Public health implications of dengue in personnel returning from East Timor

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Abstract

In north Queensland the vector of dengue fever (*Aedes aegypti*) is present; hence any viraemic individual importing dengue has the potential to transmit the disease locally. In early 2000 approximately 2,000 personnel returned from East Timor to Townsville, north Queensland. Seven importations of dengue occurred and individual cases were viraemic for up to 6 days in Townsville. No subsequent local transmission occurred. There were 3 cases each of dengue type 2 and dengue type 3. One case could not be serotyped. A response, including mosquito control measures, was initiated in another 18 cases in which dengue fever was clinically suspected but which subsequently proved not to be dengue. The planning and processes undertaken to prevent local transmission of dengue in Townsville during an intense period are described. *Commun Dis Intell* 2000;24:365-368.

Keywords: dengue, type 2, type 3, flavivirus, Aedes aegypti*, East Timor, Queensland, viraemia, transmission, mosquito control*

Introduction

Dengue fever, although not endemic in the region, is an important public health concern in north Queensland which is considered 'receptive' because *Aedes aegypti*, the mosquito vector for dengue, is present.[1] Thus any importation of dengue (via a viraemic individual) could potentially result in local transmission, and initiate an outbreak.

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Dengue fever is an acute febrile illness characterised by frontal headache, retro-orbital pain, myalgia, arthralgia and rash. An individual who has previously been infected with one dengue serotype and is subsequently infected with a different one can potentially suffer a severe form of illness characterised by circulatory failure or haemorrhagic manifestations (dengue haemorrhagic fever).

Since 1995 north Queensland has experienced 6 outbreaks of dengue, all in the Cairns/Port Douglas region or the Torres Strait (Dr Jeffrey Hanna, Queensland Health, Cairns; personal communication). Despite numerous importations to the region, Townsville (approximately 350 kilometres south of Cairns) has not experienced any local transmission since the dengue type 2 epidemic in 1992/3 during which approximately 750 cases were notified to the National Notifiable Diseases Surveillance System.[2] This number is likely to represent only a small proportion of the cases that actually occurred.[3] Therefore a significant number of Townsville residents are likely to have immune systems 'primed' by previous infection and further outbreaks of different dengue serotypes in Townsville could potentially result in severe disease or dengue haemorrhagic fever as described above.

In late 1999 the Tropical Public Health Unit (TPHU) was informed that personnel who had been in East Timor would soon be returning in considerable numbers to Townsville. The majority had been serving as part of the Australian Defence Force, but there were also a few aid workers, journalists and others. Because cases of dengue fever had been reported in East Timor in 1999,[4] a high level of importation of dengue into Townsville early in 2000 was anticipated. This report describes preparations made for, and the public health management of, the importations that did occur.

Methods

Planning for importations of dengue

Disease surveillance activities

Dengue fever is a notifiable disease in Queensland. While surveillance for many notifiable conditions is laboratory-based in the State, the public health implications of dengue fever mean that urgent notification on clinical suspicion (prior to laboratory results becoming available) is requested for dengue in north Queensland.

Clinical surveillance was enhanced by face-to-face meetings and written communication with Defence Force medical staff who were familiarised with the notification procedures with emphasis on early notification. Based on the accepted maximum viraemic period[5] and incubation period[6] for dengue, notification was requested for any cases developing a dengue-like illness within 12 days before, or 14 days after, arriving in Townsville from East Timor.

Medical practitioners in Townsville were also alerted to the situation by letter and reminded of the need for urgent notification of suspected cases.

Mosquito surveillance and control

Preventative measures aimed at reducing *Aedes aegypti* populations in high-risk areas are routine in north Queensland. In late 1999 prior to the arrival of returning personnel, and before the wet season, TPHU, local government and Defence Force staff held planning meetings. Additional mosquito control measures were undertaken including inspections and source reduction at Defence Force bases (where troops returning from East Timor were likely to be concentrated) and around other high risk areas (eg. hospitals).

Source reduction involved either removal of potential breeding sites for *Aedes aegypti* (eg. emptying containers holding water), or treatment of containers not easily emptied (eg. gully traps) with a residual larvicide.[7]

Education and awareness activities

Queensland Health's dengue fever pamphlets outlining information on the disease and dengue preventive measures were made available in large numbers for distribution by the Defence Forces to personnel prior to their departure for East Timor or return to north Queensland. The pamphlet was also made available to all general practitioners in Townsville.

Television advertisements on dengue fever emphasising measures residents can take to reduce the breeding of *Aedes aegypti* are routinely run in north Queensland during the wet season on a paid schedule; these were televised as usual with no additional paid advertising.

Contents, *continued*

Responding to importations of dengue

Disease control activities

TPHU staff obtained a history from each suspected case of dengue as soon as possible after the notification. Details, including clinical information, residential addresses and other places visited by the case while potentially viraemic were obtained. To reduce the possibility of local transmission of dengue, patients were provided with information by the clinician and/or TPHU staff on mosquito bite avoidance during the day, including use of insect repellent and insecticide mats, and advice on measures to reduce mosquito breeding around the home.

Sera collected from suspected cases at the time of the first consultation were tested at local laboratories for dengue IgM by enzyme immunoassay (EIA). EIA-negative sera collected in the first 5 days of illness were referred to Public Health Virology, Queensland Health Scientific Services (QHSS), for reverse transcriptase polymerase chain reaction (RT-PCR) testing, and/or a second specimen was collected several days later for repeat EIA. Confirmatory testing included RT-PCR, virus isolation, or haemagglutination inhibition assay.

For each confirmed case the period of 'viraemia of public health importance'[8] in Townsville was calculated. This referred to the period the case was infectious to mosquitoes and so reflects the potential for transmission. It commenced on the day the case arrived in Townsville or the date of onset of illness (if symptoms began in Townsville). The end of the period was defined as the earlier of 2 dates: either 12 days after the onset of illness or the day of notification to TPHU (ie. the point at which control activities were implemented thereby reducing the risk of transmission).

Mosquito control measures

Mosquito control measures were undertaken if a case, suspected or confirmed, had apparently been viraemic in Townsville. Larval control involved source reduction in premises within 200m of any residence (and other premises) where the case had spent significant amounts of time while viraemic. Adult *Aedes aegypti* control involved interior insecticide spraying of premises within 100 m of the case.[7]

Results

Approximately 2,000 personnel returned from East Timor to Townsville in the first few months of 2000. Over a 5-week period in January and February 2000 there were 7 confirmed importations of dengue fitting the 'viraemic' time interval described to be of public health importance. Six cases were Defence Force personnel and 1 case was an aid worker. There were 3 cases each of dengue type 2 and type 3; one could not be serotyped. No subsequent transmission of dengue occurred in Townsville.

The delay in notification of cases ranged from 0 to 4 days. Three of the cases (43%) were notified by the medical practitioner on the day of consultation. In total 5 (71%) of the cases were notified within 48 hours. The 2 longer delays occurred because the patients were notified by the testing laboratory and not on clinical suspicion.

The combined total of days of 'viraemia of public health importance' in Townsville for the 7 cases was 21 days (range 0 to 6 days for individual cases). Three cases were already unwell on arrival, and 4 became unwell after arrival in Townsville.

In addition to the 4 confirmed cases, TPHU (in collaboration with local government staff) responded to18 other notified suspected cases. Of these, 4 eventually proved to be malaria and 4 had evidence of recent flavivirus infection but with a type not specified due to cross-reacting flavivirus antibodies; no specific diagnosis was determined for the other ten. Thirteen additional notifications were received in returning personnel who were dengue EIA IgM-positive but who were no longer viraemic upon arrival in Townsville. As they were of no public health significance, no specific action was taken in relation to these cases.

Discussion

Despite an intense period with multiple importations into this receptive area, no subsequent local transmission of dengue occurred in Townsville. Prior preparedness and close collaboration between TPHU, Defence Force and local government staff contributed to this outcome.

A management plan for dengue fever in north Queensland has been developed and recently revised.[9] This plan drew on the experience of those involved in management of cases and outbreaks of dengue both in north Queensland and overseas, including input from experts from the Centers for Disease Control and Prevention, USA and from Singapore. The plan describes the preventive and responsive strategies that are currently used to control dengue in north Queensland. Measures implemented in Townsville in late 1999 and early 2000 add support to the value of this dengue fever management plan.

The timeliness of notifications of clinically suspected importations of dengue was considerably better than that previously documented in north Queensland. Seventy-one percent of all notifications were received within 48 hours compared with only 26 per cent in a previous study.[8] This promptness reflects what can be achieved when patients consult informed medical practitioners who are well aware of the public health implications of dengue.

Because dengue is not endemic in north Queensland, the public health response to suspect imported cases reflects the need to minimise risk of local transmission. For example, the maximum period of viraemia (12 days) is used to define 'viraemia of public health importance' for each imported case even though it may commonly be a shorter period.[5] Likewise any case in which dengue fever is considered a possible diagnosis is responded to immediately.

This strategy meant that mosquito control measures were implemented in response to a considerable number of suspected cases that subsequently proved not to be dengue. Although this may be considered resource intensive, such a response is necessary. Awaiting laboratory results would have introduced unacceptable delays and allowed the proliferation and dispersal of *Aedes aegypti* mosquitoes with increased risk of local transmission.

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Murray Valley encephalitis in Western Australia in 2000, with evidence of southerly spread

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Abstract

We describe the epidemiological and clinical features of human Murray Valley encephalitis (MVE) and Kunjin (KUN) virus infections in Western Australia (WA) during March to July 2000. A case series was performed. For laboratory-confirmed cases, travel histories and clinical details were collected from patients, family members, friends or treating physicians. Surveillance data from the sentinel chicken program and climatic conditions were reviewed. Nine encephalitic cases of MVE were recorded. Eight were non-Aboriginal adults (age range, 25 to 79 years; 5 male, 3 female) and 1 was an Aboriginal boy. Four cases acquired infection in the Murchison and Midwest regions of WA from which no human cases of MVE have been reported previously. One of the 9 cases was fatal and 3 had severe neurological sequelae. Five non-encephalitic infections were also recorded, 3 MVE and 2 KUN. Encephalitis caused by MVE virus remains a serious problem with no improvement in clinical outcomes in the last 25 years. Excessive rainfall with widespread flooding in the northern two-thirds of WA provided ideal conditions for mosquito breeding and favoured southerly spread of the virus into new and more heavily populated areas. Surveillance in WA with sentinel chickens and mosquito trapping needs expansion to define the boundaries of MVE virus activity. To enable timely warnings to the public, and to institute mosquito control where feasible, continued surveillance in all Australian areas at risk is indicated. *Commun Dis Intell* 2000;24:368-372.

Keywords: Murray Valley encephalitis virus, Kunjin virus, arbovirus, mosquito, outbreak

Introduction

Murray Valley encephalitis virus (MVE)* is a mosquito-borne flavivirus, named after outbreaks of disease centred on the Murray-Darling River Basin in south-eastern Australia, the last and largest of which occurred in 1974.[1] Subsequently MVE was found to be endemic in the Kimberley region of far northern Western Australia (WA) and adjacent areas of the Northern Territory (NT) where it appears to be maintained in a waterbird-mosquito cycle.[2] In these areas, regular activity leads to very high infection rates within the resident Aboriginal populations.[3,4]

Only about 1 in 1,000 infections with MVE results in clinical encephalitis,[5] while the rest cause an asymptomatic infection or a non-specific febrile illness. Occasional cases

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of flavivirus encephalitis are due to the closely related Kunjin virus (KUN) though this manifestation of infection is much less common than with MVE. Limited published data[5] and unpublished anecdotal reports suggest that Kunjin virus more often causes a febrile illness with headache, myalgia, arthralgia and sometimes a maculopapular rash.

In clinical case series reported from WA and the NT,[5,6] MVE/KUN encephalitis had a case fatality rate of 24 per cent with a further 56 per cent having neurological sequelae. The incubation period is estimated to be between 1 and 4 weeks,[7] after which time clinical cases abruptly develop fever, headache, malaise and, possibly, altered mental state, anorexia, nausea, vomiting and dizziness. Children usually present with convulsions, while tremor and cranial nerve palsies can develop in patients of any age. Severe cases may progress to coma, respiratory failure and death.[5,6]

Culex annulirostris is the major vector of MVE in Australia and this mosquito species readily feeds on birds, as well as a range of mammals including humans.[8] MVE activity is monitored by detecting specific antibodies in serum samples from sentinel chicken flocks. Flocks of 12 chickens are located in populated areas or at major dams in northern WA, covering the Kimberley, Pilbara, Gascoyne, Murchison and Midwest regions (Figure). Blood samples are collected from flocks fortnightly during and immediately following the wet season and monthly at other times.

In WA, 14 cases of MVE encephalitis were notified between 1990 and 1998.[9] Nine were during the 1993 wet season. Disease typically occurred in Aboriginal children residing in the Kimberley or among those of any age travelling through, or recently moved to, the region.[5,10] In the past, epidemic activity has occasionally been recorded in the Pilbara and Gascoyne regions with cases in both areas in 1981 and a case possibly acquired in the Gascoyne in 1997.[5]

In 2000, 9 cases of MVE encephalitis were identified which, unlike other years, occurred mainly south of the Kimberley; in addition, there were 5 non-encephalitic MVE/KUN infections in WA. In this paper we report on the epidemiological and clinical features of these MVE/KUN cases and review surveillance data from the sentinel chicken program and the environmental conditions observed during the 2000 wet season.

Methods

Case Series

We collected travel histories and clinical details from patients, family members, friends and treating physicians. For each encephalitic case, a member of the medical team was interviewed by telephone using a structured questionnaire to document the likely place of exposure, clinical severity of illness and outcome.

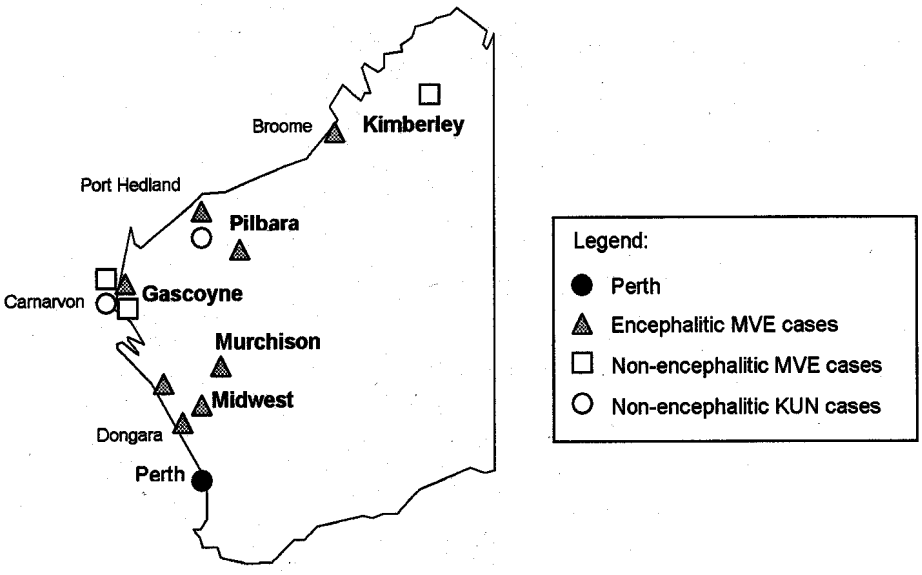
Laboratory testing

Sera were tested at the Western Australian Centre for Pathology and Medical Research using a standard flavivirus haemagglutination inhibition (HI) test and IgM to MVE and KUN was detected by an indirect immunofluorescence assay (IFA) in serum and cerebrospinal fluid (CSF) samples.[5,6] Where the HI titre failed to show a rise, IgG titres were also determined by IFA. Specific IgG to MVE or KUN was titred using a competitive enzyme immunoassay (EIA).[11] MVE-RNA in CSF or brain tissue was detected using reverse transcription, then DNA amplification by polymerase chain reaction (RT-PCR).[12]

For encephalitic illness, definite and presumptive cases were included. A definite case was a patient with a rising HI titre (at least fourfold) or rising IFA-IgG in serum, and/or detection of MVE-RNA by RT-PCR in CSF or brain tissue, or a positive HI titre and IFA-IgM with an exposure history limited to this wet season. A presumptive case was a case of encephalitis with a positive flavivirus HI titre and IFA-IgM in serum and/or CSF, without a rising antibody titre or without a documented exposure history limited to this wet season.

