish.

A questionnaire, adapted from the standard Queensland Department of Health 'Ciguatera Questionnaire', was administered by phone to all banquet attendees or their parents. Demographic details and information on presenting symptoms were obtained in addition to the particular dishes and the quantities thereof consumed by each case.

All cases were provided with written information on ciguatera and advised to abstain from consumption of alcohol and fish for at least 3 months to prevent recurrence of symptoms.[2, 3] To gather information on the duration of symptoms, and whether any new ones had appeared since the first interview, follow-up interviews were conducted with cases at weeks 3 and 10 post-exposure. The restaurant manager was contacted and interviewed about the source of the fish and the method of its preparation.

Results

On 15 September 1997 a live 16.2 kg Maori Wrasse fish (delivered to the restaurant by a seafood wholesaler) was used in the preparation of four separate dishes. One dish contained the head and another the intestines of the fish. The four dishes were served exclusively that day to a large group as part of a banquet meal. Investigations at the restaurant revealed that while most of the fish had been served at the banquet, a small portion of the flesh and the backbones had been reserved for soup.

The wholesaler reported that the fish was the largest of three in a consignment from Queensland. The other two of the same species, but smaller (8.1 kg and 8.6 kg) had been delivered to two City restaurants. One fish was eaten on 15 September with no reported illness attributed to its consumption, the other was seized (live) with the proprietor's consent.

Enquiries by Queensland Health Department established that the 16.2 kg Maori Wrasse served at the banquet had been caught off Trunk Reef in Queensland. The other two fish were purchased from another supplier and the area where they had been caught could not be confirmed.

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All 30 individuals who consumed the fish at the banquet reported having at least one symptom. Cases (age range 13 to 69 years) presented typically with gastrointestinal and neurological symptoms. The main symptoms are listed in the Table.

The incubation period ranged from 2 to 27 hours (median 8 hours). Symptoms varied in severity. Of 21 people (70%) seeking medical attention, 17 were seen at four different hospitals with 3 admitted overnight. The other 4 attended local medical practitioners. Nine were treated with intravenous mannitol.

At week three, 22/30 (73%) were still symptomatic (2 were lost to follow up). Symptoms included paraesthesia of the extremities (13), weakness (11), and myalgia (11). A few developed late symptoms of itch (13), dysuria (3), and rash (2). Of the nine given mannitol, one had recovered completely. The other eight reported feeling better but still had some residual neurological symptoms such as paraesthesia and hot and cold temperature reversal.

By week ten, 9/18 cases were still symptomatic (a further 4 cases were lost to follow up).

Discussion

Ciguatoxin, which accumulates through the food chain, is thermostable and is not destroyed by cooking, freezing or other fish-processing methods. It does not affect the taste or texture of the fish. In humans, ciguatoxin produces gastrointestinal and characteristic neurological symptoms, consisting mainly of sensory disturbances (Table).

Table. Number and proportion of symptoms reported in cases involved in an outbreak of ciguatera fish poisoning, Melbourne, September 1997

Symptom	Number of cases n = 30	%
Gastrointestinal		
Diarrhoea	20	67
Abdominal Pain	14	47
Nausea	9	30
Vomiting	5	17
Neurological		
Paraesthesia of hands	26	87
Paraesthesia of feet	23	77
Hot/cold temperature reversal	19	63
Circumoral paraesthesia	17	57
Ataxia	11	37
Tremors	7	23
Dental pain	4	13
Others		
Myalgia	23	77
Weakness	21	70
Chills	21	70
Arthralgia	17	57
Neck Stiffness	10	33
Pruritis	8	27
Shortness of breath	4	13
Dysuria	3	10

Diagnosis in this outbreak was based on the food history, and clinical presentations consistent with those reported in previous outbreaks.[1,2,4] Ciguatera poisoning is usually self-limiting with signs and symptoms generally subsiding within several days from onset. In a few cases neurological symptoms can persist for several years.[1,3] Treatment is mainly supportive, but mannitol infusion has been found to be beneficial, even when administered up to a week after the onset of symptoms.[4,5,6] We were unable to ascertain whether severity or multitude of symptoms related to the amount of fish consumed, or particular dish eaten.

Whilst reports of ciguatera fish poisoning are not infrequent in Queensland, this was only the second known outbreak reported in Victoria over the past 20 years. In 1994, coral trout was epidemiologically implicated in an outbreak of suspected ciguatera fish poisoning.

Although cases are rarely reported in Victoria, as the condition is not notifiable and is often likely to go unrecognised, the true incidence is unknown.

Public health measures

Ciguatoxin is known to be concentrated in the liver, roe, head and other viscera of suspect fish, and the larger the fish the greater its chance of being toxic[1,3,4] Many of these anatomical items are popular with the Asian community, and for banquet style meals dishes could be prepared from large fish. Certain fish known to be associated with ciguatera fish poisoning are generally not accepted for sale in Queensland.[1,2] This outbreak shows that other previously implicated species are being marketed in Victoria. It also illustrates how changing eating habits and food distribution patterns can lead to outbreaks of ciguatera fish poisoning in locations where it is uncommonly reported.

The low prevalence of the toxin in fish (in areas known to have contaminated fish, 1in 5,000 coral trout is estimated to be toxic[1]) and the lack of a simple routine test for the toxin means consumer education is important. We identified a need to inform Asian restaurateurs of the risk of ciguatera fish poisoning and the need to avoid large reef fish, especially the head and other viscera.

A fact-sheet on ciguatera, adapted from the Queensland pamphlet *Ciguatera Poisoning-Information and Treatment*,[2] was circulated with the monthly newsletter of the Victorian Association of Chinese Restaurateurs (with a membership of around 200) and information was also published in the Newsletter of the Victorian Multicultural Council. This fact-sheet has also been translated into three Asian languages and has been made available to all local government environmental health officers for distribution to relevant food premises within their municipalities.

The public health measures initiated as the result of this outbreak have demonstrated the value of notifying public health authorities of incidents such as that reported here.

Acknowledgments

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Contributions to the Bulletin Board are invited from those organisations with forthcoming events relevant to communicable disease control.

An outbreak of *Salmonella* Typhimurium RDNC A047 linked to a Chinese restaurant in South Australia

*A Milazzo**

In January 2000, a cluster of six cases of *Salmonella* Typhimurium RDNC ('Reacts Does Not Conform') A047 were notified to the Communicable Disease Control Branch. All six cases resided in the same geographical area with an age range from one to 77 years (median age 38 years). The epidemiological investigation found that five of the six cases had eaten food at a local Chinese restaurant on 24 and 25 December 1999. No other risk factors were identified.

Four cases attended the restaurant on 25 December for Christmas lunch and consumed foods from the Chinese and/or the Australian Banquet. The one case who ate at the restaurant on Christmas Eve ordered individual dishes from the restaurant menu. Common foods consumed by all cases included prawn chips; spring rolls, special fried rice, roast pork plum sauce and chicken and sweet corn soup. All food including sauces and fillings for spring rolls is prepared at the restaurant. Subsequent microbiological sampling on fresh and pre-prepared foods and equipment and environmental surfaces was negative. Restaurant staff did not report gastrointestinal illness or overseas travel within the previous six months.

This previously unrecognised *Salmonella* Typhimurium phage type (RDNC A047) was epidemiologically linked with the consumption of food prepared at a local Chinese restaurant.

An outbreak of *Salmonella* Typhimurium phage type 44 linked to a restaurant in South Australia

Ingrid G Tribe, James Walker†*

In October 2000, a cluster of eleven cases of salmonellosis were investigated by the Communicable Disease Control Branch. Hypothesis generating interviews sought demographic, illness, food purchasing practices, food consumption, social activities, and animal contact information for the seven day period prior to the onset of symptoms. One case reported a history of overseas travel. Of the remaining ten (6 female, 4 male) cases, the age range was 16 to 80 years. Cases were reported from both metropolitan and rural areas of South Australia. All ten cases reported eating at an Adelaide metropolitan restaurant between the 4 and 7 October 2000. The predominant symptoms were abdominal pain (80%), diarrhoea (80%), fever (60%), nausea (60%), and bloody diarrhoea (40%). The median incubation period was 39.5 hours (range: 24 to 168 hours). One urine and nine stool specimens obtained from cases were positive for *Salmonella* Typhimurium phage type 44. Faecal specimens obtained from two symptomatic family members, were negative for *Salmonella* sp. There were no reports of gastrointestinal illness in restaurant employees.

A case-control study suggested de-boned roast pork, (Odds Ratio (OR) undefined, 95% Confidence Interval (CI) 1.24 - undefined) and apple sauce, (OR 21..33, CI 2.27 – 263.58), as two possible vehicles for the infections. An environmental investigation conducted two weeks after the exposures found customary cooking times and storage temperatures for de-boned roast pork were adequate. Although one patron reported the consumption of inadequately cooked roast pork, no other reports were received. The de-boned pork are spiced and roasted in pairs. The turn over of individual de-boned roast pork in this restaurant was estimated at 1.5 days. Assuming this, to suggest de-boned roast pork was responsible for these infections would indicate abnormalities in food handling had occurred on no less than two consecutive occasions. Similarly, the preparation and storage of the apple sauce was consistent with good hygiene practices. Commercial canned apples were used to make the apple sauce. The apple sauce was prepared in bulk and stored in a sealed container in the cool room. The apple sauce was decanted to serving dishes as required. The turn over of individual batches of apple sauce in this restaurant was estimated at three to four days. There were no food samples available for microbiological testing. Spices used in the preparation of the roast pork were negative for *Salmonella* sp. Cross-contamination of food items could not be identified.

Although the source for this outbreak was not established, patrons reported lapses in restaurant hygiene practices. These included the presence of insects in the restaurant environment and flies in prepared food and drinking water. However, the environmental investigation was unable to substantiate these claims. No further cases of *Salmonella* Typhimurium phage type 44 infection reported dining at this restaurant.

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Communicable Diseases Surveillance

Presentation of NNDSS data

In the March 2000 issue an additional summary table was introduced. Table 1 presents 'date of notification' data, which is a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health unit. Table 2 presents the crude incidence of diseases by State or Territory for the current reporting month. Table 3 presents data by report date for information only. In Table 3 the report date is the date the public health unit received the report.

Table 1 now includes the following summary columns: total current month 2000 data; the totals for previous month 2000 and corresponding month 1999; a 5-year mean which is calculated using previous, corresponding and following month data for the previous 5 years (*MMWR Morb Mortal Wkly Rep*, 2000:49;139-146); year to date (YTD) figures; the mean for the year to date figures for the previous 5 years; and the ratio of the current month to the mean of the last 5 years.

Highlights for October, 2000

Communicable Disease Surveillance Highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence*. These national data collections are complemented by intelligence provided by State and Territory communicable disease epidemiologists and/or data managers who have recently formed a Data Management Network. This additional information has enabled the reporting of more informative highlights each month.*

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand, and the CDI *Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.*

Three types of data are included in National Influenza Surveillance, 2000. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network (ASPREN), the Department of Human Services (Victoria), the Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health 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. Data from ASPREN are referred to as 'consultations' or 'encounters'. For further information about these schemes, see* Commun Dis Intell *2000;24:9-10.*

Highlights for October 2000

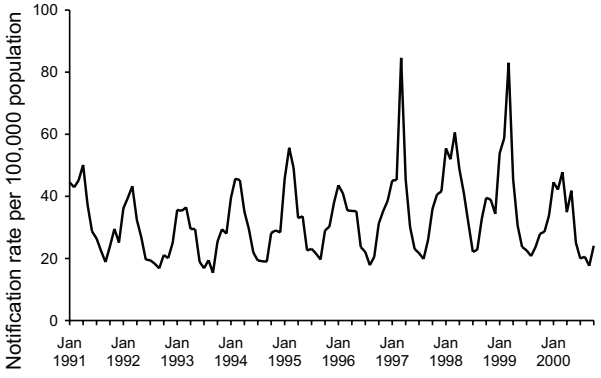
In October 2000 the number of reports of chlamydial infection (Ratio 1.6), legionellosis (Ratio 1.4) and meningococcal infection (Ratio 1.4) has increased compared with the 5-year mean (Figure 7, Table 1).

Gastrointestinal illness

Campylobacter and *Salmonella* notifications continue to be fewer than in previous years with a rate of 118/100,000 population for Campylobacter and 24.1/100,000 population for *Salmonella* (Figure 1). The Australian Capital Territory had the highest rate for *Campylobacter* (237/100,000 population) and the Northern Territory the highest rate for *Salmonella* (112/100,000 population)

An outbreak of ten (4 male, 6 female) cases of *Salmonella* Typhimurium phage type 44 infection linked to a restaurant in metropolitan Adelaide was investigated by local government Environmental Health Officers and the Communicable Disease Personnel in South Australia. Cases report eating at the restaurant from the

Figure 1. Notification rate of salmonellosis, Australia, 1 January 1991 to 31 October 2000, by month of notification



4 to 7 October 2000. A case-control study is currently being conducted. Also an apparent cluster of cases of *Campylobacter* infection in residents of a small rural community in South Australia is being investigated. Hypothesis generating interviews are being conducted with cases to identify the source of the cluster. Two cases have reported the consumption of raw milk and raw milk products from local dairies.

There was one case of typhoid reported in New South Wales in a 33-year-old male.

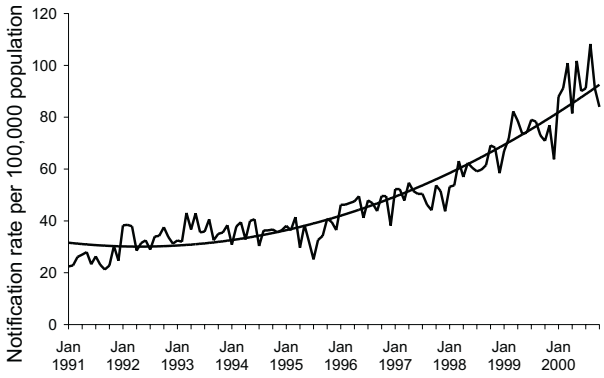
There were two notifications of Shiga-like *Escherichia coli* (SLTEC)/verotoxigenic *Escherichia coli* (VTEC) in October 2000 from South Australia, both were in males one aged 33 years and one aged 66 years.

There was a small outbreak in a student residential setting in Tasmania with 3 confirmed cases of *Campylobacter*. Nine other residents had the same symptoms but were not tested. The total population was 150 persons. Several deficiencies in food handling practices but no common food items were identified. No further cases have been reported.

Chlamydial infection

There were 1,327 notifications of chlamydial infection in October 2000 - a notification rate of 84.0/100,000 population which is an increased rate from previous months (Figure 2). Of these cases, 78 per cent were in the 15 to 29 age groups. The Northern Territory continues to have the highest rate for chlamydial infection (385.7/100,000 population).

Figure 2. Notification rate of chlamydial infection, Australia, 1 January 1991 to 31 October 2000, by month of notification



Vaccine preventable diseases

All vaccine preventable diseases had fewer reports this month than for the 5-year-mean for October. Pertussis notifications are down from last month (396 cases with a rate of 25.1/100,000 population compared with 624 cases with a rate of 39.5/100,000 population) (Figure 3).

Measles cases continue to be at their lowest level since the national notification system began (Figure 4). Of the ten cases reported in October 2000, four were reported in New South Wales, three in Queensland, two in Victoria and one in South Australia.

The New South Wales cases were part of two measles clusters which have recently occurred in that State. The first cluster of 10 cases began with an imported case of measles in late August, which resulted in four generations of cases affecting mostly young unimmunised adults. The second cluster of five cases has occurred in unimmunised or incompletely immunised children, with confirmed links between four of the cases. The cases in Queensland, Victoria and South Australia included a 32-year-old male (unknown vaccination status who acquired the infection interstate at the Olympics), an 8-year-old female with two documented vaccinations (who acquired the infection locally), a 27-year-old female vaccinated once only (who acquired the infection locally), a one-year-old male, a 21-year-old female and a one-year-old female.