Non-encephalitic cases were included if there was a rising HI titre or rising IFA-IgG, or if a positive HI titre and IFA-IgM

Figure. Cases of Murray Valley encephalitis, Western Australia, 1 January to 20 August 2000, by region



Case 5 not shown as the most likely place of infection is unclear
Regions are shown in bold

with an exposure history limited to this wet season was documented.

Cases were assigned as MVE or KUN based on the competitive EIA results and the RT-PCR in the CSF or brain tissue.

Results

Nine cases of encephalitis caused by MVE putatively acquired in WA between March and May 2000 were recorded (Table). Six were definite and 3 presumptive. Three females and 6 males were affected. Eight cases were non-Aboriginal adults, aged between 25 and 79 years, and 1 case was an Aboriginal boy of 10 months. For the first time, MVE infection acquired in the Murchison and Midwest regions was identified, with 1 and 3 clinical cases, respectively. One case acquired the illness in the town of Dongara, only 315 km north of Perth (Figure).

Travel histories

Four cases had not travelled in the month preceding illness and presumably acquired infection in their region of residence (Pilbara, Murchison and Midwest). Two worked on remote stations and 2 lived in towns. High numbers of mosquitoes and extensive ground water were reported near their abodes and 2 cases distinctly recalled being heavily bitten by mosquitoes in the weeks prior to illness onset.

The remaining 5 cases were travellers to northern WA. The 10-month-old child probably acquired the infection while camped south of Newman in the Pilbara. The 55-year-old male was almost certainly infected in Kalbarri in the Midwest, where he had been staying between 13 March and 6 April. The 64-year-old male went on a fishing expedition with 4 male companions in late March. The party reported being heavily bitten by mosquitoes while camping north of Carnarvon in the Gascoyne and infection presumably occurred there. The 25-year-old male travelled on a road tour from Perth to Broome between 13 and 20 April and became ill on 25 April. The 79-year-old female departed Geraldton by road on 23 April, travelling to far-north WA before arriving in Katherine in the NT on 27 April, 11 days before she fell ill. Her 2 fellow travellers reported being heavily bitten by mosquitoes at a roadside truck stop in the Kimberley and, as sentinel chicken data also showed high levels of virus activity, this was the most likely site of infection. By comparison, her companions noted few mosquitoes in Katherine and sentinel chicken data on 20 April revealed only 1 MVE seroconversion there.

Clinical features

All 9 cases required hospitalisation and presented with fever, 8 with headache and 7 with various other neurological signs including neck stiffness, photophobia, vomiting, irritability and decreased consciousness (Table). Seven were admitted to an intensive care unit, 5 needed mechanical ventilation and 2 remained ventilator-dependent on 30 June. There was 1 fatal case (11%), while another 3 (33%) had significant neurological sequelae. Of the others, 3 had resolving neurological manifestations and 2 had made a complete recovery by mid-June.

Non-encephalitic MVE cases

Two cases of non-encephalitic illness and 1 asymptomatic case were recorded in WA to August 2000. In early March, a 15-year-old Aboriginal female who lived in the north-east Kimberley, developed headache, fever and myalgia. These symptoms resolved, with documented MVE seroconversion (by competitive EIA) between November 1999 and March 2000. Of the 4 male travelling companions of the holidaying fisherman (case 3), 1 developed a non-encephalitic illness with headache, back pain and nausea but no fever. A convalescent serum sample was positive for MVE IgM and MVE-specific antibody by competitive EIA, and showed a positive flavivirus HI titre. History suggested that exposure had not occurred before the fishing trip. Another companion had similar serology results but remained asymptomatic. Laboratory testing was negative on the third, and no specimen was collected from the fourth.

Non-encephalitic Kunjin cases

A 37-year-old woman from Carnarvon in the Gascoyne developed fever, myalgia, rash and headache at the beginning of May. She recovered fully and samples revealed seroconversion to flavivirus between December 1999 and May 2000, with a positive HI titre to KUN and a positive KUN IgM. The competitive EIA showed antibodies to KUN only. She had not travelled recently and remembered being heavily bitten by mosquitoes prior to illness onset.

The second case involved a 62-year-old man from Port Hedland in the Pilbara, who presented to his general practitioner on 11 July with a history of intermittent myalgia and rash. Serial samples showed a rising antibody HI titre to KUN and competitive EIA confirmed specific KUN antibody only.

Sentinel chicken surveillance and mosquito trapping in WA

Seroconversions to MVE were first detected in sentinel chickens from 2 Kimberley flocks and 1 Pilbara flock in January 2000. MVE activity spread quickly to other areas of the Kimberley and Pilbara in February and further south to the Gascoyne and Murchison in March. In late April, MVE activity was detected 315 km north of Perth, in Dongara in the Midwest (Figure), when 4 of 12 chickens seroconverted.[13] Antibodies to MVE had not been detected previously during the 3 years of testing in the Midwest region. Mosquito collections were also carried out in late March and April in the Kimberley and Pilbara and large numbers of *C. annulirostris* were seen in the traps.

Discussion

There were 9 cases of MVE encephalitis acquired in WA between 1 January and 31 August, 2000. This figure equals the previous record in 1993. A further 5 infections with MVE/KUN were identified, 4 had a non-encephalitic illness and 1 was asymptomatic. In total, this represents the highest recorded number of human infections with MVE/KUN in WA within a single season.

The marked southward migration of the virus in 2000 is of significant public health concern. 'Very much above average' monsoonal rainfall was recorded in the Kimberley and Pilbara from December 1999 to February 2000 (Source: Bureau of Meteorology). In March, there was exceptionally high rainfall resulting from tropical cyclone 'Steve' causing major flooding from the Kimberley to the Midwest. These factors provided ideal conditions for extensive mosquito breeding, allowing MVE transmission cycles to become widely established. Conditions differed significantly from the previous 'record' year in 1993 when flooding was confined to

Table. Diagnostic criteria and clinical features of encephalitic Murray Valley encephalitis cases in Western Australia, 1 January to 20 August, 2000

Case no.	Sex	Age	Ethnicity	Likely place of exposure	Onset date	Diagnostic criteria*	Prodrome	Neurological manifestations	Residual signs
1	M	10 m	A	Newman, **Pilbara**	6 Mar	1, 2, 3	Fever, irritability	Seizures, rigidity, choreiform movements, bulbar palsy, internuclear ophthalmoplegia	Bulbar palsy, hypertension, irritability
2	M	55 y	N	Kalbarri, **Midwest**	8 Apr	2, 3, 4	Fever, headache	Coma, flaccid paralysis	Quadraparesis, ventilator-dependent
3	M	64 y	N	Carnarvon, **Gascoyne**	16 Apr	2, 3, 5	Fever, headache, neck stiffness, photophobia	Decreased consciousness, confusion, memory loss	Resolving
4	M	32 y	N	Meekatharra, **Murchison**	20 Apr	1, 2, 3	Fever, headache, neck stiffness	Decreased consciousness, left-right disorientation, short-term memory loss dizziness, lethargy	Resolving
5	M	25 y	N	**Midwest** to **Kimberley**	25 Apr	1, 3, 6, 7	Fever, headache, malaise, vomiting, disorientation	Coma, flaccid paralysis	Quadraparesis, ventilator-dependent
6	F	41 y	N	Wickham, **Pilbara**	28 Apr	2, 3, 5	Fever, headache, malaise, vomiting	Confusion, short-term memory loss, dysarthria, dysphasia, extra-pyramidal gait disorder, tremor	Resolving
7	M	69 y	N	Mullewa, **Midwest**	3 May	2, 3, 5	Fever, confusion, irritability	Decreased consciousness, disorientation	Nil
8	F	61 y	N	Dongara, **Midwest**	5 May	2, 8	Fever, confusion, drowsiness, nausea	Aphasia, Parkinsonian syndrome	Nil
9	F	79 y	N	Broome, **Kimberley**	8 May	1, 2, 8	Fever, headache, malaise	Coma, spastic paralysis	Deceased 27 May

*

1, Rising serum HI titre;
2, Positive MVE IgM in serum and CSF;
3, Positive monoclonal antibody blocking EIA in serum;
4, Rising serum IFA-IgG titre;
5, Positive HI in serum;
6, Positive MVE IgM in serum;
7, Positive PCR in CSF;
8, Positive PCR in brain tissue.

M = male, F = female; m = months, y = years; A = Aboriginal, N = non-Aboriginal
regions shown in bold.

the Kimberley and Pilbara regions, and human disease to the Kimberley. Sentinel chicken monitoring, for the first time, detected MVE activity in the Midwest. Our first identified human cases of MVE infection in the Murchison and Midwest regions were also recorded.

There is evidence that MVE may persist in the desiccation-resistant eggs of some mosquito species and that this may be important in initiating activity in the Kimberley.[14] Therefore, there is concern that a similar situation may now have been established in the Murchison and Midwest. As seroconversions occurred in our most southerly and easterly sentinel flocks, opportunistic sampling of other flocks is in progress to define these limits and determine future sentinel sites.

The unusually wet weather, MVE seroconversions among sentinel chickens and subsequent occurrence of clinical disease in humans, prompted repeated health warnings from the Health Department of Western Australia to the public via the media, public health units, general practitioners and hospitals. Preventive measures against mosquito bites were advised for people living in or travelling to the Kimberley, Pilbara, Gascoyne, Murchison and Midwest regions.

When MVE virus is present in non-endemic areas, pre-existing immunity within the population is expected to be low. Therefore, in these regions, individuals of all ages are potentially at risk of infection, though our series supports the previous finding that most clinical cases have occurred in adults.[5] Few cases have occurred in adult Aboriginals. There were none in our series, or that of Burrow et al.[6] Aboriginals comprised only 2 of 9 adult cases in the series presented by Mackenzie et al[5] and there was a third case from WA in 1993 (unpublished observation). The low rates in Aboriginal adults in the Kimberley is presumed to result from the high rates of MVE infection in childhood[3,4] which renders adults non-susceptible. For adult Aboriginals in non-endemic regions there are no comparable seroprevalence data to assess whether exposure in childhood also explains the low numbers of cases in this group, or whether it just reflects the proportion of Aboriginal to non-Aboriginal populations in these areas.

The presentation and clinical outcomes of our cases were similar to those described previously for encephalitic MVE.[5,6] Burrow et al[6] summarised the findings from both their case series and those reported by Mackenzie et al.[5] With the addition of our 9 encephalitic cases and the case reported by McMinn et al,[15] the case fatality rate of MVE/KUN encephalitis was 9/46 (19.6%). Among survivors, 8/37 (21.6%) had major neurological sequelae and 12/37 (32.4%) had minor sequelae. Only 17 of 46 (37.0%) were documented to have made a complete recovery.

The pattern of MVE virus infection in WA in 2000 occurred as a result of the unusual environmental conditions. Though these weather patterns are observed infrequently, they create an environment conducive to viral spread into new areas. There is concern that replication of these climatic conditions could pose a threat to the more populous south-western region. This includes Perth, which has a population of 1.3 million and is only 315 km south of Dongara. These events also provide a warning to south-eastern Australia about the ongoing potential for further epidemics in the Murray Valley and their likely impact among these large, and probably immunologically naïve,[16] populations.

*** Abbreviations:**

CSF, cerebrospinal fluid; EIA, enzyme immunoassay; HI, haemagglutination inhibition; IFA, indirect immunofluorescence assay; KUN, Kunjin virus; MVE, Murray Valley encephalitis virus; NT, Northern Territory; RT-PCR, reverse transcription-polymerase chain reaction; WA, Western Australia.

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Editorial: Notifiable disease reporting in the new millennium

The regulation of disease in Australia is vested in the States and Territories; the only specific public health powers vested in the Commonwealth under the Australian Constitution relate to quarantine under the *Quarantine Act 1908*.[1] There are recognised benefits to some degree of harmonisation of public health legislation across Australia;[2] with respect to the surveillance of communicable diseases all jurisdictions have reviewed, or are reviewing, their public health legislation to allow greater flexibility and responsiveness to the need for rapid additions to their notifiable diseases list.

The surveillance of a communicable disease is fundamental for disease prevention and control. The World Health Organization defines surveillance as the '*ongoing systematic collection, collation, analysis and interpretation of data; and the dissemination of information to those who need to know in order that action may be taken*'.[3] Last's definition states that surveillance generally uses *'…methods distinguished by their practicability, uniformity, and frequently their rapidity, rather than by complete accuracy. Its main purpose is to detect changes in trend or distribution in order to initiate investigative or control measures*.'[4]

Public Health Units in the various States and Territories are the bodies which receive reports of cases of notifiable and other infectious diseases of public health importance from clinicians, laboratories and other specified agencies within their jurisdiction. To enable the Commonwealth to monitor disease trends and estimate the burden of disease at a national level, data are forwarded to the Commonwealth Department of Health and Aged Care. In addition, reports of non-notifiable diseases are forwarded by various laboratories contributing to the Commonwealth's *Communicable Diseases Intelligence* Virology and Serology Reporting Scheme and by various other specialised surveillance networks as detailed elsewhere.[5] Also, the Commonwealth receives reports of non-notifiable communicable diseases under sentinel general practice surveillance collected by the Australian Sentinel Practice Research Network.[6] The Surveillance and Management Section, Communicable Diseases and Environmental Health Branch in the Population Health Division of the Department of Health and Aged Care is currently responsible for collating these surveillance data and reporting on them nationally.

There has long been a list of notifiable diseases which, over the years, has been modified as old diseases lose significance and new ones appear. Thus in 1977, for example, infantile diarrhoea, puerperal fever and scarlet fever were notifiable nationally. In the October 1978 revision they were deleted, whereas smallpox continued to be reported and Lassa fever was added.[7] In 1990 the Commonwealth and States and Territories agreed to work towards a national list. In 1991 the National Notifiable Diseases Surveillance System (NNDSS) was established and collection of data at a national level for this list commenced, and in 1994 the National Health and Medical Research Council published case definitions for a list of communicable diseases of national importance.[8] Since then additional diseases have been made notifiable in various States and Territories before being added to the national list; for example Australian bat lyssavirus and/or haemolytic uraemic syndrome were made notifiable in some jurisdictions in the 1990s depending on local imperatives.[9,10]

Although all jurisdictions adopted the 1994 case definitions for notification purposes (and as the basis of local surveillance case definitions) there still remained differences in which diseases they reported to the Commonwealth. This issue was resolved by the Strategic Steering Committee of the Communicable Diseases Network Australia New Zealand (CDNANZ) at its February 2000 meeting where it agreed to a revised national list of diseases to be reported to the NNDSS.[11] For the first time from 1 January 2001, exactly 100 years after Federation, all jurisdictions have agreed to legislate to report all the infectious diseases on the national list. Jurisdictions will also continue to keep diseases of local importance under surveillance as appropriate.

From 1 January 2001, in those jurisdictions that have updated their legislation, influenza will be notifiable when 'laboratory confirmed', while anthrax will again be notifiable nationally. Also new to the national list are cryptosporidiosis, Australian bat lyssavirus, lyssavirus (other) and invasive pneumococcal disease. 'Haemorrhagic fevers (quarantinable)' covers infections with Lassa, Ebola, Marburg and Crimea-Congo haemorrhagic fever viruses. Arbovirus infections formerly covered by 'arbovirus infection not otherwise specified' will be reported as Japanese encephalitis virus, Kunjin virus, and Murray Valley encephalitis (MVE) virus infections. The southerly spread of MVE to within 315 km of Perth is reported in this issue.[12] For others, such as hydatid infection, chancroid, lymphogranuloma venereum and yersiniosis, national notification will cease in 2001.