Figure 3. Notification rate of pertussis, Australia, New South Wales and Australian Capital Territory, 1 January 1999 to 31 October 2000, by month of notification

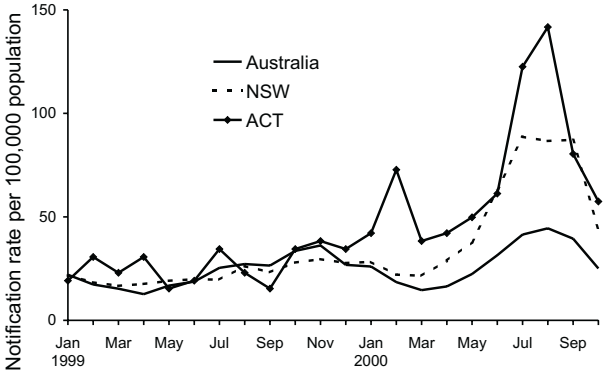
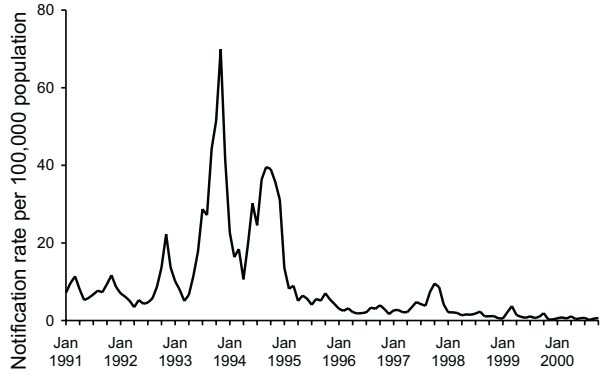


Figure 4. Notification rate of measles, Australia, 1 January 1991 to 31 October 2000, by month of notification



Legionellosis

There were 22 notifications of legionellosis in October 2000 - a notification rate of 1.4/100,000 population (Figure 5) with South Australia having the highest rate for legionellosis (7.2/100,000 population).

Meningococcal infections

There were 63 notifications of meningococcal infection in October 2000 - a notification rate of 4.0/100,000 population (Figure 6). Of these cases, 33 per cent were under 5 years of age and 38 per cent were in the 15 to 24 year age group. The serogroups were available for 22 cases; these were serogroup B (45%), serogroup C (50%) and serogroup W135 (5%).

Figure 5. Notification rate of legionellosis, Australia, 1 January 1991 to 31 October 2000, by month of notification

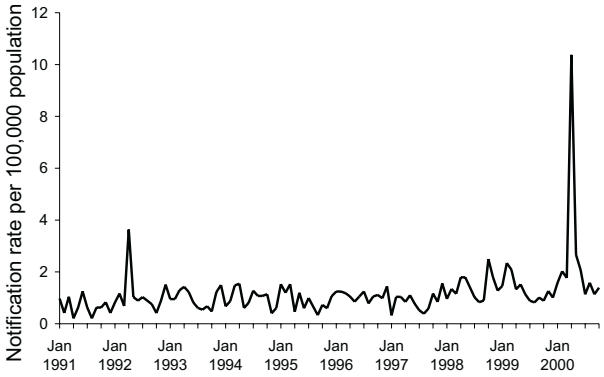
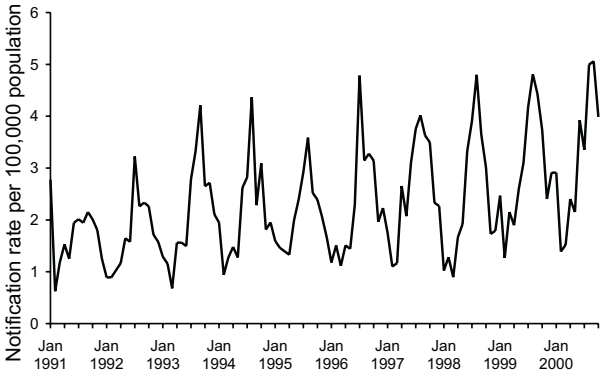


Figure 6. Notification rate of meningococcal infection, Australia, 1 January 1991 to 31 October 2000, by month of notification



Special report on psittacosis in Tasmania

David Coleman, Scientific Officer, Communicable Diseases Surveillance, Public and Environmental Health Service, Department of Health and Human Services, Tasmania

All six cases of psittacosis from northern Tasmania either owned or had recent contact with cockatiels but there was no common source for all cases. Some had recently purchased these birds. The sellers of the birds were checked but no sick birds were identified. However, as there may have been asymptomatic shedding of bacteria, these birds were treated with antibiotics.

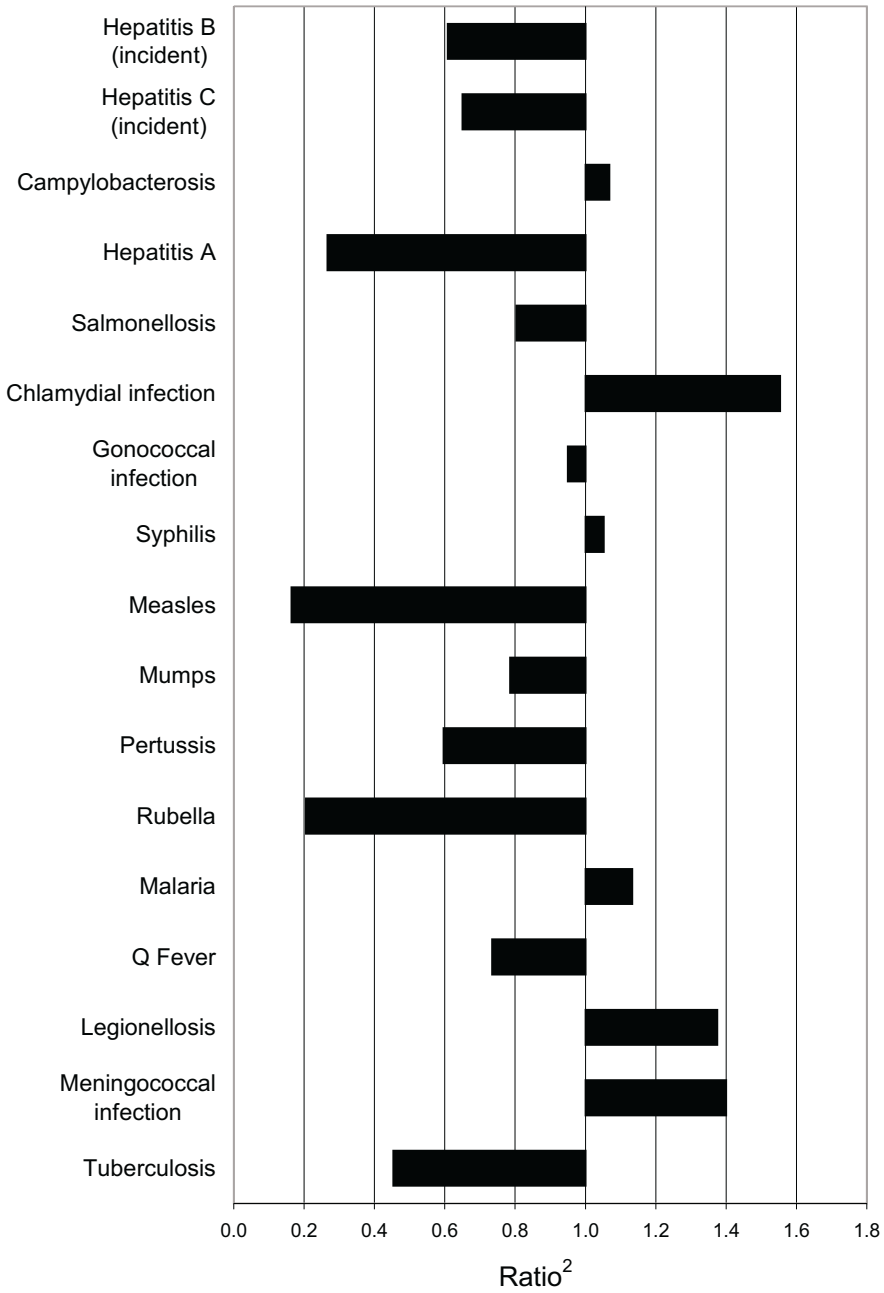
Tables

There were 6,060 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in October 2000 (Table 1). The crude incidence of diseases per 100,000 population for each State or Territory (Table 2) was included for the first time in the August 2000 issue of *Commun Dis Intell*. Data by date of report for October 2000, are included in this issue of *Commun Dis Intell* (Table 3). Figure 7 illustrates, for selected diseases, the October 2000 totals as ratios to the mean of their September to November levels for the previous 5 years (1995 to 1999).