From the New Year, all jurisdictions will report against an expanded core dataset that will make the NNDSS more specific by the inclusion of, for example, information on organism typing. In addition, enhanced surveillance of certain high priority diseases is being negotiated. In collaboration with the Public Health Laboratory Network, members of the CDNANZ and some of its disease-specific working groups are revising case definitions and expanding data sets for these diseases which include tuberculosis, hepatitis C, measles, invasive meningococcal disease and invasive pneumococcal disease. As a result these diseases will be reported nationally in greater detail.

The need to maintain surveillance is emphasised in a recent review on the emergence of Japanese encephalitis in the Australasian region,[13] in the article on introduced dengue cases reported in this issue,[14] and by the recent rise in malaria cases diagnosed in new arrivals in Western Australia (*Special report*, p 394 this issue). These events again remind us of the need for continual vigilance in the area of communicable diseases.

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Editorial: Wither *CDI*?

This publication has now completed its twenty-fourth year. In January 1977 it started as the 'National Microbiological Reporting Service' bulletin published fortnightly. Reported in this initially were viral (plus certain rickettsial and mycoplasma) diseases. The longer established 'ADH (Australian Department of Health) notifiable diseases in the States and Territories of Australia' bulletin had been distributed weekly and separately from August 1975 by the (then) Environmental Health Branch of the Department of Health. As the mailing lists had many addresses in common the two bulletins were amalgamated in August of 1977, and a month later the name was changed to '*Australia: Communicable Diseases Intelligence*' (*CDI*).[1] In 1980 the Australian Commonwealth Arms was first used on the front cover '*to signify that CDI is an Australian Government publication distributed overseas (about 10% of the circulation of 750 goes overseas)*'[2] and 'Australia' was dropped from the title.

At the beginning of a new millennium, it seems appropriate to review the role of *CDI* in national surveillance and examine the information needs of its current readership and the public health sector more broadly. Over the years *CDI* has evolved as a vehicle to disseminate information on the national picture of communicable diseases in Australia. Initially it was a fortnightly collation of reports from contributing laboratories, plus brief outbreak reports and the weekly updates on notifiable diseases. The latter were collated 4-weekly from January 1979 until March 2000 when monthly reporting was started. The earlier emphasis of *CDI* as a vehicle for communicating laboratory data, timely outbreak reports and late-breaking news changed. Over time and with successive editors, contributed articles and reports increased in number and length, and *CDI* had become an international publication reporting on any aspect of communicable disease in Australia. In late 1995 reporting of *CDI* on the Population Health Division Website started and an Advisory Board met for the first time. Peer review of submitted articles was introduced in 1996 as a prelude to citation of *CDI* by Medlars and Medline, which started early in 1997. In October 1997 *CDI* switched from a 2-weekly to 4-weekly publication and in April 2000, to a monthly one.

The 24-year history of *CDI* has also spanned some major changes in communication technology, to a stage where this publication, in its present form, is no longer fulfilling its original function. In the 1970s surveillance data were communicated by telephone and mail and data for at least one issue were not received due to a postal strike.[3] Today, the data are transmitted electronically by e-mail and discussed at a fortnightly teleconference by members of the CDNANZ. Production of *CDI* takes time; the printed form of *CDI* has thus ceased to be the vehicle for timely distribution of the latest communicable disease information to those who need to know quickly.

From early 2001 the national surveillance data will be continually updated on the *CDI* Website,[4] and the first issue of *CDI* Volume 25 (January 2001), which will report the December 2000 surveillance data, will be the last in its current form. Thereafter *CDI* will be published quarterly with an emphasis on periodic reviews of, and reports on, the surveillance data, plus reviews and original articles on any aspect of communicable diseases of relevance to Australia.

The editorial team is keen for *CDI* (both the hard copy and the Website) to become more relevant to the needs of its readers. For example should *CDI*'s charter be broadened by a more liberal interpretation of 'any aspect of communicable disease'? The team would be pleased to hear from readers of ways that the new *CDI* could continue to be improved.

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The outbreak that had to happen: *Bordetella pertussis* in North-West Western Australia in 1999

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Abstract

In late 1999, an outbreak of *Bordetella pertussis* occurred in a small town in North-West Western Australia. We undertook an investigation to describe the outbreak and to identify strategies to minimise the impact of future pertussis outbreaks in Australia. In November, people with respiratory symptoms were reviewed in an emergency pertussis clinic, which provided antibiotic treatment or prophylaxis. We conducted a school survey to enhance case ascertainment and followed up those attending the clinic by telephone. Fifty-nine cases of confirmed or probable *B. pertussis* infection were identified from 124 households (482 persons). Ages ranged from 5 months to 67 years, with children aged 9 to 11 years comprising 24 cases (41%). Early missed diagnoses and a school camp in September attended by 2 symptomatic children appeared to facilitate spread of infection, with the outbreak peak occurring in November. From immunisation records, childhood vaccine coverage in this sample was estimated at 96 per cent. All 21 cases of pertussis among the group under 10 years of age were at least partially vaccinated. There was only one laboratory confirmed case in the high-risk, under one-year of age category. Even in highly immunised populations periodic pertussis outbreaks are inevitable reflecting a vaccine efficacy of about 80 per cent and waning immunity with increasing age. Prevention of pertussis outbreaks depends not only on high vaccination coverage among young children but also early diagnosis and management of cases and their contacts. Clinicians should consider pertussis in the differential diagnosis of persistent cough illness in people of all ages – even those previously immunised. *Commun Dis Intell* 2000;24:375-379.

Keywords: pertussis, outbreak, chronic cough, immunisation

Introduction

Whooping cough, caused by the bacterium *Bordetella pertussis*, is a highly contagious respiratory disease that can cause serious illness or death among infants and young children. Transmission is by airborne contact with respiratory secretions and the incubation period is 6 to 20 days. Communicability is high during the early catarrhal period, but becomes negligible about 3 weeks after the onset of coughing paroxysms.[1] In Australia, childhood immunisation, consisting of a primary course at 2, 4 and 6 months followed by boosters at 18 months and 4 to 5 years, is recommended.[2] The school entry dose was introduced in August 1994 and acellular pertussis vaccines replaced whole cell ones for booster doses in Western Australia (WA) in February 1997 (Source: Perth Immunisation Clinic).

In April 1999, within a remote community in the Gascoyne region of WA, an elderly male with a history of chronic cough was diagnosed as having *B. pertussis* by IgA serology. On 24 November, after a woman and her daughter presented with a persistent cough, a local general practitioner concerned about pertussis contacted the Gascoyne Public Health Unit (PHU) and organised nasopharyngeal aspirates. On 26 November, *B. pertussis* was confirmed in both cases by polymerase chain reaction (PCR). It was subsequently discovered that another 30 individuals, including school children, teachers and household contacts, had been affected during preceding months.

To achieve disease control and reduce the risk of infection to the very young, we established a short-term pertussis clinic for the diagnosis and management of cases and their household contacts.[3,4] A public forum was held and an information bulletin distributed to raise community awareness of the illness. Concurrently, non-immunised or incompletely immunised children under 8 years of age were vaccinated by the community health nurse, and accelerated childhood immunisation of infants with the first dose at 4 to 6 weeks and the second and third at 4-week intervals was undertaken.[3] This immunisation program was continued until Christmas 1999.

This paper describes the epidemiology of the outbreak and outlines strategies for minimising the impact of future pertussis epidemics.

Methods

Setting

The setting was a small coastal town, population about 2600 (Source: Australian Bureau of Statistics 1998). It has one school, which provides primary and high school education.

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Epidemiological investigation

Interviews with hospital staff, general practitioners, the community health nurse and the school principal were conducted to gather information on the circumstances and extent of the outbreak. Standardised pertussis data collection forms[3] detailing demographic and illness characteristics were completed for those who presented to the short-term clinic with presumptive pertussis. Where possible, to clarify illness progress and confirm the diagnosis on the basis of the case definition, those attending the clinic were followed up 2 weeks later by telephone interview.

In early December, because clinical pertussis had been identified among children, we undertook a school survey to review progress of the outbreak. To ascertain cases, describe the illness characteristics, determine the pattern of spread and obtain vaccination data for the under 10 years age group, a written questionnaire was developed and distributed to school children. As a low response rate was expected, telephone interviews of every fifth family on the school's roll were also conducted to review the presence of disease among a systematic sample of the school community. Data were entered and analysed in Epi Info 6.04c and descriptive statistics are presented.

The immunisation status for the sample of children under 10 years of age was verified by review of the community health nurse's vaccination records and the vaccine coverage for those aged between 1 and 9 years was estimated from these records. For this calculation, vaccine coverage was defined as documented completion of the primary course of pertussis immunisation.

Case definition for *Bordetella pertussis* infection[3]

Probable

A cough illness lasting 14 days or more with one of the following: coughing paroxysms, inspiratory whoop or post-tussive vomiting without other apparent cause, or a cough illness lasting 14 days or more in a patient with *B. pertussis*-specific IgA detected in serum.

Laboratory-confirmed

Isolation of *B. pertussis* from a clinical specimen, or a positive PCR assay for *B. pertussis* undertaken in a laboratory with established expertise in this area.

Epidemiologically confirmed

A cough illness lasting 14 days or more in a patient who was epidemiologically linked to a laboratory confirmed case. Any person in close contact with a laboratory confirmed case during the infectious period and with cough onset between 30 days before and 30 days after the cough onset in the confirmed case was considered epidemiologically linked.

Microbiological sampling

A nasopharyngeal aspirate was collected from consenting patients who presented in the acute phase; this was used for *B. pertussis*-specific and Respiratory Syncytial Virus (RSV)-specific PCR testing, and viral culture. A single blood sample for *B. pertussis* IgA serology was taken from those with a prolonged-cough illness. Specimens were forwarded to the Western Australian Centre for Pathology and Medical Research (PathCentre) for processing.

Results

Epidemiological investigation

Data were collected from a total of 124 households or 482 people (Table 1). Seventy-one households, comprising 285 people, returned the survey (response rate: 28.5%). Of the households systematically sampled from the school's roll, 14 (28%) had already returned their questionnaires and a further 28 were successfully contacted on 17 December by telephone.

Table 1. Sources of information regarding households sampled during the investigation

Method	No. of households	People represented
Pertussis clinic:		
seen at clinic only	7	10
followed up by telephone interview	18	73
School survey questionnaire returned	71	285
School-roll telephone interview	28	114
Total	124	482

Of the 482 persons, 259 (53.7%) were female, ages ranged from 5 months to 67 years (median: 16 y, mode: 9 y) and the number of household members varied from 2 to 7 (mean/mode: 4).

Case characteristics

From this sample, 59 people had a cough illness that fulfilled the case definition for pertussis as follows. Five (9%) were laboratory-confirmed by PCR, 11 (19%) were epidemiologically confirmed and 43 (73%) were probable cases. Among the probable cases, 4 had positive *B. pertussis* IgA serology and a further 16 were close contacts of these.

Twenty-six (44%) were identified in the pertussis clinic at presentation or on telephone interview follow up. Forty-one (70%) cases were female and ages ranged from 5 months to 67 years (median and mode: 11y). There were 24 cases (41%) among children aged 9 to 11 years and 14 cases (24%) among adults, both teachers and parents. Incidence estimates of whooping cough by school year and age group are shown in Tables 2 and 3, respectively. There was only one case in the under 1-year-old category (Figure 1).

All cases had a history of cough illness lasting at least 2 weeks; 57 (97%) complained of coughing paroxysms or inspiratory whoop and 15 (25%) of post-tussive vomiting. By 17 December, complete recovery had been documented in 21 (36%) cases, persistent cough remained in 32 (54%) and status was unknown in 6 (10%). Forty-eight (81%) had seen a medical practitioner and 32 (54%) received a course of erythromycin or roxithromycin. No cases required hospitalisation.

Extent of the outbreak

The epidemic curve for the pertussis outbreak is shown in Figure 2. The first case of pertussis was identified in an

Table 2. Pertussis outbreak in a remote Western Australian town, 1999. Incidence rate per 100 persons by school grade

School class	No. of pupils	Incidence
Preschool	37	0.0
Kindergarten	34	8.8
Year 1	32	6.3
Year 2	36	8.3
Year 3	36	8.3
Year 4	40	20.0
Year 5	29	17.2
Year 6	37	29.7
Year 7	27	11.1
Year 8	26	11.5
Year 9	30	0.0
Year 10	27	4.2

Table 3. Pertussis outbreak in a remote Western Australian town, 1999. Incidence estimates per 100 persons by age group

Age group (years)*	No. of people	Incidence†
0-4	170	2.4
5-9	194	8.8
10-14	162	13.0
15-19	96	2.1
20-24	124	0.0
25-29	162	0.0
30-34	233	0.0
35-39	224	2.7
40-44	187	2.1
45-49	191	1.0
50-54	172	1.2

* Those aged 55 years and over are not included, as our school survey methods did not adequately sample this age stratum which had only one documented case. The 20 to 29 year age group may have also been underestimated.

† 1996 ABS census figures used as the denominators.

elderly male with positive *B. pertussis* IgA serology in April. The epidemic reached a peak during November and the Gascoyne PHU was notified on 24 November, late in the course of the outbreak. By the end of December, the outbreak had subsided.

A Year Six camp (8-17 September) appeared to facilitate spread. Two students with persistent cough had attended this camp. During the next 2 months, a further nine 11-year-old children developed clinical pertussis. Teachers and pupils in other grades were also affected. In 15 households (12%), more than one person fulfilled the case definition for pertussis. In 7 instances, a Year Six camp participant introduced the illness into their household with siblings or parents subsequently falling ill. This resulted in 12 secondary cases.

Immunisation data

Within our total sample, there were 21 cases and 126 non-cases under 10 years of age with a vaccination status

Figure 1. Whooping cough outbreak in a remote Western Australian town, 1999. Cases by age

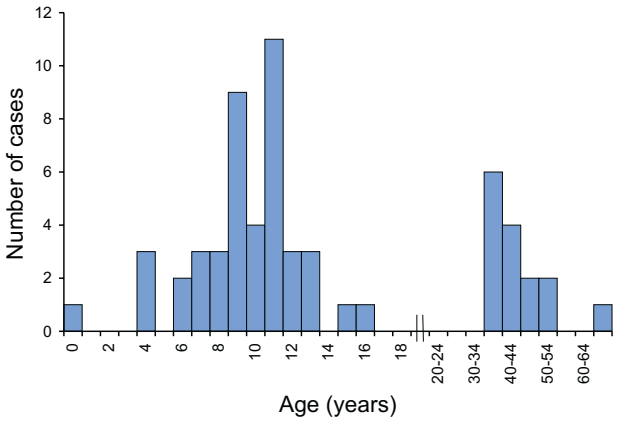
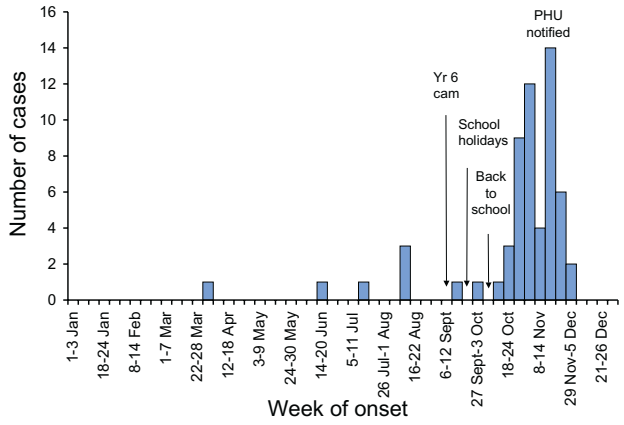


Figure 2. Whooping cough outbreak in a remote Western Australian town, 1 January to 31 December 1999, by week of onset



as shown in Table 4. There were 17 cases in the 5 to 9 year age group; 15 were fully vaccinated and 2 had received (at least) 4 triple antigen (DTP) doses. Ten of the 17 (59%) were confirmed cases: one was PCR positive and 9 were epidemiologically linked. All three 4-year-old children affected received the fifth dose of DTP after their illness had commenced. The infant aged 5.5 months had received the 2- and 4-month vaccinations but the second was given on 4 November in the midst of the outbreak: as its 2 school-aged siblings had a persistent cough in the fortnight prior to 4 November, it is probable that this baby had already been infected.