There were 1,705 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 31 October 2000 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 39 to 42, ending 22 October 2000, are included in this issue of *Commun Dis Intell* (Table 6).

Figure 7. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 31 October 2000 with historical data[2]



1. Selected diseases are chosen each calendar month according to current activity
2. Ratio of current month total to mean of September to November data for the previous five years

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 31 October 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total October 2000[1]	Total September 2000[1]	Total October 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	0	4	0	3	3	2	1	1	14	38	29	23	336	234	0.6
Hepatitis B (unspecified)[2]	2	140	0	73	11	3	140	55	424	674	691	582	6,696	5,810	0.7
Hepatitis C (incident)	3	1	0	-	5	0	3	1	13	31	42	20	403	166	0.7
Hepatitis C (unspecified)[2]	17	336	26	280	39	43	392	87	1,220	1,584	1,745	1,347	17,193	13,266	0.9
Hepatitis D	0	0	0	0	0	0	0	0	0	0	4	2	15	17	0.0
Gastrointestinal															
Botulism	0	0	0	0	0	0	1	0	1	0	0	0	1	0	0.0
Campylobacterosis[3]	62	-	21	292	204	60	425	172	1,236	1,028	1,057	1,157	11,135	9,749	1.1
Haemolytic uraemic syndrome	0	0	0	1	0	0	0	0	1	1	3	1	8	NA	-
Hepatitis A	1	11	3	10	1	0	10	4	40	49	128	150	719	1,871	0.3
Hepatitis E	0	0	0	0	0	0	0	0	0	0	0	0	0	3	0.0
Listeriosis	0	1	0	1	0	1	0	0	3	3	7	6	55	54	0.5
Salmonellosis	3	51	18	155	28	9	57	60	381	278	442	474	5,036	5,603	0.8
Shigellosis[3]	1	-	14	8	1	0	5	7	36	34	44	51	403	570	0.7
SLTEC,VTEC[4]	0	0	0	NN	2	0	0	NN	2	6	3	1	31	NA	-
Typhoid	0	1	0	0	0	0	0	0	1	2	3	4	58	63	0.3
Yersiniosis[3]	0	-	0	4	0	0	0	1	5	4	9	17	64	198	0.3
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	1	4	-
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Sexually transmissible															
Chancroid	0	0	0	0	0	0	0	0	0	0	0	0	0	1	-
Chlamydial infection[5]	30	229	62	418	138	24	210	216	1,327	1,439	1,120	854	14,663	8,265	1.6
Donovanosis	0	0	0	1	NN	0	0	0	1	0	1	4	11	39	0.3
Gonococcal infection[6]	2	41	59	91	6	1	70	89	359	495	427	378	5,255	3,879	0.9
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Syphilis[7]	0	36	13	88	0	1	0	3	141	159	134	134	1,582	1,433	1.1

Table 1 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 31 October 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total October 2000[1]	Total September 2000[1]	Total October 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Vaccine preventable															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Haemophilus influenzae type b	0	0	0	0	0	0	0	0	0	4	4	4	21	42	0.0
Measles	0	4	0	3	1	0	2	0	10	8	30	61	93	521	0.2
Mumps	0	5	1	0	2	0	3	0	11	19	17	14	185	142	0.8
Pertussis	15	235	0	37	48	3	50	8	396	624	530	664	4,422	4,530	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Rubella[8]	1	48	0	1	1	0	3	0	54	24	36	263	235	1,469	0.2
Tetanus	0	0	0	0	1	0	0	0	1	1	0	0	7	4	-
Vectorborne															
Arbovirus infection NEC	0	0	0	0	0	0	0	0	0	1	0	3	64	49	0.0
Barmah Forest virus infection	0	10	0	27	0	0	0	1	38	34	24	36	458	621	1.1
Dengue	0	0	0	2	0	0	0	0	2	6	1	15	203	144	0.1
Malaria	2	1	7	12	0	0	8	21	51	50	46	45	799	635	1.1
Ross River virus infection	0	13	2	78	5	0	3	3	104	63	105	107	3,710	4,549	1.0
Zoonoses															
Brucellosis	0	0	0	2	0	0	0	0	2	5	6	5	19	32	0.4
Hydatid infection	0	NN	0	1	0	0	0	0	1	4	3	4	21	37	0.3
Leptospirosis	0	7	1	0	1	0	3	0	12	11	14	17	189	166	0.7
Ornithosis	0	NN	0	NN	0	3	5	0	8	9	6	10	69	65	0.8
Q fever	0	7	0	22	0	0	5	2	36	50	59	49	432	450	0.7
Other															
Legionellosis	0	1	0	2	9	0	4	6	22	18	14	16	407	170	1.4
Leprosy	0	0	0	0	0	0	0	0	0	0	1	1	3	7	0.0
Meningococcal infection	0	24	2	6	3	0	21	7	63	80	59	45	501	395	1.4
Tuberculosis	0	9	0	5	0	0	27	3	44	63	93	97	757	876	0.5
Total	139	1,215	229	1,623	509	150	1,448	747	6,060	6,899	6,937	6,661	76,260	66,145	

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella

\# Date of notification = a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health unit.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

* Ratio = ratio of current month total to the mean of the last 5 years (where data are available) calculated as described above.

NA Not calculated as only notifiable for under 5 years.

Table 2. Crude incidence of diseases by State or Territory, 1 to 31 October 2000. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Bloodborne									
Hepatitis B (incident)	0.0	0.7	0.0	1.0	2.4	5.1	0.3	0.6	0.9
Hepatitis B (unspecified)[2]	7.7	26.2	0.0	24.9	8.8	7.7	35.7	35.5	26.8
Hepatitis C (incident)	11.5	0.2	0.0	-	4.0	0.0	0.8	0.6	1.0
Hepatitis C (unspecified)[2]	65.1	62.9	161.8	95.7	31.3	109.7	99.8	56.1	77.2
Hepatitis D	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gastrointestinal									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.1
Campylobacterosis[3]	237.4	-	130.6	99.8	164.0	153.1	108.2	110.9	118.1
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Hepatitis A	3.8	2.1	18.7	3.4	0.8	0.0	2.5	2.6	2.5
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Listeriosis	0.0	0.2	0.0	0.3	0.0	2.6	0.0	0.0	0.2
Salmonellosis	11.5	9.5	112.0	53.0	22.5	23.0	14.5	38.7	24.1
Shigellosis[3]	3.8	-	87.1	2.7	0.8	0.0	1.3	4.5	3.4
SLTEC,VTEC[4]	0.0	0.0	0.0	NN	1.6	0.0	0.0	NN	0.2
Typhoid	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Yersiniosis[3]	0.0	-	0.0	1.4	0.0	0.0	0.0	0.6	0.5
Quarantinable									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible									
Chancroid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chlamydial infection[5]	114.9	42.9	385.7	142.8	110.9	61.2	53.5	139.3	84.0
Donovanosis	0.0	0.0	0.0	0.3	NN	0.0	0.0	0.0	0.1
Gonococcal infection[6]	7.7	7.7	367.1	31.1	4.8	2.6	17.8	57.4	22.7
Lymphogranuloma venereum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis[7]	0.0	6.7	80.9	30.1	0.0	2.6	0.0	1.9	8.9
Vaccine preventable									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Measles	0.0	0.7	0.0	1.0	0.8	0.0	0.5	0.0	0.6
Mumps	0.0	0.9	6.2	0.0	1.6	0.0	0.8	0.0	0.7
Pertussis	57.4	44.0	0.0	12.6	38.6	7.7	12.7	5.2	25.1
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[8]	3.8	9.0	0.0	0.3	0.8	0.0	0.8	0.0	3.4
Tetanus	0.0	0.0	0.0	0.0	0.8	0.0	0.0	0.0	0.1
Vectorborne									
Arbovirus infection NEC	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Barmah Forest virus infection	0.0	1.9	0.0	9.2	0.0	0.0	0.0	0.6	2.4
Dengue	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.1
Malaria	7.7	0.2	43.5	4.1	0.0	0.0	2.0	13.5	3.2
Ross River virus infection	0.0	2.4	12.4	26.6	4.0	0.0	0.8	1.9	6.6