Among children aged 1-9 years in this sample, vaccine coverage was 96 per cent (139/145; Table 4). Adolescents aged 11 years and above in our cohort did not receive a school entry booster and the peak number of cases occurred among the 11-year-old children (Figure 1). Those aged 7 years and above had received whole cell vaccines. Those aged between 18 months and 6 years received one booster dose of acellular vaccine.

Table 4. Pertussis vaccination status of cases and non-cases under-10 years of age

Age (years)	Total no sampled aged <10y (n = 147)	Cases (n = 21)			Non-cases (n = 126)			
		No.	Age-appropriately vaccinated	Other*	No.	Age-appropriately vaccinated	Other*	Unknown†
<1	2	1	1	-	1	1	-	-
1	5	0	-	-	5	5	-	-
2	5	0	-	-	5	5	-	-
3	14	0	-	-	14	13	-	1
4	12	3	3	-	9	8	-	1
5	16	0	-	-	16	15	1	-
6	23	2	2	-	21	20	1	-
7	20	3	3	-	17	15	1	1
8	18	3	3	-	15	10	4	1
9	32	9	7	2	23	21	-	2

* All cases were verified to have received four doses of DTP vaccine.

† Documented evidence of immunisation was unavailable.

Microbiological sampling

Of the 59 cases, 9 had a nasopharyngeal aspirate collected for laboratory confirmation of *B. pertussis* by PCR; five (an infant, a teacher, 2 school children and a parent) were positive. The remaining 4, all sampled over one month after illness onset, were *B. pertussis*-negative on PCR testing. No other pathogens were identified. Two other cases were positive for *B. pertussis* on IgA serology. One was the first reported case, the other a school student. A further 2, both teachers with clinical symptoms, had low-positive results.

Discussion

Prior to this outbreak, there had been no notified cases of whooping cough in the community since at least 1988,[5] despite an epidemic in the rest of WA in 1997-8 (1164 notifications in 1997, 380 in 1998. Source: Health Department of WA). A cyclone in March 1999 resulted in increased mobilisation of people into and out of the town and may have led to the introduction of *B. pertussis*. Low numbers of infections occurred between April and September and we surmise that not all were detected. The number of cases rose in mid-October with a peak in mid- to late November (Figure 2).

Illness spread appeared to have been facilitated in September by the Year Six school camp attended by 2 ill children. It is possible that several children were incubating during the school holidays and then returned to school in the highly infectious catarrhal phase. Moreover, there was evidence of household transmission with secondary cases among households with camp participants. The outbreak subsided in December.

Immunisation appears to reduce disease frequency and transmission.[6] The high level of childhood immunisation coverage in this community, estimated from this sample to be 96 per cent, is likely to have protected the very young who are at highest risk for severe complications from infection. There was just one confirmed case in a partially immunised baby who fully recovered.

However, even in highly immunised communities, cyclic pertussis epidemics do occur because vaccine efficacy has been estimated at around 80 per cent in children who have received at least 3 doses and immunity is know to wane over time and may be negligible after 12 years.[1,7,8] Disease among immunised children has increasingly been described[9,10] and we report 20 cases of whooping cough (10 confirmed and 10 probable) among children under 10 years of age who had received 4 (5/20) or 5 (15/20) pertussis vaccinations. Nine (45%) of the 20 cases had been vaccinated at least 4 years previously.

Those who have been previously immunised tend to have less severe illness, but may be more difficult to diagnose.[11] In this outbreak, pertussis was only considered after early cases with chronic cough had failed to respond to various therapies for other presumed conditions. This limited the benefit of public health action as widespread community exposure had already occurred.

By our methods, misclassification by outcome is possible. Some true positive cases would have been excluded because mild illness would not have met the case definition. Indeed 8 people, 6 of whom had received antibiotic treatment, were excluded on this basis. On the other hand, the emergency pertussis clinic and public notices about the outbreak heightened awareness of pertussis. This raised the possibility that false positive clinical diagnoses were made and antibiotic treatment over-used. Fortunately, treatment resulted in only one known case of diarrhoea.

With respect to case ascertainment, telephone interviews using systematic sampling from the school roll identified just one additional case, suggesting that the reason for non-response was absence of illness among these families. However, under-ascertainment in the 20 to 30 and over 65-year age groups may have occurred because these groups were not adequately sampled by our school survey methods.

Recommendations

Childhood immunisation is the most important means of pertussis prevention. The public needs reminding regularly about the need for vaccination to protect the very young. As pertussis outbreaks still occur, general practitioners should include pertussis in the differential diagnosis of prolonged cough illnesses even in previously vaccinated individuals.

Prompt diagnosis, laboratory confirmation and notification ensures early public health intervention to minimise disease spread. Regular articles in general practice journals about the '100-day cough' are required to keep pertussis on the agenda. Additionally, public health departments should use pertussis notification data to identify towns and regions with prolonged minimal activity. Education can then be provided to medical practitioners in these areas, which may result in early detection and action when *B. pertussis* infections arise. In the outbreak setting, information dissemination, treatment of cases, prophylaxis of contacts, follow up of those attending clinics, and accelerated immunisation in the young are the public health interventions used for the prevention and control of the disease.

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Laboratory-supported influenza surveillance in Victorian sentinel general practices

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Abstract

Laboratory-supported influenza surveillance is important as part of pandemic preparedness, for identifying and isolating candidate vaccine strains, for supporting trials of anti-influenza drugs and for refining the influenza surveillance case definition in practice. This study describes the implementation of laboratory-supported influenza surveillance in Victorian sentinel general practices and provides an estimate of the proportion of patients with an influenza-like illness proven to have influenza. During 1998 and 1999, 25 sentinel general practices contributed clinical surveillance data and 16 metropolitan practices participated in laboratory surveillance. Serological, virus-antigen detection, virus culture and multiplex polymerase chain reaction procedures were used to establish the diagnosis of influenza. Two laboratories at major teaching hospitals in Melbourne provided additional data on influenza virus identification. General practice sentinel surveillance and laboratory identification of influenza provided similar data on the pattern of influenza in the community between May and September. The clinical suspicion of influenza was confirmed in 49 to 54 per cent of cases seen in general practice. *Commun Dis Intell* 2000:24;379-383.

Keywords: influenza, diagnosis, surveillance, community medicine

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Introduction

Laboratory confirmation of the diagnosis of influenza in sentinel general practices was introduced into Victoria in 1998 as a joint initiative of the Victorian Infectious Diseases Reference Laboratory (VIDRL)* and the Department of Human Services (DHS). This complemented the clinical influenza surveillance that had been coordinated by DHS through a sentinel general practitioner (GP) network in previous years.

Surveillance with laboratory support has been recognised as an important part of the pandemic response in Australia's recently published pandemic plan.[1] In late 1997, during the planning of this initiative, the significance of a small number of human cases of influenza virus type A (H5N1) in Hong Kong was unclear and the possibility of the emergence of a new pandemic strain remained very real.[2]

Moreover, in recent years, laboratory surveillance has also been critical in supporting general practice trials of anti-influenza drugs, specifically neuraminidase inhibitors, used in the treatment and prevention of influenza.[3] However, not all influenza-like illness (ILI) seen in general practice will be confirmed as influenza. Published estimates of the proportion of patients with ILI confirmed as having influenza by laboratory testing vary from as low as 6 to14 per cent at the introduction of surveillance networks[4] up to 70 per cent as part of a trial of influenza antiviral medication.[3] The present study describes the implementation of laboratory supported influenza surveillance in Victorian sentinel practice sites in 1998-9 and provides an estimate of the proportion of patients with an ILI proven to have influenza.

Methods

Recruitment of GPs to the sentinel surveillance network was restricted to those who were anticipated to have an interest in surveillance. Random selection of general practice sites was not attempted. Medical officers of health in local government authorities and general practitioners who had participated in influenza surveillance in previous years, or those known to have an interest in immunisation, were invited to become part of the sentinel surveillance network. At the end of the recruitment process, in an attempt to include sentinel practices throughout Victoria, practices in regions that were not represented in the network were contacted. In all 17 practices participated in 1998 and 26 in 1999. GPs in 14 practices participated in both years.

General practice surveillance and specimen collection

Two types of surveillance were undertaken in sentinel general practices. The first required practices only to notify the proportion of patients with an ILI as a proportion of total patients seen each week. As in previous years, ILI was defined using the Australian Sentinel Practice Research Network (ASPREN) criteria for the diagnosis of influenza, criteria also used for the International Classification of Health Care Problems in Primary Care (ICHPP-2).[5] These are:

an influenza epidemic and 4 of the criteria below, or 6 of the criteria below in the absence of an epidemic;

1. onset within 12 hours
2. cough
3. rigours or chills
4. fever
5. prostration or weakness
6. myalgia, widespread aches and pains
7. no significant respiratory signs other than redness of the throat and nasal mucous membranes
8. influenza in close contacts (modified for this study to 'history of influenza-like illness' to allow inclusion of illness in non laboratory-confirmed close contacts).

The second type of surveillance used laboratory support for the confirmation of the diagnosis of influenza; this was restricted to 16 metropolitan practices for logistical reasons. Eight practices participated in both years; 2 practices participated only in 1998 and 6 new practices were recruited in 1999. At each site the GP determined which patients were to be asked to provide a clinical specimen, gave these patients an information sheet and discussed the project with them. Following consultation with the Victorian DHS, since this study was considered an extension of normal patient care, formal ethics approval was not sought. Patients were, however, asked to sign a consent form indicating they understood this process and consenting to the collection of specimens, including a convalescent serum sample.

Participating patients provided details of which ASPREN criteria they met, their influenza vaccination history, and any history of recent travel or contact with travellers. From those fulfilling the ASPREN criteria, a throat swab and nose swabs from each nostril were pooled in a single vial of viral transport medium. Acute phase blood was collected. In 1998 attempts to collect a convalescent blood sample were made only for patients for whom a laboratory diagnosis could not be made by detection of viral antigen using an immunofluorescence assay, viral isolation or serology (detection of influenza virus specific IgM, or IgG titre³ 160, see below). In 1999 all patients were asked to provide a convalescent blood sample. Where possible, following collection, specimens were stored at 4°C before transport to VIDRL on the same day.

Laboratory testing

In both 1998 and 1999 epithelial cells drawn from nose and throat swabs were examined for the presence of influenza antigen using an indirect immunofluorescence method. Viral isolation was also attempted using standard methods.[6] Acute and convalescent sera, where available, were screened in parallel using a complement fixation test.[7] A 4-fold rise in titre between acute and convalescent samples or a titre 160 in the acute phase serum was taken to indicate recent infection with influenza virus. An immuno-fluorescence based assay to detect antibodies to influenza A and influenza B virus was used when the complement fixation test gave indeterminate results (2-4 fold rise in antibody titre or acute phase titre = 80). In 1999 a multiplex polymerase chain reaction (PCR) assay that detected and differentiated influenza virus type A (H1N1), influenza virus type A (H3N2) and influenza virus type B was used as an additional test of the nose and throat swabs.[8,9] All tests were performed on all available specimens in both years. A patient was considered to have influenza virus infection if one or more of the laboratory tests were positive.

Results and data analysis

Patient results were sent to the referring GPs in the usual way. A progress report of the influenza season was sent to participating general practices and laboratories each

fortnight. Data were analysed in Epi Info (version 6).[10] Fisher's exact test and the Chi-squared distribution were used to test for association, assuming that patients who were part of surveillance were representative of all patients from the sentinel practices.

Surveillance at other laboratories

Virology laboratory staff at the Royal Children's Hospital and the Monash Medical Centre provided information on the number of identifications of influenza virus types A and B made each week. Data on age, sex and vaccination status were included when available. In addition VIDRL provided data on patients identified with influenza A or B who were not part of the surveillance project.

Results

In both 1998 and 1999, an influenza virus type A (H3N2) Sydney/5/97-like strain was the predominant circulating strain. This strain was a component of the influenza vaccine distributed in Australia in both years.

General practice clinical surveillance

Between April and September 1998, 463 patients (73 per cent from metropolitan practices) satisfying the ASPREN criteria for the clinical diagnosis of influenza were notified from 10 metropolitan and 7 regional practices. Over a similar period in 1999, 351 patients were notified, 30 per cent from 12 metropolitan practices and the remainder from 14 regional practices. There was no significant difference between the sex distribution of notified cases in 1998 (46% male) and 1999 (42% male). However, there was a significant difference in age (Figure 1).

Vaccination data were available for 807 of the 814 patients notified over both years. Only 8 per cent of all notified patients were vaccinated. Those aged 65 years and over were more likely to be vaccinated than younger people (46.5 per cent compared with 5.1 per cent respectively; $p<0.0001$).

General practice laboratory surveillance

A diagnosis of influenza was considered laboratory-confirmed if at least one of the laboratory tests for influenza was positive. The diagnosis was excluded if all tests, including convalescent serology, were negative. When a patient provided an incomplete set of specimen types and the results of all available tests were negative, the diagnosis was classified as not determined.

In the 2 years of this study, 152 patients provided clinical specimens for laboratory surveillance. The median age of patients was 40 years in 1998 (inter-quartile range 25 to 53 years) and 38 years (28 to 45 years) in 1999. There were fewer males (47%) than females. In 1998 the clinical diagnosis of influenza was confirmed by laboratory testing in 45/110 (41%) of all patients tested and in 45/92 (49%) of patients for whom a definite laboratory diagnosis or exclusion was made. In 1999 influenza was confirmed in 20/42 (48%) of all patients who provided clinical specimens and 20/37 (54%) for whom a definite diagnosis could be made (Table).

The proportion of patients with ILI confirmed by laboratory testing to have influenza virus infection was not significantly different in the 2 years during which the same type A (H3N2) Sydney/5/97-like influenza strain was circulating, so further analysis combined data for both years. Of all patients notified with an ILI, 46/85 (53%) were confirmed as having

Figure 1. Patients with influenza-like illness notified from Victorian sentinel general practices, 1998 to 1999, by age-group

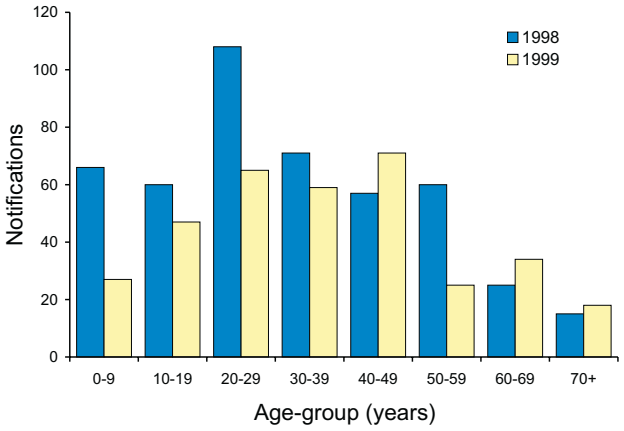


Table. Laboratory diagnosis of patients with influenza-like illness from sentinel general practice surveillance, Victoria, 1998 to 1999

	1998[1]		1999[2]	
Laboratory diagnosis	Patients	Per cent	Patients	Per cent
Influenza virus type A[3]	44	40	18	43
Influenza virus type B	1	1	2	5
Other viruses[4]	3	3	0	0
No virus detected[5]	44	40	17	40
Not determined[6]	18	16	5	12
Total	110	100	42	100

1 Tests used, 1998: serology, influenza virus antigen detection by immunofluorescence and virus isolation

2 Tests used, 1999: serology, influenza virus antigen detection by immunofluorescence, virus isolation and PCR

3 Coxsackie type B4 also cultured in one patient who seroconverted to influenza A

4 Rhinovirus, adenovirus, respiratory syncytial virus

5 Virus not detected by any method with complete set of specimens available

6 Virus not detected by any method with incomplete set of specimens available

influenza during the peak weeks of the season, compared with 19/44 (43%) in the lower incidence weeks at the start and end of the seasons (p = 0.50). The proportion of patients for whom the clinical diagnosis of influenza was confirmed was not significantly different between the under 20 years, 20 to 59 years and 60 years-and-over age groups (71, 49 and 53 per cent respectively; p = 0.45). Nor was there any significant difference in the confirmation rate by practice, although this analysis is based on small numbers.