Table 2 (continued). Crude incidence of diseases by State or Territory, 1 to 31 October 2000. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Zoonoses									
Brucellosis	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.1
Hydatid infection	0.0	NN	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	1.3	6.2	0.0	0.8	0.0	0.8	0.0	0.8
Ornithosis	0.0	NN	0.0	NN	0.0	7.7	1.3	0.0	1.1
Q fever	0.0	1.3	0.0	7.5	0.0	0.0	1.3	1.3	2.3
Other									
Legionellosis	0.0	0.2	0.0	0.7	7.2	0.0	1.0	3.9	1.4
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	0.0	4.5	12.4	2.0	2.4	0.0	5.3	4.5	4.0
Tuberculosis	0.0	1.7	0.0	1.7	0.0	0.0	6.9	1.9	2.8
Total	532.3	227.4	1424.7	554.5	409.1	382.8	368.7	481.7	383.4

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 3. Notifications of diseases received by State and Territory health authorities in the period 1 to 31 October 2000, by date of report*

Disease[1]	State or Territory								Total this period	Year to date total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Bloodborne										
Hepatitis B (incident)	0	6	0	5	2	1	2	3	19	346
Hepatitis B (unspecified)[2]	9	282	0	75	27	5	140	60	598	6,933
Hepatitis C (incident)	3	3	0	-	9	0	4	1	20	418
Hepatitis C (unspecified)[2]	30	490	25	298	68	34	393	97	1,435	17,542
Hepatitis D	0	0	0	0	0	0	0	0	0	15
Gastrointestinal										
Botulism	0	0	0	0	0	0	1	0	1	1
Campylobacterosis[3]	61	-	14	303	213	69	445	167	1,272	11,197
Haemolytic uraemic syndrome	0	1	0	0	0	0	0	0	1	7
Hepatitis A	1	16	4	9	0	0	14	6	50	747
Hepatitis E	0	0	0	0	0	0	0	0	0	0
Listeriosis	0	2	0	1	0	1	1	0	5	56
Salmonellosis	4	60	17	143	28	11	62	55	380	5,186
Shigellosis[3]	1	-	9	13	1	0	8	7	39	406
SLTEC,VTEC[4]	0	0	0	NN	3	0	0	NN	3	33
Typhoid	0	3	0	0	0	0	0	0	3	63
Yersiniosis[3]	0	-	0	3	0	0	0	0	3	64
Quarantinable										
Cholera	0	0	0	0	0	0	0	0	0	1
Plague	0	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0	0
Sexually transmissible										
Chancroid	0	0	0	0	0	0	0	0	0	0
Chlamydial infection[5]	28	284	62	470	158	23	259	228	1,512	14,730
Donovanosis	0	0	0	1	NN	0	0	0	1	12
Gonococcal infection[6]	1	81	77	107	17	1	73	95	452	5,325
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0
Syphilis[7]	2	47	10	92	1	1	0	4	157	1,635
Vaccine preventable										
Diphtheria	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	0	0	0	0	0	0	0	0	23
Measles	1	6	0	1	1	0	2	0	11	93
Mumps	0	8	1	0	2	0	2	1	14	189
Pertussis	29	461	0	38	72	5	56	11	672	4,576
Poliomyelitis	0	0	0	0	0	0	0	0	0	0
Rubella[8]	1	43	0	1	1	0	3	0	49	227
Tetanus	0	0	0	1	1	0	0	0	2	8
Vectorborne										
Arbovirus infection NEC	0	0	0	0	0	0	0	0	0	65
Barmah Forest virus infection	0	12	0	26	0	0	0	2	40	507
Dengue	0	1	0	5	0	0	0	0	6	227
Malaria	2	1	8	22	2	0	7	23	65	865
Ross River virus infection	0	17	1	77	6	0	3	4	108	3,987

Table 3 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 31 October 2000, by date of report*

Disease[1]	State or Territory								Total this period	Year to date total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Zoonoses										
Brucellosis	0	0	0	5	0	0	0	0	5	20
Hydatid infection	0	NN	0	2	0	0	0	0	2	22
Leptospirosis	1	3	0	5	1	0	3	1	14	196
Ornithosis	0	NN	0	NN	0	5	8	0	13	77
Q fever	0	10	0	34	1	0	5	3	53	455
Other										
Legionellosis	0	1	0	2	10	0	5	2	20	407
Leprosy	0	0	0	0	0	0	0	0	0	4
Meningococcal infection	0	30	1	7	3	1	20	9	71	507
Tuberculosis	0	23	0	5	27	0	31	8	94	851
Total	174	1,891	229	1,751	654	157	1,547	787	7,190	78,023

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

* Date of report is the date the public health unit received the report.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 to 31 October 2000[1]

State or Territory	Laboratory	This period	Total this period[2]
Australian Capital Territory	The Canberra Hospital	-	-
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	134	170
	New Children's Hospital, Westmead	-	-
New South Wales	Repatriation General Hospital, Concord	-	-
	Royal Prince Alfred Hospital, Camperdown	42	44
	South West Area Pathology Service, Liverpool	-	-
Queensland	Queensland Medical Laboratory, West End	551	535
	Townsville General Hospital	7	7
South Australia	Institute of Medical and Veterinary Science, Adelaide	566	643
Tasmania	Northern Tasmanian Pathology Service, Launceston	8	12
	Royal Hobart Hospital, Hobart	-	-
Victoria	Monash Medical Centre, Melbourne	-	-
	Royal Children's Hospital, Melbourne	127	122
	Victorian Infectious Diseases Reference Laboratory, Fairfield	135	149
Western Australia	PathCentre Virology, Perth	-	-
	Princess Margaret Hospital, Perth	132	125
	Western Diagnostic Pathology	3	0
Total		1,705	1,807

1. The complete list of laboratories reporting for the 12 months, January to December 2000, will appear in every report from January 2000 regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.
2. Total reports include both reports for the current period and outstanding reports to date.