Vaccination and travel history

Of all patients indicating whether they had received vaccination against influenza prior to April in either year, influenza infection was either diagnosed or excluded in 107 of them. Of these, 23 (21%) were vaccinated. Influenza infection was confirmed in 9/23 (39%) vaccinated patients and 48/84 (57%) unvaccinated patients (p = 0.12).

Only 12 patients gave a history of recent travel in 1998 or 1999; there was no relationship between travel and a positive diagnosis for influenza (p = 0.27). Fifty patients gave a history of close contact with at least one person recently travelling outside Australia; there was no significant relationship between such contact and the confirmation of influenza (p = 0.16).

Combined surveillance data from all testing laboratories

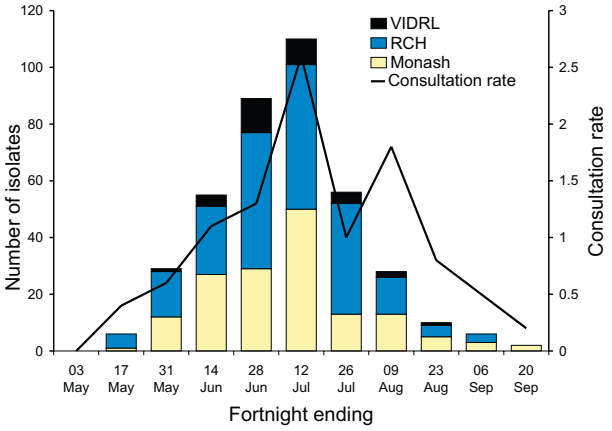
Over the 2 years there were 730 notifications from 3 laboratories. Data contributed by VIDRL consisted of specimens referred from hospital in-patients who were not part of sentinel surveillance. Data from the Royal Children's Hospital and the Monash Medical Centre consisted of influenza virus identification in hospital in-patients. Except for 3 type B identifications in 1998 and 17 in 1999, all were influenza virus type A. Because 2 of the contributing laboratories service a predominantly paediatric population, 70 per cent of laboratory notifications were from patients aged less than 10 years. Despite the differences in the age of hospital in-patients and general practice sentinel surveillance patients, the seasonal pattern of influenza and ILI was similar in both years (Figure 2a and Figure 2b).

Discussion

Neither the circulating strains of seasonal influenza virus nor the severity of epidemics is readily predictable. Since 1981, influenza virus type A has been the predominant circulating strain in one season, with a mixture of influenza A and B virus types in alternate seasons.[11,12] Based on this pattern, an influenza season involving both influenza A and B had been expected for, but did not eventuate in, 1999 in Victoria or nationally.[13]

Influenza surveillance with laboratory confirmation of an influenza diagnosis allows the differentiation of ILI from influenza. In 2 years of surveillance in Victoria the proportion of patients confirmed to have influenza varied from 49 to 54 per cent, which is higher than the proportion reported from most other surveillance studies but lower than that reported from therapeutic trials[3,14] when a more specific clinical diagnosis may be required. A study from France during the 1995-6 influenza season examined the positive predictive value of 12 different surveillance case definitions for influenza and found predictive values of 12 to 40 per cent.[15] These values were higher than those obtained in the first 2 years of laboratory surveillance in England and Wales when

Figure 2a. Hospital admission based influenza surveillance compared with influenza-like illness surveillance in sentinel general practices, 1998, by fortnight*



*Monash Monash Medical Centre, Clayton, Victoria

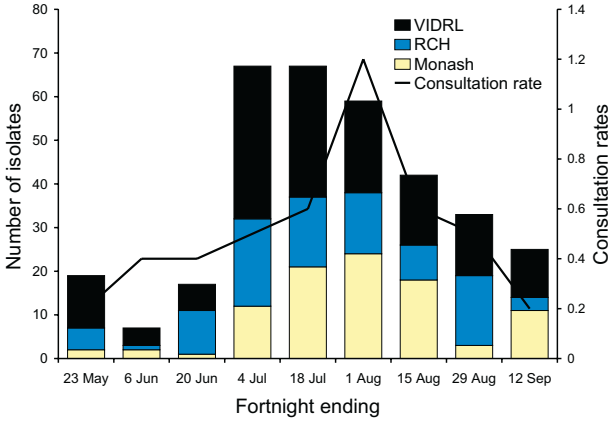
RCH Royal Children's Hospital, Parkville, Victoria

VIDRL Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria

Consultation rate the number of patients with influenza-like illness as a proportion of all patients in sentinel general practice sites

Influenza notifications from all laboratories, including VIDRL, are from hospital in-patients. Cases of influenza confirmed from general practice ILI surveillance are not included in the surveillance from VIDRL.

Figure 2b. Hospital admission based influenza surveillance compared with influenza-like illness surveillance in sentinel general practices, 1999, by fortnight*



*Monash Monash Medical Centre, Clayton, Victoria

RCH Royal Children's Hospital, Parkville, Victoria

VIDRL Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria

Consultation rate the number of patients with influenza-like illness as a proportion of all patients in sentinel general practice sites

Influenza notifications from all laboratories, including VIDRL, are from hospital in-patients. Cases of influenza confirmed from general practice ILI surveillance are not included in the surveillance from VIDRL.

influenza was confirmed using viral isolation and direct IF in only 6 per cent of patients in 1993-4 and 14 per cent in 1994-5.[4] More recent virological surveillance of community influenza in 6 Scottish general practices using PCR, culture and serology confirmed the diagnosis of influenza in 67 per cent of notified cases.[16] The positive predictive value of any test rises with the prevalence. In the 2 years of this study we were able to demonstrate a lower positive predictive value for influenza diagnosis in the early and later weeks of the season (43%) compared with the higher prevalence weeks (53%); this difference was not significant.

There are many different definitions of influenza for surveillance purposes[15] and the utility of these definitions is related both to the clinical symptoms included in the definition and the sensitivity of the tests against which the definitions are evaluated. The ASPREN criteria have recently been evaluated as the ICHPP-2 in people aged 65 years and over.[5] The use of all the ASPREN/ICHPP-2 criteria proved more likely to predict an ILI than influenza, and the evaluation concluded that the 3 criteria of sudden onset, fever and cough could be used as a reliable surveillance definition of influenza. Using these criteria, influenza was confirmed in 30 per cent of patients in the ICHPP-2 criteria evaluation study, with serological testing as the only test for laboratory confirmation of influenza infection. In our patients, using a combination of laboratory tests, the symptoms identified in the ICHPP-2 evaluation study predicted influenza in 54 per cent of patients with an ILI. This is similar to the proportion predicted by all the ASPREN criteria but has the distinct advantage of being a much simpler surveillance definition. We are currently reviewing various influenza case definitions using surveillance data from Victoria and Western Australia.

Although travel may play an important part in the global spread of the influenza virus, our data do not show a significantly increased risk of infection in people who had travelled recently or who had been in contact with travellers. Both the destination and the timing of international travel will impact on the likelihood of any association between travel and infection.

Laboratory confirmation of influenza infection from patients in sentinel general practices has been established in Victoria and continues to be refined. Because a number of laboratory tests, including convalescent serology, were used in the present study, the confirmation of influenza infection amongst patients notified with ILI was relatively high (49-54%). Pattern differences in influenza surveillance from laboratories in teaching hospitals and ILI in general practice may relate to the proportion of patients with ILI in general practice proven to have influenza and the different age groups and disease severity in the two surveillance systems.

***Abbreviations**

ASPREN, Australian Sentinel Practice Research Network; DHS, Department of Human Services; GP, general practitioner; ICHPP-2, International Classification of Health Care Problems in Primary Care; ILI, influenza-like illness; PCR, polymerase chain reaction; VIDRL, Victorian Infectious Diseases Reference Laboratory.

Acknowledgments

The participation of GPs is critical for sentinel surveillance and we would like to thank all GPs who participated in this scheme. Mr Alan Hampson reviewed an earlier draft of this report.

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Australian recommendations for the Influenza Vaccine for 2001 season an update

As reported in *Commun Dis Intell* in October,[1] the Australian Influenza Vaccine Committee (AIVC) recommended that, for the influenza vaccine for 2001 in Australia, the Influenza B strain be changed to a B/Sichuan/379/99-like strain; whether the B/Victoria/504/00 influenza strain would be suitable as an endorsed 2001 vaccine strain awaited further testing.

At a recent meeting the AIVC has decided that both B/Johannesburg/5/99 and B/Victoria/504/2000 are suitable B/Sichuan/379/99-like strains for vaccine production. The decision to recommend an alternative strain was taken to give the manufacturers flexibility in maximising yield in the production of the vaccine.

The AIVC emphasised that the decision related solely to production yield considerations and that in terms of antigenicity and effectiveness, the two strains should be regarded as equivalent.

1. Australian Influenza Vaccine Committee. Australian recommendations for the Influenza Vaccine composition for the 2001 season. *Commun Dis Intell* 2000;24:306.

Special surveillance report

Cases of leptospirosis in hunters in the Top End – don't go barefoot[1]

Six cases of leptospirosis reported in the greater Darwin region in the past 6 weeks have highlighted hunting as a risk activity for contracting this potentially fatal disease.

Leptospirosis is a bacterial disease that can be mild or severe and can cause death. The disease presents with a variety of symptoms but most commonly with fever, sudden-onset headache, chills, aches and conjunctivitis. Kidney and liver failure can occur as well as bleeding (eg into the lungs) and mental confusion. Prompt and specific treatment for the disease as early as possible is essential.

Leptospirosis is an occupational hazard to those working on the land or with animals and a recreational hazard to bathers, campers and sportsmen in infected areas. Contact of the skin (especially abraded skin) with water, wet soil or vegetation that is contaminated with urine of infected animals (notably rats, pigs, cattle and dogs) constitutes the main mode of transmission of leptospirosis.

Of the recent 6 cases, 2 men (and a possible 3rd suspect male case) were duck hunters at the Harrison Dam/Fogg Dam area during the time consistent with them acquiring their disease. All were hunting in bare feet. The third male case lives on a rural block where there are many animals and he is most often barefoot.

The 3 female cases were turtle hunting around Oenpelli also in bare feet.

The Fogg Dam/Harrison Dam area is home to a very large and dense population of the 'dusky rat' (*Rattus colletti*), a native rat. Rats are considered the most significant reservoirs of leptospirosis worldwide.

Earlier in the year a male abattoir worker from Katherine was notified with leptospirosis making him the 7th reported case to date this year. All 7 cases have been hospitalised and 2 have required intensive care treatment.

From 1992 to 1999 there had been 9 cases of leptospirosis notified in the Northern Territory. Of note, all had been males aged 26 to 60 years, all were non- Aboriginal (the ethnicity of one 1993 case was unknown). Six cases were from the Katherine area with the remaining 3 from the Darwin area. Occupation or 'risk activity' for acquisition of the disease had not been systematically collected, however anecdotally, several Katherine cases were involved in activities around the Katherine Gorge – as tourists or guides.

The need for those dealing with animals and this environment to wear protective footwear and clothing is highlighted. Health practitioners need to be aware of the disease. Parks and Wildlife and Environmental Health are aware of the cases and health alerts will be distributed to hunters, tourists and workers in the area.

Contact Officer: Dr Vicki Krause, Director, Centre for Disease Control, Territory Health Service.
Telephone: +61 8 922 8044.

1. Media Release. Darwin: Territory Health Service; 15 December 2000.

Imported beef products: suspension extended

Because of concerns about the link between BSE-infected British beef and variant Creutzfeldt Jakob Disease (vCJD) in humans, importation into Australia of specified foods that contained British beef has been banned since 1996. As a precaution, because it is becoming clearer now that more countries in Europe may be affected by BSE in their cattle, the Federal Government has announced (Media Release, 5 January 2001) that it has decided to suspend temporarily the importation of foods containing beef or beef products from other countries in Europe.

There are small but significant amounts of these foods on the Australian market around 1000 tonnes per annum, which accounts for only 0.2 per cent of beef consumed annually in Australia. This food is imported almost entirely as canned or prepared food products.

The grocery industry, in consultation with State and Territory authorities, has been asked to introduce a voluntary withdrawal of all these products and consumers are being advised to dispose of any food they may have that contains beef from a specified European country of origin.

Australia's Chief Medical Officer, Professor Richard Smallwood, said Australia and New Zealand have no BSE in their cattle and Australian and New Zealand meat products are the safest in the world to eat.

The decision by Australia to suspend the import of foods containing beef from other countries in Europe from 8 January 2001 will be implemented in conjunction with the New Zealand Government, which has worked closely with Australia on this action.

More information on the voluntary food withdrawal can be obtained by ringing a special information phone line on 1800 200 701 or from the Commonwealth Department of Health and Aged Care's Internet site at:
http://www.health.gov.au/issues.htm or the Australian and New Zealand Food Authority Website:
http://www.anzfa.gov.au/documents/fs054.asp.

In case you missed it

The Cutting Edge

Contributed by Neil Branch, Media Advisor, Commonwealth Departmnet of Health and Aged Care. (edited)

My microbial friends, for your information: on 19 December The Cutting Edge on SBS ran a rather interesting program from the BBC titled 'Future Plagues.' It features some of the world's leading microbiologists and for the first 40 minutes was a bit of a history lesson about disease and humankind's oft futile battle against the bugs.

However the last 10-15 mins was devoted to some interesting theories such as heart attack and cancer being infectious diseases rather than medical conditions brought about by environs and personal habits and an interesting insight into New York City's handling of its Multi-Resistant Tuberculosis outbreak including enforced quarantine of citizens in former prisons for periods of several years.

If you are interested, a full transcript of the program, as well as CVs of the main speakers and other info can be found on the web at:
http://www.bbc.co.uk/science/horizon/future_plagues.shtml

Please note this piece went to air in Britain on 5 January 2000.

AIDS epidemic update

UNAIDS/WHO has just released the following publication -

AIDS epidemic update: December 2000

The electronic copy is located at:
http://www.unaids.org/epidemic_update/report_dec00/index_dec.html

APEC Emerging Infections Network (EINet)

The EINet listserv was created to foster discussion, networking, and collaboration in the area of emerging infectious diseases (EID's) among academics, scientists, and policy makers in the Asia-Pacific region. We strongly encourage you to share their perspectives and experiences, as your participation directly contributes to the richness of the 'electronic discussions' that occur.

Subscribers are encouraged to share their material with colleagues in the Asia Pacific Rim. To subscribe (or unsubscribe) free of charge, please contact
apecein@u.washington.edu

Further information about the APEC Emerging Infections Network is available at: http://www.apec.org/infectious.