\- Nil reports

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 October 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Mumps virus	-	-	-	-	-	-	1	-	1	4	41	50
Rubella virus	-	1	-	4	1	-	1	-	7	18	39	266
Hepatitis viruses												
Hepatitis A virus	-	-	1	3	-	-	1	-	5	24	126	406
Hepatitis E virus	-	-	-	-	-	2	-	-	2		4	1
Arboviruses												
Ross River virus	-	1	-	33	6	-	-	-	40	77	1,138	1,487
Barmah Forest virus	-	-	-	7	1	-	-	-	8	8	129	187
Adenoviruses												
Adenovirus type 1	-	-	-	-	1	-	-	-	1		6	8
Adenovirus type 3	-	-	-	-	1	-	-	-	1	1	17	23
Adenovirus type 6	-	-	-	-	2	-	-	-	2		2	
Adenovirus type 7	-	-	-	-	-	1	-	-	1	1	5	5
Adenovirus not typed/pending	2	12	-	2	29	-	6	8	59	103	803	910
Herpes viruses												
Cytomegalovirus	1	13	5	72	37	2	30	5	165	80	1,029	1,064
Varicella-zoster virus	2	3	3	42	10	1	14	1	76	156	1,039	1,741
Epstein-Barr virus	-	2	2	55	73	-	12	1	145	191	1,609	2,519
Other DNA viruses												
Parvovirus	-	1	-	38	1	-	4	-	44	31	288	477
Picornavirus family												
Echovirus type 11	-	1	-	-	-	-	-	-	1	16	7	149
Poliovirus type 1 (uncharacterised)	-	2	-	-	-	-	-	-	2	5	12	22
Poliovirus type 2 (uncharacterised)	-	1	-	-	-	-	-	-	1	4	5	13
Poliovirus type 3 (uncharacterised)	-	2	-	-	-	-	-	-	2	2	7	8
Rhinovirus (all types)	-	8	-	1	-	-	-	-	9	56	292	377
Enterovirus not typed/pending	-	-	-	-	-	2	3	-	5	48	663	669
Ortho/paramyxoviruses												
Influenza A virus	3	13	-	7	118	-	14	30	185	64	1,121	2,146
Influenza B virus	-	11	-	4	28	1	8	2	54	22	456	292
Parainfluenza virus type 1	-	-	-	-	2	-	-	-	2		222	40
Parainfluenza virus type 2	-	-	-	-	2	-	-	1	3	2	34	100
Parainfluenza virus type 3	-	2	-	3	9	-	4	48	66	135	294	693
Respiratory syncytial virus	-	7	-	2	13	1	7	8	38	191	2,586	3,329
Other RNA viruses												
Rotavirus	-	48	-	-	65	-	31	22	166	184	1,349	1,817
Norwalk agent	-	-	-	-	2	-	32	-	34	3	51	54
Other												
Chlamydia trachomatis not typed	1	38	7	94	75	2	8	7	232	291	2,446	3,812
Chlamydia psittaci	1	-	-	-	-	-	8	-	9	6	76	68
Mycoplasma pneumoniae	-	-	-	24	11	-	20	1	56	135	502	1,267
Mycoplasma hominis	-	1	-	-	-	-	-	-	1	1	7	5
Coxiella burnetii (Q fever)	-	2	-	7	1	-	3	-	13	26	67	310
Rickettsia prowazeki	-	-	-	-	-	-	1	-	1		2	
Streptococcus group A	-	2	4	30	-	-	11	-	47	58	294	584
Yersinia enterocolitica	-	2	-	2	-	-	-	-	4	1	13	13
Bordetella pertussis	-	11	-	2	35	-	29	-	77	85	520	1,077
Legionella pneumophila	-	-	-	-	1	-	4	-	5	1	37	18

Table 5 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 October 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Legionella longbeachae	-	-	-	-	2	-	-	-	2	6	45	29
Legionella species	-	-	-	-	-	-	3	-	3		5	
Cryptococcus species	-	1	-	-	1	-	-	-	2	1	13	7
Leptospira species	-	-	-	2	1	-	-	-	3	6	45	83
Treponema pallidum	-	1	40	40	39	-	-	-	120	118	673	1,115
Entamoeba histolytica	-	-	-	1	-	-	2	-	3	2	14	5
Toxoplasma gondii	-	-	-	-	1	-	1	1	2	1	13	6
Total	10	186	62	475	568	12	258	135	1,705	2,164	18,146	27,252

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. 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.

\- No data received this period.

Table 6. Australian Sentinel Practice Research Network reports, weeks 39 to 42, 2000

Week number	39		40		41		42	
Week ending on	1 October 2000		8 October 2000		15 October 2000		22 October 2000	
Doctors reporting	62		62		54		52	
Total encounters	7,698		6,778		6,038		6,421	
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
Influenza	93	12.1	57	8.4	54	8.9	31	4.8
Chickenpox	10	1.3	5	0.7	6	1.0	13	2.0
Gastroenteritis	76	9.9	49	7.2	47	7.8	65	10.1
Gastroenteritis with stool culture	12	1.6	12	1.8	5	0.8	12	1.9
ADT immunisations	36	4.7	40	5.9	28	4.6	21	3.3

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of close to 50 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia New Zealand and the National Public Health Partnership. 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 Commun Dis Intell *2000;24:6-7.*

LabVISE is a sentinel reporting scheme. Currently 17 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. Data are collated and published in Communicable Diseases Intelligence *monthly. 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* Commun Dis Intell *2000;24:10.*

ASPREN currently comprises about 120 general practitioners from throughout the country, not all of whom report each week. Between 7,000 and 8,000 consultations are reported each week, with special attention to 14 conditions chosen for sentinel surveillance in 2000. Communicable Diseases Intelligence *reports the consultation rates for five of these. For further information, including case definitions, see* Commun Dis Intell *2000;24:7-8.*

Additional Reports

Australian encephalitis: 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 29 flocks are maintained in the north of Western Australia, seven in the Northern Territory, nine 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 and details of the locations of chicken flocks see Commun Dis Intell *2000;24:8-9.*

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Sentinel chicken serology was carried out for 23 of the 29 flocks in Western Australia in September and October 2000. Only a single seroconversion to Murray Valley Encephalitis (MVE) virus was detected in the Kimberley region during this period. In early September one chicken seroconverted to MVE virus in the flock located in the Bidyadanga Aboriginal community, south of Broome. In the Pilbara region there were five seroconversions to MVE virus in September and all occurred in the first half of the month. One seroconversion was detected in both Karratha and Exmouth and three as yet unconfirmed seroconversions were detected in Onslow. There was no evidence of flavivirus activity in Western Australia in October.

Serum samples from six of the seven Northern Territory sentinel chicken flocks were tested in our laboratory in September and October 2000. One new seroconversion to MVE virus was detected in October at Beatrice Hill Farm, east of Darwin, but this has not yet been confirmed.

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 (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in HIV/AIDS and related diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: (02) 9332 4648. Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 30 June 2000, as reported to 30 September 2000, are included in this issue of Commun Dis Intell *(Tables 7 and 8).*

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 June to 30 June 2000, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2000	This period 1999	Year to date 2000	Year to date 1999
HIV diagnoses	Female	0	2	0	0	0	0	2	0	4	5	39	34
	Male	0	19	0	11	1	0	17	3	51	35	318	307
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total[1]	0	22	0	11	1	0	19	3	56	40	359	341
AIDS diagnoses	Female	0	0	0	0	0	0	1	0	1	1	9	7
	Male	0	4	0	1	0	1	4	1	11	7	89	71
	Total[1]	0	4	0	1	0	1	5	1	12	8	98	78
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	4	2
	Male	0	6	0	0	0	0	2	0	8	8	43	53
	Total[1]	0	6	0	0	0	0	2	0	8	8	47	56

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

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2000, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	28	613	9	158	61	5	220	119	1,213
	Male	225	11,060	108	2,026	681	78	3,945	928	19,051
	Sex not reported	0	246	0	0	0	0	24	0	270
	Total[1]	253	11,940	117	2,191	742	83	4,203	1,051	20,580
AIDS diagnoses	Female	9	188	0	49	25	3	71	26	371
	Male	87	4,683	35	830	347	45	1,645	356	8,028
	Total[1]	96	4,883	35	881	372	48	1,724	384	8,423
AIDS deaths	Female	4	113	0	32	15	2	49	17	232
	Male	66	3,192	24	570	231	29	1,279	250	5,641
	Total[1]	70	3,313	24	604	246	31	1,334	268	5,890

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

In case you missed it

Influenza and tuberculosis

A study from the University of California at Berkeley (*Population and Development Review*, Vol 26, No 3, September 2000) suggests that the large number of the deaths in 1918, when approximately 500,000 Americans died in an influenza pandemic, was due to a concurrent outbreak of tuberculosis (TB). At the time, factory employees worked in close, poorly ventilated conditions, allowing TB to spread. Studies of bodies recovered from the Alaskan tundra showed that people aged 20-40 years (the group most affected by TB) were most likely to die in the 1918 pandemic. After the pandemic, deaths from TB fell sharply because, the researchers suggest, influenza could have killed many of the TB patients.