A decision to end a periodic syphilis-screening program in the Kimberley region

Donna B Mak,[1,2] C D'Arcy J Holman[2]

Abstract

Syphilis rates in the Kimberley region of far-northern Western Australia are among the highest in the nation. In 1986, a formal program of periodic syphilis screening was established. Decreasing syphilis rates since the early 1990s prompted, in 1999, re-evaluation of the value of periodic screening. All confirmed cases of syphilis identified in the Kimberley between January 1996 and early December 1999 as a result of syphilis serology were classified by reason for the test and staged according to disease progression. During the study period, 196 cases of syphilis (117 male, 79 female) were diagnosed; 14 (7.1%) were primary, 32 (16.3%) secondary and 150 (76.5%) latent. The periodic screening program contributed only about 10 per cent of cases, whereas testing as a result of sexually transmitted disease symptoms, sexually transmitted disease contact, institutional screening and other screening contributed the remaining cases. In January 2000, the periodic syphilis-screening program was discontinued. The effect of this policy change will be closely monitored using indicators to ensure that, should the decision not to screen prove to have been misjudged, any increase in syphilis incidence is detected early and managed appropriately. *Commun Dis Intell* 2000;24:386-390.

Key words: syphilis, STD, screening, epidemiology, Aborigines, immigrants

Introduction

Syphilis rates in the Kimberley, a remote and sparsely populated region in far-northern Western Australia, are among the highest in the Nation.[1] A structured program of periodic syphilis screening, based on a regional population register of Aboriginal Kimberley residents, was established in 1986. It aimed to reduce the incidence of syphilis by detecting and treating prevalent cases.

The program offered annual syphilis serology (SS) testing to all Aboriginal Kimberley residents aged 15 to 40 years, and testing every second year to those aged below 40 years. In addition, syphilis testing was also recommended for all patients presenting with sexually transmitted disease (STD) symptoms, and named contacts of STD cases (at the initial consultation and 3 months later), and as part of routine antenatal screening (at booking and at 28 to 36 weeks gestation).

In 1996, following evaluation of the program and discussions with the Kimberley Aboriginal Services Council, the target group was modified to include all Kimberley residents aged 15 to 25 years. The basis for this decision has been discussed elsewhere.[2] In addition, rather than being centrally managed using a regional population register, the responsibility for initiating SS testing in first-time patients and for recalling patients for repeat testing was devolved to local health services. People outside the target group for whom a repeat SS had been recommended prior to the policy change were still offered the test, even if it became due after 1 January 1996. No changes were made regarding the other recommendations for SS as described above.

Setting

Aboriginal people comprise one half of the resident population of about 30,000 people scattered across the Kimberley, an area of more than 420,000 square kilometres. The age structure of the population is much younger than the State average. This reflects the demographics of Aboriginal Australia, as well as the predominance of young people who move to the Kimberley for work.

The landscape ranges from coastal sub-tropical areas to open savanna and semi-desert. Much of the terrain is rugged and accessible only by four-wheel drive vehicle or light aircraft. There are 6 major towns (with populations ranging from 2,000 to 10,000) and more than 200 discrete Aboriginal communities ranging in size from just a few families to over 500 people. Health care is provided predominantly by government and community-controlled organisations. Each of the major towns has a hospital and one or more primary-care services. Remote-area clinics staffed by nurses and Aboriginal health workers are present in fewer than 20 Aboriginal communities.

In recent years, increasing numbers of 'boat' people from Indonesia and the Middle East have entered the Kimberley illegally. On arrival, these people are taken immediately to either the regional prison or immigration detention centre, so they have very little unsupervised contact with Kimberley residents.

The unique cultural and demographic mix of the Kimberley, coupled with its geographic features and low population density, has considerable implications for communicable disease control.

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In this paper we report on SS testing activity in this region for the years 1986-1999 and discuss why periodic screening has been discontinued. Preliminary reports of parts of this work have appeared elsewhere.[3]

Methods

All pathology services are provided by one laboratory, which sends the pathology request form and results of SS tests done in the Kimberley to the Kimberley Public Health Unit (KPHU). With the agreement of all State government and Aboriginal community controlled health services, and private general practitioners in the region, the KPHU maintains a regional syphilis register of syphilis serology results and treatments. The first author (DM) interprets results in the light of the clinical history on the request form and the patient's previous SSs, as recorded on the register, and recommends when the next SS is due and whether any treatment is required. The annotated SS result is then sent to its health service of origin for filing in the SS recall system. Information about syphilis treatment is obtained from the patient's doctor on a case-by-case basis.

All cases of syphilis identified in the Kimberley as a result of syphilis serology taken between 1 January 1996 and 6 December 1999 inclusive, with diagnosis confirmed before 14 December 1999, were examined. Information obtained for each case from the KPHU syphilis database included the patient's age, sex, race and address, the district of their health service provider, their stage of syphilis, date of SS and date of completed treatment, and the clinical indication for SS testing.

Syphilis cases were staged according to the following criteria:

- Primary – serological evidence of syphilis infection or re-infection of less than 6 months' duration and/or clinical signs of primary syphilis, eg chancre.
- Secondary - serological evidence of syphilis infection or re-infection of 6 to 24 months' duration and/or clinical signs of secondary syphilis, eg condylomata lata, alopecia.
- Tertiary - serological evidence of syphilis infection or re-infection of greater than 24 months' duration and clinical signs of tertiary syphilis.
- Late latent - serological evidence of syphilis infection or re-infection of greater than 24 months' duration or no serological evidence that syphilis is less than 24 months' duration and no clinical signs of primary, secondary or tertiary syphilis.*

Due to the absence of information about clinical signs in many patients, an additional category of 'early syphilis, confirmed or probable' was defined. This included all cases of primary and secondary syphilis, and cases of late latent syphilis if the rapid plasma reagin test titre was greater than sixteen.

According to information on the pathology request form, clinical indications for SS testing were classified into the categories:

- STD symptoms – patient had symptoms of STD, eg urethral discharge, epididymo-orchitis, pelvic inflammatory disease, genital lesion.
- STD contact – named contact of a patient with STD.
- Antenatal – routine SS during pregnancy.
- Institutional screening – routine SS on admission to prison or detention centre.
- Periodic screening – SS done as part of the periodic screening program.
- Other screening – SS done in other clinical contexts, eg well person's check-up, diabetes/chronic disease review, asymptomatic patient requests STD screen.

In accordance with ethical requirements, access to the data was restricted to the person responsible for generating the data in the course of re-evaluating the screening program (the first author, DM). The confidentiality of study subjects was thus preserved at all times.

Results

SS testing activity increased after implementation of periodic screening in 1986 (Figure 1). After the program was modified in 1996, testing activity decreased initially but by 1999 had returned almost to pre-1996 levels.

Figure 1. Number of syphilis serology tests done in the Kimberley, by year*

* 1991-94 data are missing because these data were collected manually and are no longer accessible.

As part of a regional evaluation of the quality of STD management, syphilis-screening coverage of a random sample of 384 Aboriginal people aged 15 to 25 years was examined in 1998. This showed that only 21 per cent had been tested for syphilis within the past year, 29 per cent within the past 2 years and 45 per cent at any time in the past, leaving 55 per cent who had never been tested. An audit of the SS recall systems in 8 Community Health and remote area clinics showed that 18 per cent of patients within the system were 1 month overdue for their SS, 14 per cent were 2 to 3 months overdue and 41 per cent were over 3 months overdue. The largest proportion of overdue tests (43%) was from patients overdue for periodic screening (Figure 2).

The stability of syphilis incidence rates over the 5 years to 1999 and the consistent drop in the incidence of early syphilis over the last 4 of these years (Kimberley Public

* There was no early latent category because the limited clinical information obtained from pathology request forms does not allow accurate differentiation of early latent and secondary syphilis. It is likely that some cases classified as secondary syphilis, were in fact, early latent.

Figure 2. Distribution of overdue syphilis serology tests in the Kimberley, 1 January 1996 to 6 December 1999, by indication for test

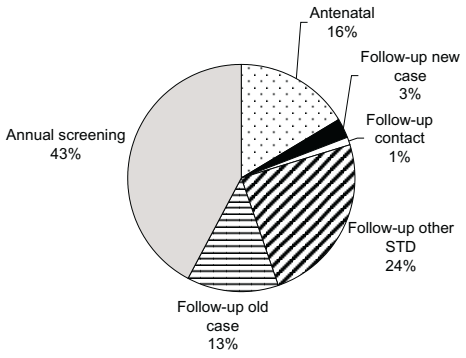


Figure 3. Kimberley syphilis rates per 1,000 person years, 1986 to 1998

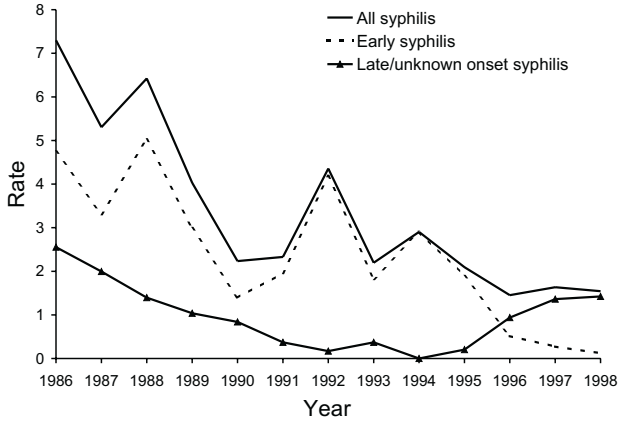


Table 1. Demographic characteristics of syphilis cases in the Kimberley, 1 January 1996 to 6 December 1999, by clinical stage*

	All cases[a]		Primary and secondary[b]		Early syphilis, confirmed or probable[c]		Late latent[d]	
Male: female	117:79		21:25		29:30		96:54	
Age (years)								
Range	10-67		15-46		15-48		10-67	
Median	30		25		25		32	
Race	n	%	n	%	n	%	n	%
Aboriginal	154	78.6	44	95.7	55	93.2	111	73.3
Indonesian/Middle East	26	13.4	0	0.0	2	3.4	26	17.3
Other non-Aboriginal	16	8.2	2	3.4	2	3.4	14	9.3
NT residents	23	11.7	4	8.7	7	11.9	19	12.7

* a = b + d ; c = b + some cases from d

Health Unit, unpublished data; Figure 3) prompted, in December 1999, re-evaluation of the merit of periodic screening.[3]

During the study period, 196 cases of syphilis (117 male, 79 female) were diagnosed; 14 (7.1%) were primary, 32 (16.3%) secondary and 150 (76.5%) late latent cases. Early syphilis, confirmed or probable, accounted for 59 (30.1%) of cases. Cases of late latent syphilis were more likely to be male, older and of overseas origin (Table 1). Health services in Broome, Fitzroy Crossing and Halls Creek districts contributed the majority of cases (Table 2).

As syphilis rates in the Northern Territory (NT) districts that border the Kimberley are up to twice the Kimberley rates, it was noted that NT residents accounted for around 10 per cent of cases.[4] An increasing number and proportion of cases were of overseas origin (Figure 4). Almost all of these were inactive, non-infectious cases.

Periodic screening contributed to only about 10 per cent of syphilis cases, whereas SS testing as a result of STD symptoms, STD contact, institutional screening and other screening contributed to much larger proportions of cases (Figures 5 and 6).

Table 2. Distribution of syphilis cases in the Kimberley, 1 January 1996 to 6 December 1999, by district of health service provider

District	n	%
Broome	57	29.1
Derby	27	13.8
Fitzroy Crossing	39	19.9
Halls Creek	39	19.9
Wyndham	12	6.1
Kununurra	22	11.2
Total	196	100.0

Figure 4. Syphilis cases in the Kimberley, 1 January 1996 to 6 December 1999, by race and year

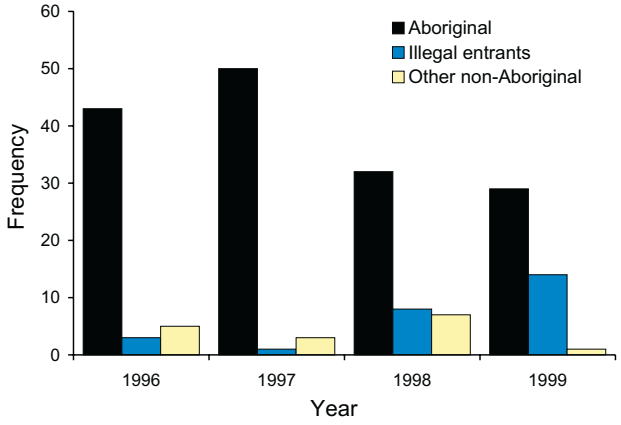
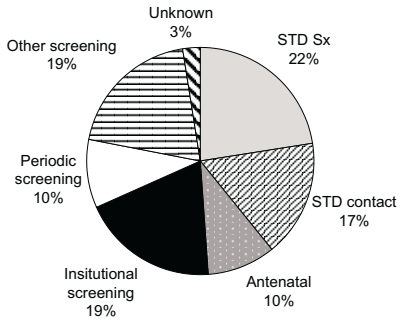
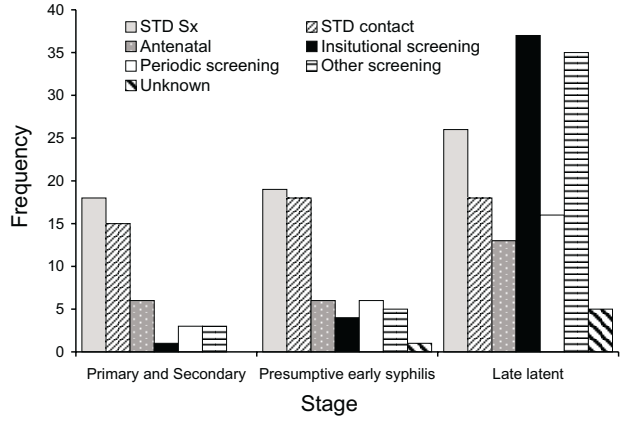


Figure 5. Clinical indication for syphilis testing, n = 196



Sx = symptoms

Figure 6. Clinical indication for syphilis testing by stage of syphilis



Sx = symptoms

Discussion

Syphilis represents a STD control success story in the Kimberley. The syphilis incidence rate has decreased 10-fold since the mid-1980s and the proportion of cases transmitted locally is decreasing. Declining yields by age group could not be determined, as data on testing activity are incomplete (Figure 1). The decrease in SS testing activity between 1990 and 1995-7 was probably due to a combination of factors including decreased syphilis incidence (and therefore fewer post-treatment SSs being done), changes in the organisational management of some health services resulting in STD control being lower on their list of priorities, and the increasing movement of Aboriginal people to out-stations without a commensurate reorganisation of health resources.

In January 2000, the periodic syphilis-screening program was discontinued because it had contributed to only 10 per cent of cases detected during the recent period. This could be, at least in part, due to the fact that too few tests are done for periodic screening. KPHU does not have sufficient data on the proportion of cases detected in this way in the past to prove or disprove this suggestion. However, as KPHU receives approximately 1,000 STD notifications each year - and there are approximately 700 births in the Kimberley each year (with each STD or birth generating two SS tests ideally) - it is probably reasonable to assume that at least 1,000 to 2,000 SSs each year are done for periodic or other screening unrelated to STDs and pregnancy. Thus, in absolute terms the number of SSs done for periodic screening is quite large, even though screening of those 15 to 25 years old is incomplete.

It might be argued that an increasing incidence of late/unknown-onset syphilis coupled with a decreasing incidence of early syphilis, as observed since 1995 (Figure 3), indicates delay in the detection and management of syphilis cases. However, closer scrutiny of the late/unknown-onset cases detected since 1995 showed that the vast majority were middle-aged or elderly people who had been seropositive (negative or low titre RPR and positive TPHA) for a decade or more but had no documentation of having had a definitive syphilis treatment. Some of these people may have been treated for syphilis but without adequate documentation. Others may have been seropositive due to yaws.[6,7,8] Probably only a small proportion had untreated late latent syphilis, but it was decided to err on the side of caution and offer these patients a definitive treatment for late latent syphilis.

Further evidence against a delay in the detection and management of syphilis cases comes from the fact that, in contrast to some other areas of rural Australia, there have been no reports of congenital syphilis in the Kimberley since 1989.[5,9,10]

In 1996 azithromycin (1.0 g to cover chlamydia) was introduced as one component of the standard epidemiological treatment (with amoxycillin and probenecid) for uncomplicated urethritis/cervicitis in the Kimberley and neighbouring areas of northern Australia. This is a possible reason for the observed decrease in early syphilis rates in the Kimberly. While azithromycin is not a recognised syphilis treatment, a single dose of one gram has been shown to be efficacious for prevention of syphilis in people exposed to infected sexual partners.[11]

The authors are aware of recent initiatives in STD control in Central Australia where, in addition to targeted STD-screening in known high-risk groups, mass screening for gonorrhoea and chlamydia, and opportunistic screening for syphilis, are currently recommended.[12] The Kimberley situation is quite different from that of central Australia. Organised syphilis screening, STD contact tracing, STD notification and a regional syphilis register were established over 15 years ago in the Kimberley, whereas a similar level of health service infrastructure in central Australia was established in much more recent times. This is why the Kimberley is stopping one specific syphilis-screening strategy, whereas other areas in Australia are increasing syphilis-screening activity.

To ensure that stopping the periodic syphilis-screening program does not result in increased syphilis transmission, KPHU will be encouraging local health services to redirect resources previously devoted to periodic syphilis screening into more effective strategies to diagnose and control transmission of the disease. For example, ensuring that all patients with STD symptoms and STD contacts have syphilis testing both at presentation (currently not well done) and 3 months later, and more timely administration of syphilis treatment for infectious patients.[3,13]

In addition, KPHU will be monitoring the effects of the new policy on the number of syphilis tests done each month, and health services will be asked to audit their SS recall systems in late 2000 to ensure that the proportion of overdue serology tests has decreased. Should the decision not to screen prove to have been misjudged, existing Kimberley policies of syphilis screening for all antenatal women and newborns (cord blood SS) and prison inmates will provide sentinel populations for detecting any increase in syphilis incidence.

Acknowledgments

Thank you to Ruth Southern and Maree Hose (Public Health Nurses) for maintaining the register without which this work would have been impossible. Many thanks to all health staff in the Kimberley who contributed towards the periodic syphilis-screening program during its lifetime, 1986-99.

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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 November, 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'.*

In November 2000, compared with the 5-year mean, there was an increase in reports of chlamydial infection (ratio 1.5), *Haemophilus influenzae* type b (ratio 1.6), Barmah Forest virus (ratio 1.5), legionellosis (ratio 1.2) and meningococcal infection (Ratio 1.2) (Figure 10, Table 1).

Gastrointestinal illness

There continues to be fewer notifications of *Campylobacter* and *Salmonella* than in previous years with rates of 113.8/100,000 population for *Campylobacter* (Figure 1) and 27.0/100,000 population for *Salmonella*. Tasmania had the highest rate for *Campylobacter* (171.0/100,000 population) and the Northern Territory the highest rate for *Salmonella* (192.9/100,000 population).

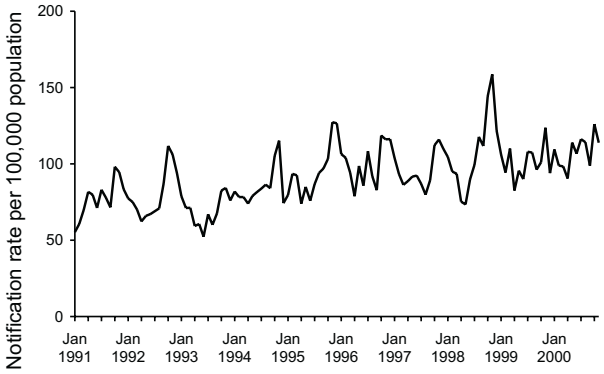
The Communicable Disease Control Branch in South Australia is investigating an apparent cluster of cases of *Campylobacter* infection in residents of a small rural community. To determine the source of the cluster, hypothesis-generating interviews were conducted with cases. A case-control study found a statistically significant association between *Campylobacter* infection and the consumption of raw milk.

South Australia is also investigating an outbreak of gastroenteritis in an aged residential care facility. Norwalk virus has been detected in several faecal specimens.

Shiga toxin producing *Escherichia coli* was detected in South Australia in a 4-year-old male. The parents reported extensive contact with calves and other farm animals.

New South Wales reported one case of typhoid in a 67-year-old female.

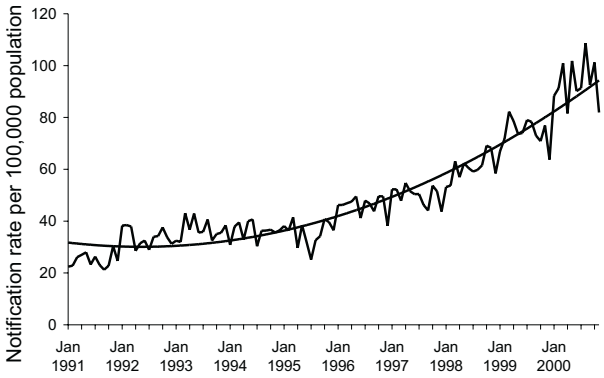
Figure 1. Notification rate of campylobacteriosis, Australia, 1 January 1991 to 30 November 2000, by month of notification



Chlamydial infection

There were 1297 notifications of chlamydial infection in November 2000 – a notification rate of 81.9/100,000 population which is an increase from previous years (Figure 2). Of these cases, 77 per cent were in the 15 to 29 years age group and the male:female ratio was 0.6:1. The Northern Territory continues to have the highest rate for chlamydial infection (429.3/100,000 population).

Figure 2. Notification rate of chlamydial infection, Australia, 1 January 1991 to 30 November 2000, by month of notification

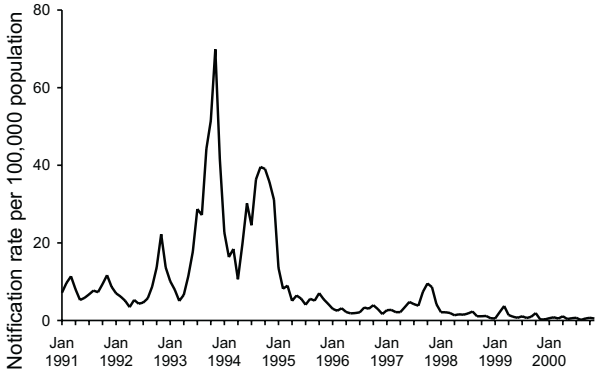


Vaccine preventable diseases

With the exception of *Haemophilus influenzae* type b, there were fewer reports of all vaccine preventable diseases this month than for the 5-year mean for November. Of the *Haemophilus influenzae* type b cases, 3 were males (aged 2, 12 and 49 years) and 3 were females (aged 53 and 58 years, and age unknown).

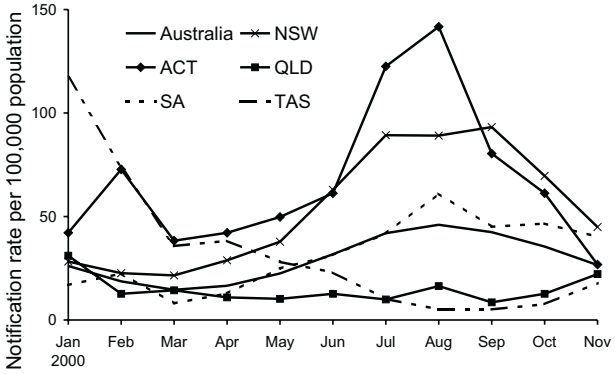
Measles cases continue to be at their lowest level since the national notification system began (Figure 3). Of the 8 cases reported in November 2000, 7 were reported in New South Wales and one in Victoria. The cases included 2 four-year-old females (partially vaccinated), 2 one-year-old males (one unvaccinated and one vaccination status unknown), 2 infants under the age of one year (not yet vaccinated), a 5-year-old male (partially vaccinated) and a 21-year-old male (vaccination status unknown).

Figure 3. Notification rate of measles, Australia, 1 January 1991 to 30 November 2000, by month of notification



Pertussis notifications are down from last month (420 cases with a rate of 26.6/100,000 population compared with 561 cases with a rate of 35.5/100,000 population). Since August 2000 (when the national rate peaked at 46.0/100,000 population) the rates for New South Wales, the Australian Capital Territory and South Australia have decreased, while those for Queensland and Tasmania have increased (Figure 4). The rates for Western Australia and the Northern Territory remain unchanged.

Figure 4. Notification rate for pertussis, Australia, Australian Capital Territory, New South Wales, Queensland, South Australia and Tasmania, 1 January to 30 November 2000, by month of notification

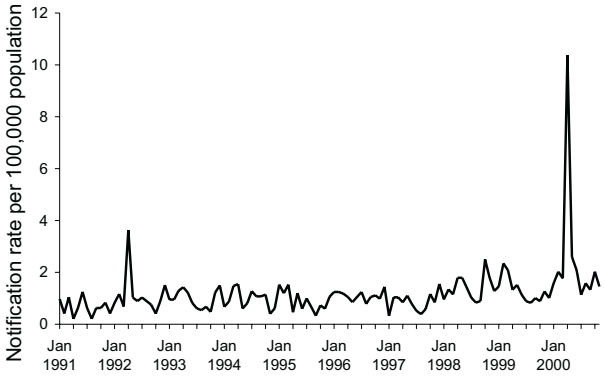


Legionellosis

There were 23 notifications of legionellosis in November 2000 a notification rate of 1.5/100,000 population (Figure 5). All of the cases in New South Wales (2) and South Australia (4) were *Legionella longbeachae* and all of the cases in Victoria (11) and Queensland (3) were *L. pneumophila*. For the other cases information on the *Legionella* species involved was not available.

According to the Melbourne Age (6 January) a man who died on 12 December 2000 from legionnaires' disease was probably infected while a patient at the Royal Melbourne Hospital. The man was being treated for an unrelated condition when he displayed fever symptoms later diagnosed as legionnaires' disease. On the date the patient died, the hospital commissioned tests of its air-conditioning cooling towers. Samples from 2 of the towers contained several strains of *Legionella*, including the one the patient contracted. Three other people known to have visited the hospital have also been diagnosed with legionnaires' disease. Samples are being tested to see whether strains responsible match those in the cooling tower water.

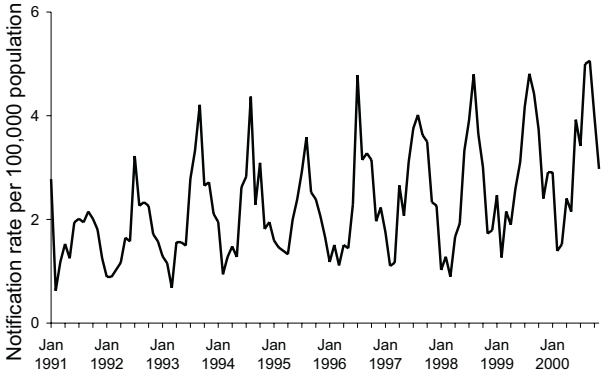
Figure 5. Notification rate for legionellosis, Australia, 1 January 1991 to 30 November 2000, by month of notification



Meningococcal infections

There were 47 notifications of invasive meningococcal disease in November 2000 – a notification rate of 3.0/100,000 population (Figure 6). Of these cases, 34 per cent were under 5 years of age and 23 per cent were in the 10 to 19 years age group. The serogroups were available for 27 cases; these were serogroup B (44%) and serogroup C (56%).

Figure 6. Notification rate of invasive meningococcal disease, Australia, 1 January 1991 to 30 November 2000, by month of notification



Vectorborne diseases

Barmah Forest virus infection notifications have increased since last month (57 cases with a rate of 3.6/100,000 population compared with 43 cases with a rate of 2.7/100,000 population) and from the 5-year mean (37 cases). Queensland reported the highest rate (13.7/100,000 population) and the majority of cases (40) (Figure 7).

Ross River Virus notifications have also increased since last month (129 cases with a rate of 8.2/100,000 population compared with 124 cases with a rate of 7.8/100,000 population; Figure 8) with the highest rate being in South Australia (25.7/100,000 population).

During the past 9 weeks, 56 cases of Ross River virus infection have been notified in South Australia. Of these, 46 notifications have been received for residents of the West Coast region. Laboratory notifications in small numbers have been received for residents of Adelaide, Murray Bridge, Mount Gambier, York Peninsula, Adelaide Hills and the Riverland. Literature on the prevention of Ross River virus infection has been distributed to local council and tourist information centres located in the West Coast region of South Australia.

In a press release dated 27 December 2000 (sourced through ProMED) the Department of Human Services, South Australia has warned visitors to take particular care to avoid being bitten by mosquitoes. The warning follows an increase in reported cases of Ross River virus across

Figure 7. Notification rate of Barmah Forest virus, Australia and Queensland, 1 January 1995 to 30 November 2000, by month of notification

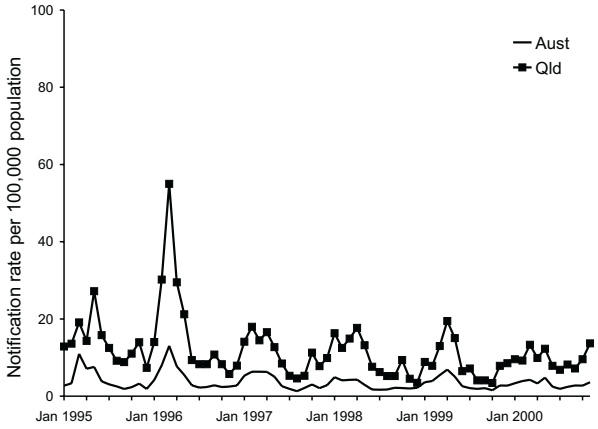
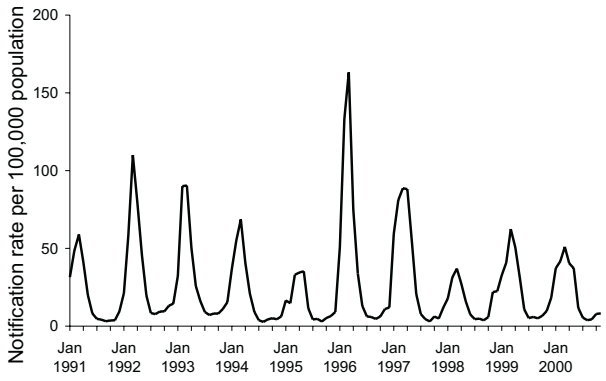


Figure 8. Notification rate of Ross River virus, Australia, 1 January 1991 to 30 November 2000, by month of notification



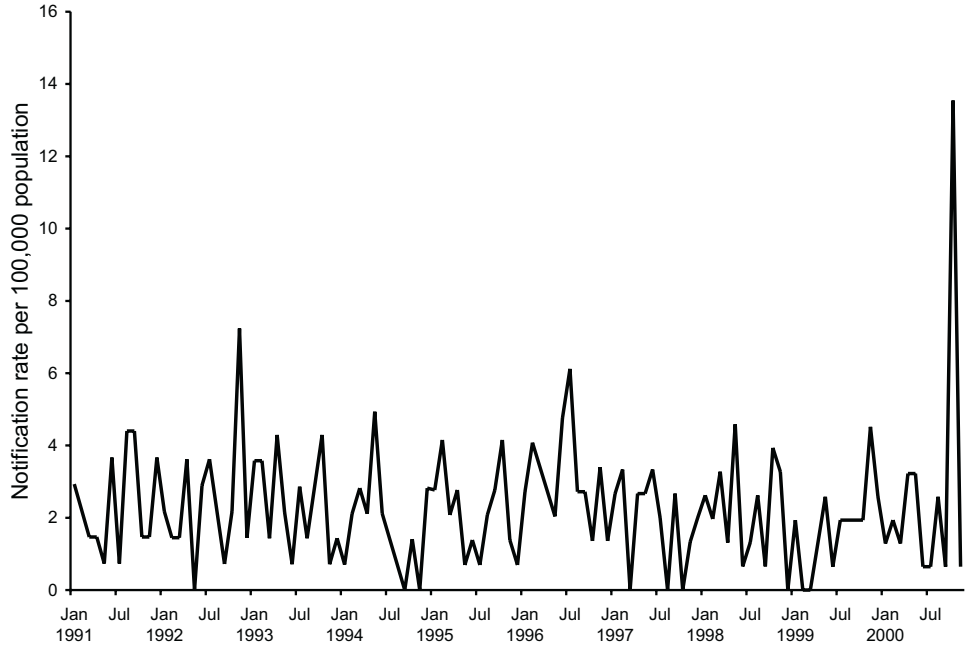
different parts of the State. Cases of Ross River virus infection have been reported on the West Coast, Kangaroo Island, and near Lake Albert. People living or on holiday in South Australia should take protective measures against mosquitoes by covering exposed skin with appropriate clothing and using repellents. The State Government recently launched a television and radio community awareness campaign called 'Don't Let the Bloodsuckers Bite'. Brochures are also being distributed to local councils in rural areas.