Public Health Dispatch: Outbreak of Poliomyelitis — Cape Verde, 2000

According to Center for Disease control and Prevention (CDC. Poliomyelitis prevention in the United States: updated recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 2000;49:RR-5) during August 16 - October 17, 2000, 33 cases of acute flaccid paralysis (AFP), including seven (21%) deaths, were reported in Cape Verde, an archipelago of 10 islands west of Senegal and Mauritania. Preliminary laboratory results identified wild type 1 poliovirus among eight cases. The first patient was a child aged 2 years from the capital city of Praia; paralysis onset occurred 16 August. The child had received one dose of the recommended three doses of oral poliovirus vaccine (OPV). Twenty-two cases were reported from the island of Santiago, seven from Sal, three from San Vincente, and one from Maio. The ages of the AFP patients ranged from 3 months to 38 years; eleven (33%) were aged under 5 years, fifteen (46%) were 5 to14 years, and seven (21%) were ≥15 years. No deaths were reported among patients aged less than 5 years. Three deaths (case fatality rate (CFR): 20%) occurred among patients aged 5 to14 years and four deaths occurred among patients aged ≥15 years (CFR: 57%). Of 33 cases with known vaccination status, 13 (39%) were fully vaccinated.

The estimated population of Cape Verde in 2000 was 437,500 (World Health Organization (WHO), unpublished data, 2000). Reported routine vaccination coverage with three doses of OPV has been <80% every year since 1995. The country has not conducted mass vaccination campaigns against poliomyelitis and has not established AFP surveillance. In response to the outbreak, a mass vaccination campaign was initiated 16 October with the goal of vaccinating every child aged 0 to 59 months with two OPV doses. Investigations by the Cape Verde Ministry of Health and WHO are under way to determine the circumstances associated with the outbreak, whether the outbreak has spread to other territories (such as the neighbouring countries of West Africa), and whether additional interventions will be required to control the outbreak, including a mass campaign targeting persons aged 5 to14 years.

Travellers to Cape Verde and West Africa who are not vaccinated adequately must be considered at risk for polio. Recommendations for children in the United States include a four-dose vaccination series with inactivated poliovirus vaccine (IPV) at ages 2, 4, and 6 to18 months, and 4 to 6 years. Unvaccinated adults should receive three doses of IPV, the first two doses at 4- to 8-week intervals and the third dose 6 to12 months after the second. If three doses cannot be administered within the recommended intervals before protection is needed, alternative schedules are proposed. For incompletely vaccinated persons, additional IPV doses are recommended to complete a series. Booster IPV doses should be considered for persons who have completed a primary series of poliovirus vaccination and who may be travelling to areas where poliomyelitis is endemic.

Drug-resistant cholera in India attributed to antibiotic misuse

Contributed by Ganapati Mudur New Delhi. Source BMJ 2000;321:1368 (2 December 2000) (Edited)

A new study, published in the Indian *Journal of Medical Research* (2000;112:78-85), claims that strains of *Vibrio cholerae* may be transferring multi-drug resistant genes among each other. Strains of *V cholerae* in India have

become resistant to several antibiotics, and multi-drug resistance is increasing, researchers at the National Institute of Cholera and Enteric Diseases in Calcutta have reported. Over the past 6 years strains of *V cholerae* isolated from patients in eastern India show resistance to several old as well as new antibiotics (ampicillin, tetracycline, furazolidone, norfloxacin, and ciprofloxacin). Resistance has moved from the *Vibrio cholerae* serotypes non-01 and non-0139 associated with sporadic cholera, to the serotypes 01 and 0139 associated with epidemics,

Diarrhoeal diseases account for around 600,000 deaths each year in India in children aged under 5 years. Despite advice to doctors and the public against it, the routine use of antimicrobial agents in diarrhoea is still 'capricious' with government surveys showing that nearly half of patients with diarrhoea in India are given antimicrobial agents, though less than 15 per cent really need them. Dr Sujit Bhattacharya, director of the National Institute of Cholera, claims the emergence of resistance to specific antibiotics almost parallels the sequence in which they were introduced.

During the early 1990s multi-drug resistant *Salmonella typhi* appeared across India and this has also been attributed to the indiscriminate use of antibiotics. Pharmaceutical companies produce drugs containing two antimicrobial agents - for example, combinations of norfloxacin and metronidazole, or ciprofloxacin and tinidazole. Dr Bhattacharya claims such irrational combinations are often wrongly prescribed for diarrhoea.

In an attempt to track drug resistance nationwide and to correlate the different serotypes of *V cholerae* with drug susceptibility, the National Institute of Cholera and Enteric Diseases has begun to train Indian microbiologists in molecular epidemiology techniques.

Overseas briefs

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://ww.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Vaccine protects monkeys from Ebola virus infection

Contributed by C Griot. Source: BBC on-line, 29 November 2000 (edited)

Researchers have produced a vaccine that will protect monkeys from Ebola virus infection. Dr Gary Nabel, Director of the Dale and Betty Bumpers Vaccine Research Center (VRC) at the US National Institutes of Health claims that studies show animals can launch an effective immune response against Ebola virus. The new vaccine was tested at a high-containment facility run by the US Centers for Disease Control and Prevention. The researchers, who report their work in the journal 'Nature,' exposed eight macaques to a lethal dose of Ebola virus. Only the four animals given the new vaccine survived.

The vaccine worked on two levels. First, the monkeys were given injections of DNA that produced in their systems certain proteins found on the surface of the virus. These were sufficient to stimulate an immune response but did not make the animals ill. The response was then boosted by exposing the macaques to an adenovirus vector that had been re-engineered to express another specific component of the virus. This further increased the production of antibodies and T cells in the animals to fight infection. More than 6 months after being exposed to Ebola virus, the monkeys in the study remained symptom-free with no detectable virus in their blood.

The multivalent vaccine is to be tested for effectiveness against all three strains of Ebola virus. Dennis Burton and Paul Parren (of the Scripps Research Institute at La Jolla, California) commented in Nature that there was still some way to go before a human vaccine became available; in the meantime the more urgent requirement was to channel resources into surveillance, hygiene and training in barrier nursing, which can be highly effective in containing an outbreak.

Moderators comment. In this paper (Sullivan NJ, Sanchez A, Rollin PE, Yang Z-y, Nabel GJ. Development of a preventive vaccine for Ebola virus infection in primates. *Nature* 2000;408:605-609) the authors describe an effective vaccine strategy for Ebola virus infection in non-human primates that involves a combination of DNA immunisation and boosting with adenoviral vectors that encode viral proteins generated cellular and humoral immunity in cynomolgus macaques. Challenge with a lethal dose of the highly pathogenic, wild-type, 1976 Mayinga strain of Zaire Ebola virus resulted in uniform infection in controls, who progressed to a moribund state and death in less than one week. In contrast, all vaccinated animals were asymptomatic for more than 6 months, with no detectable virus after the initial challenge. The authors conclude that these findings demonstrate that it is possible to develop a preventive vaccine against Ebola virus infection in primates. The Zaire Ebola virus was chosen because it exhibits the highest lethality of known Ebolaviruses. This is a very significant result, although the practicality of such an immunisation strategy in rural Africa must be open to question.

France acts on BSE

Source: The Times (London), 15 Nov 2000 (edited)

French Prime Minister Lionel Jospin has announced a ban on T-bone steaks and a moratorium on the use of animal products in livestock feed. The measures are intended to calm public fears in France over the rising cases of BSE. Other measures announced include random tests on cattle entering slaughterhouses and more funds for research into BSE.

French restrictions in force since 1990 ban the use of meat and bone meal in cattle feed, but the new temporary ban will apply to all livestock fodder. The new ban has been prompted by fears that feeds containing cattle products have been accidentally or deliberately fed to cattle, despite the existing ban. The ban on T-bone steaks is intended to eliminate from the food chain the vertebrae of cattle. Butchers will also be ordered to provide a new cut of cote de boeuf, a traditional French dish.

Last week Agriculture Minister Jean Glavany rejected a proposal by farmers to slaughter millions of cattle in an attempt to wipe out the disease. Mr Glavany said it would be too costly and would only create more 'psychosis' among consumers.

BSE in Spain

Contributed by Jack Woodall. Source: BBC TV, 22 November 2000

On 21 November 2000 Spain reported its first cases of BSE in two cows originally imported from Austria and the Netherlands, respectively. Austria has never reported a case of BSE; the Netherlands has reported only five. The finding is due to upgrading of inspection procedures in Spain in preparation for a Europe-wide inspection program to be introduced on 1 January 2001.

First case of BSE in Germany

Contributed by Stefan Brockmann Source: Associated Press, 25 November 2000 [edited]

After a meeting of the 'bund-laender BSE crisis commission', the German Ministry of Agriculture issued a statement on 25 November 2000 that feeding of animals on bone meal will be illegal in Germany, beginning 29 November 2000.

This statement was made after the first case of BSE in Germany was confirmed by the national reference centre in Tuebingen. The Government of the State of Schleswig Holstein, where the suspected cow was stabled, has arranged a BSE hotline.

The first suspected case of vCJD, a 22 year old male Bavarian patient, suffering from progression of the disease for 18 months, was also reported by the press. However, the head of the Division of Neuropathology at the Munich 'Klinikum GroDFhadern', could not confirm or deny vCJD or the sporadic form of the disease.

BSE in Germany

From: H. Larry Penning, MD Source: Reuters, 27 November 2000 (edited)

German authorities said a backup test confirmed the country's first case of BSE. Heide Simonis, state premier of the northern region of Schleswig-Holstein, said a second test by federal health authorities confirmed the results of an earlier test on Friday.

The German government, which initially resisted strict measures, reversed itself and called for tests on cattle throughout the European Union (EU).

The Agriculture Minister has demanded fast national testing of slaughtered cattle and urged mandatory testing throughout the EU and on beef imported into the EU.

State and federal ministers also agreed at a meeting in Bonn to back Schroeder's call for an immediate ban on the import, export and use of animal feeds containing meat and bonemeal.

The measure will become law on 29 November 2000, and Germany will push for an EU-wide ban in December. Infected feeds have been blamed for the spread of BSE and the human form of mad cow disease, variant Creutzfeldt-Jakob Disease (vCJD).

Mosquito diseases could reach epidemic levels

Contributed by Environmental Health News. Source: Australian Associated Press. 21 November 2000 (edited)

The New South Wales Health Minister Craig Knowles warned that mosquito-borne diseases could reach epidemic levels in NSW because of the floodwaters that had inundated a third of the State. Potentially the biggest breeding ground for mosquitoes in years has resulted from a combination heavy rain and humid weather.

Mr Knowles said outbreaks of diseases such as Ross River Fever and Barmah Forest Disease usually occur from February to March but the dramatic increase in the number of mosquitoes was likely to bring these outbreaks forward. People are also being warned to beware the rarer and deadly Murray River Encephalitis. All three diseases are characterised by nausea, vomiting, diarrhoea and fever, as well as stiffness in the neck and upper back. People are advised to use strong insect repellents and mosquito nets and to cover up with clothing. They are also advised to clean out house gutters, to keep pools chlorinated, salted or empty, and to remove other sources of water lying around in which mosquitoes can breed.

Up to the end of July, 624 cases of Ross River and Barmah Forest Disease have been reported to NSW Health - a third of those coming from the Hunter, New England and areas of the mid-north coast. The reports come mostly from the mid-winter months when mosquito activity is usually at its lowest.

Update on cholera outbreak on Pohnpei

Contributed by J P Chaine, PIHOA Regional Epidemiologist (edited)

As of December 3, the total number of cases of Cholera on Pohnpei is 3,429. This is broken down to: 1,814 cases from OPD/ER that were treated and sent home, 792 cases admitted to Pohnpei hospital, 782 seen in the outlying dispensaries, and 41 seen at the two private clinics. Nineteen deaths have been reported but this may be under-reported because of the stigma attached to cholera. In the period 27 November to 3 December all units saw only 11 cases. The hospital has not had an admission since November 19. All cases of cholera are from the main island of Pohnpei; there has been no exportation to neighbouring islands.

The oral cholera vaccination campaign reached about 15,000 people on Pohnpei proper (a 50% coverage). The vaccine is still available, but demand is mostly from travellers going to Chuuk and Kosrae.

Kosrae received 7,000 doses of vaccine and requested another 500 doses on 1 December. This will bring their coverage into the high 90's per cent.

Chuuk has vaccine on the island and the authorities are currently planning their campaign. The main island of Weno and the lagoon islands are their first priority.

Poliomyelitis - Dominican Republic & Haiti: alert

Contributed by Epstein, Mr. Daniel B. (WDC) Source: Pan American Health Organization, Regional Office for the Americas

A current outbreak of poliomyelitis in the Dominican Republic and Haiti has raised serious concerns because the Western Hemisphere has been free of wild poliovirus circulation since 1991, and because the virus identified is an unusual derivative of the Sabin type 1 oral poliovirus vaccine (OPV), according to Dr Ciro de Quadros, who directs the Pan American Health Organization's Division of Vaccines and Immunization.

The Ministries of Health of the Dominican Republic and Haiti, with the assistance of the Pan American Health Organization (PAHO) and the Centers for Disease Control and Prevention (CDC), are investigating the outbreak to determine the extent of spread and to evaluate the reasons for the outbreak. Aggressive control measures have already been put in place. A mass vaccination campaign with OPV has already started in the Dominican Republic, initially covering the three provinces with suspected cases, followed shortly by the rest of the country. In Haiti, three nationwide vaccination rounds with OPV are planned for January, February and March 2001. Since 12 July 2000, a total of three laboratory-confirmed cases due to (vaccine-)derived poliovirus type 1 isolates have been identified. An additional 16 persons with acute flaccid paralysis (AFP) are now under investigation in the Dominican Republic. To date in Haiti, a single laboratory-confirmed case due to the (vaccine-) derived type 1 virus has been reported, with paralysis onset on 30 August 2000. After intensive case-finding activities, no other cases have been found so far. The virus detected, first isolated by the PAHO Poliovirus Laboratory at the Caribbean Epidemiology Center and subsequently characterized at the Poliovirus Laboratory CDC, is unusual because it is derived from OPV, has 97 per cent genetic similarity to the parental OPV strain, and appears to have assumed the characteristics of wild poliovirus type 1, both in terms of neurovirulence and transmissibility. The difference in nucleotide sequence suggests the virus has been either replicating for a prolonged period in an immunodeficient individual, or circulating for as long as 2 years in an area where vaccination coverage is very low, resulting in ongoing genetic changes in the original Sabin virus that gave it the properties of wild poliovirus.

Prolonged circulation of OPV-derived polioviruses in areas with very low OPV coverage has been documented in only one other setting — type 2 OPV-derived virus circulated in Egypt for an estimated 10 years (1983-1993) and was associated with more than 30 reported cases. In this instance, vaccination coverage was very low in the affected areas, and circulation of a vaccine-derived poliovirus was terminated rapidly once OPV vaccination coverage increased. The key factor for control of circulating OPV-derived viruses is the same as that required to control wild poliovirus circulation: achieving and maintaining high vaccination coverage. Dr de Quadros said that: 'No evidence for circulation of OPV-derived virus has ever been found in any area with high coverage. The current outbreak is a powerful reminder that even polio-free areas need to maintain high coverage with polio vaccine until polio eradication has been achieved. Nearly 4 decades of experience with OPV has shown that it is very safe and effective in preventing poliomyelitis. OPV is the vaccine of choice for the eradication of wild polioviruses. However, it is crucial to maintain high OPV coverage to protect against imported wild polioviruses and to prevent person-to-person transmission of OPV-derived viruses.' He added that: 'It is also important that all countries maintain high quality AFP and poliovirus surveillance, that current activities to complete the global eradication of wild polioviruses be accelerated, and that a global strategy is developed for the orderly cessation of immunisation with OPV after global certification of polio eradication is achieved.'

Travelers to the Dominican Republic and Haiti who are not adequately immunised must be considered at risk of acquiring poliomyelitis, and should make certain they are fully immunised against poliomyelitis. Those countries using OPV for routine immunisation recommend at least a 3-dose primary vaccination series.

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