According to an Australian Broadcasting Corporation news report (8 January 2001) a warning about the threat of Ross River virus has been issued by the Northern Territory Centre for Disease Control. Mosquitoes are in high numbers in the Katherine, Barkly and Gove regions because of flooding. The centre reports 9 cases in Katherine (more than normally expected) and 9 in East Arnhem and Darwin in the past 2 weeks.

Malaria in illegal entrants - Western Australia

The malaria cases notified in October 2000 from Western Australia (Figure 9) were primarily among a boatload of 48 illegal entrants who arrived in the Kimberley via Ashmore Reef and were sent to Curtin Detention Centre. Among this group there were 9 falciparum cases, 7 vivax cases and a single combined falciparum/vivax infection. Three falciparum cases required hospital admission. Just prior to departing for Australia the group had spent 1 to 3 weeks on Sabo Island in Indonesia and it is assumed they were infected there as mosquitoes were prevalent and there was no mosquito control or antimalarial prophylaxis. (*Information provided by Dr Gary Dowse, based on a report by Dr Richard Thomas, published in the* Kimberley Public Health Bulletin*, November 2000*).

Figure 9. Notification rate of malaria, Western Australia, 1 January 1991 to 30 November 2000, by month of notification



Case report: Murine typhus acquired in Bali

Contributed by Dr Gary Dowse, Communicable Diseases Control Branch, Health Department of Western Australia (edited)

We have been notified of a case of murine typhus (*R. typhi*) in a woman who attended a conference of about 360 travel consultants in Bali from approximately 27 November to 1 December 2000. During this period registrants were confined to a 5-star hotel except for an evening function on the other side of the island. On that occasion the woman walked barefoot through some damp grass, but otherwise had no obvious exposure risks. After the Conference she stayed at another 5-star hotel for 4 days before returning to Perth. Her illness onset was 7 December. Exposure in Perth is very unlikely so it seems that her infection was almost certainly acquired in Bali.

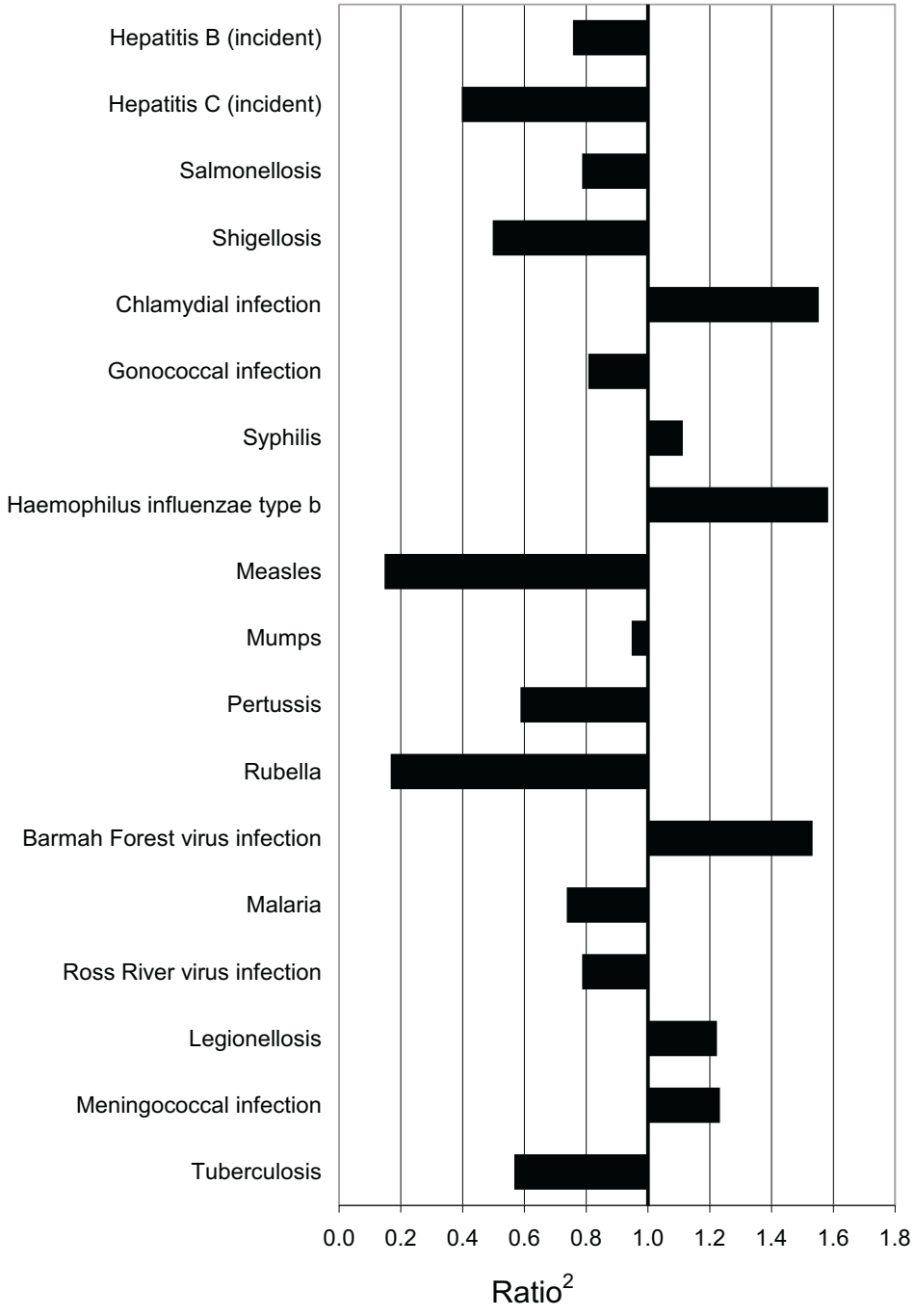
Tables

There were 6,054 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in November 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 November 2000, are included in this issue of *Commun Dis Intell* (Table 3). Figure 10 illustrates, for selected diseases, the November 2000 totals for selected diseases as ratios to the mean of their October to December levels for the previous 5 years.

There were 1,398 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 30 November 2000 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 43 to 47, ending 26 November 2000, are included in this issue of *Commun Dis Intell (*Table 6*).*

Figure 10. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 30 November 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 xx to xx data for the previous five years

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 30 November 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total November 2000[1]	Total October 2000[1]	Total November 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	0	3	1	4	2	3	2	2	17	23	21	22	368	255	0.8
Hepatitis B (unspecified)[2]	3	156	0	79	14	1	207	49	509	651	745	535	7, 540	6,388	1.0
Hepatitis C (incident)	0	2	0	-	1	1	1	3	8	22	51	20	433	189	0.4
Hepatitis C (unspecified)[2]	13	350	15	273	41	20	363	114	1,189	1521	1,768	1,303	18,810	14,641	0.9
Hepatitis D	0	0	0	0	0	0	1	0	1	0	1	2	25	19	0.4
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	2	0	0	2	0	0.0
Campylobacterosis[3]	35	-	14	292	150	67	484	149	1,191	1319	1,294	1,218	12,419	1,1061	1.0
Haemolytic uraemic syndrome	0	0	0	0	0	0	0	0	0	2	5	1	9	n/a	-
Hepatitis A	1	11	2	8	2	0	3	8	35	50	123	151	764	2,013	0.2
Hepatitis E	0	0	0	0	0	0	0	NN	0	1	0	0	1	3	0.0
Listeriosis	0	2	0	0	1	0	0	0	3	4	3	5	59	59	0.6
Salmonellosis	9	77	31	145	25	21	78	40	426	429	452	539	5, 517	6,140	0.8
Shigellosis[3]	0	-	11	5	1	0	8	1	26	39	32	52	432	624	0.5
SLTEC, VTEC[4]	0	0	0	NN	1	0	0	NN	1	2	12	2	32	n/a	-
Typhoid	0	1	0	0	0	0	0	0	1	1	2	5	59	66	0.2
Yersiniosis[3]	0	-	0	3	0	0	0	0	3	5	9	18	67	215	0.2
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]	16	278	69	444	144	23	169	154	1,297	1,600	1,215	837	16,274	9,151	1.6
Donovanosis	0	0	0	0	NN	0	0	0	0	2	1	3	12	42	0.0
Gonococcal infection[6]	0	49	66	57	3	0	73	59	307	413	550	381	5,623	4,277	0.8
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Syphilis[7]	2	40	9	82	0	0	0	4	137	179	137	124	1768	1555	1.1

Table 1 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 30 November 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total November 2000[1]	Total October 2000[1]	Total November 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	2	1	1	0	0	2	0	6	1	3	4	28	46	1.6
Measles	0	7	0	0	0	0	1	0	8	11	4	54	102	577	0.2
Mumps	0	4	1	0	2	0	2	3	12	12	11	13	200	157	1.0
Pertussis	7	240	0	65	50	7	45	6	420	561	572	709	5,099	5,268	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Rubella[8]	1	27	0	6	0	0	4	3	41	61	30	248	282	1,738	0.2
Tetanus	0	0	0	0	0	0	0	0	0	2	0	1	7	5	-
Vectorborne															
Arbovirus infection NEC	0	0	0	0	0	0	0	0	0	1	1	3	64	53	0.0
Barmah Forest virus infection	0	12	1	40	1	0	3	0	57	43	44	37	561	660	1.5
Dengue	0	0	0	1	1	0	0	1	3	3	8	23	209	161	0.1
Malaria	0	6	4	11	2	1	8	1	33	69	50	45	889	678	0.7
Ross River virus infection	1	12	3	71	32	0	6	4	129	124	155	163	3,937	4,716	0.8
Zoonoses															
Brucellosis	0	0	0	4	0	0	0	0	4	3	9	4	24	37	0.9
Hydatid infection	0	NN	0	0	0	0	3	0	3	2	0	5	25	40	0.6
Leptospirosis	0	5	2	3	3	0	1	0	14	18	17	19	213	186	0.7
Ornithosis	0	NN	0	NN	1	0	4	0	5	11	10	13	78	78	0.4
Q fever	0	5	0	38	0	0	1	0	44	40	41	45	481	497	1.0
Other															
Legionellosis	1	2	1	3	4	0	11	1	23	32	20	19	442	187	1.2
Leprosy	0	1	0	0	0	0	0	0	1	0	0	0	4	7	2.2
Meningococcal infection	0	18	0	4	0	2	19	4	47	63	38	38	549	428	1.2
Tuberculosis	0	15	0	2	3	0	26	7	53	71	113	93	854	975	0.6
Total	89	1,325	231	1,641	484	146	1,525	613	6,054	7,393	7, 547	6,755	84,263	73,197	

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 mean of last 5 years calculated as described above.

n/a Not calculated as only notifiable for under 5 years.

Table 2. Crude incidence of diseases by State or Territory, 1 to 30 November 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	0.6	6.2	1.4	1.6	7.7	0.5	1.3	1.1
Hepatitis B (unspecified)[2]	11.5	29.2	0.0	27.0	11.3	2.6	52.7	31.6	32.2
Hepatitis C (incident)	0.0	0.4	0.0	-	0.8	2.6	0.3	1.9	0.6
Hepatitis C (unspecified)[2]	49.8	65.5	93.3	93.3	33.0	51.0	92.4	73.5	75.2
Hepatitis D	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.1
Gastrointestinal									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[3]	134.0	-	87.1	99.8	120.6	171.0	123.3	96.1	113.8
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	3.8	2.1	12.4	2.7	1.6	0.0	0.8	5.2	2.2
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.0	NN	0.0
Listeriosis	0.0	0.4	0.0	0.0	0.8	0.0	0.0	0.0	0.2
Salmonellosis	34.5	14.4	192.9	49.5	20.1	53.6	19.9	25.8	27.0
Shigellosis[3]	0.0	-	68.4	1.7	0.8	0.0	2.0	0.6	2.5
SLTEC, VTEC[4]	0.0	0.0	0.0	NN	0.8	0.0	0.0	NN	0.1
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	1.0	0.0	0.0	0.0	0.0	0.3
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]	61.3	52.0	429.3	151.7	115.7	58.7	43.0	99.3	82.1
Donovanosis	0.0	0.0	0.0	0.0	NN	0.0	0.0	0.0	0.0
Gonococcal infection[6]	0.0	9.2	410.6	19.5	2.4	0.0	18.6	38.0	19.4
Lymphogranuloma venereum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis[7]	7.7	7.5	56.0	28.0	0.0	0.0	0.0	2.6	8.7
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	0.4	6.2	0.3	0.0	0.0	0.5	0.0	0.4
Measles	0.0	1.3	0.0	0.0	0.0	0.0	0.3	0.0	0.5
Mumps	0.0	0.7	6.2	0.0	1.6	0.0	0.5	1.9	0.8
Pertussis	26.8	44.9	0.0	22.2	40.2	17.9	11.5	3.9	26.6
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[8]	3.8	5.1	0.0	2.0	0.0	0.0	1.0	1.9	2.6
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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	2.2	6.2	13.7	0.8	0.0	0.8	0.0	3.6
Dengue	0.0	0.0	0.0	0.3	0.8	0.0	0.0	0.6	0.2
Malaria	0.0	1.1	24.9	3.8	1.6	2.6	2.0	0.6	2.1
Ross River virus infection	3.8	2.2	18.7	24.3	25.7	0.0	1.5	2.6	8.2

Table 2 (continued). Crude incidence of diseases by State or Territory, 1 to 30 November 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	1.4	0.0	0.0	0.0	0.0	0.3
Hydatid infection	0.0	NN	0.0	0.0	0.0	0.0	0.8	0.0	0.3
Leptospirosis	0.0	0.9	12.4	1.0	2.4	0.0	0.3	0.0	0.9
Ornithosis	0.0	NN	0.0	NN	0.8	0.0	1.0	0.0	0.7
Q fever	0.0	0.9	0.0	13.0	0.0	0.0	0.3	0.0	2.8
Other									
Legionellosis	3.8	0.4	6.2	1.0	3.2	0.0	2.8	0.6	1.5
Leprosy	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Meningococcal infection	0.0	3.4	0.0	1.4	0.0	5.1	4.8	2.6	3.0
Tuberculosis	0.0	2.8	0.0	0.7	2.4	0.0	6.6	4.5	3.4
Total	340.8	248.0	1,437.1	560.6	389.0	372.6	388.6	395.3	383.1

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 30 November 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	5	1	3	3	5	6	3	26	380
Hepatitis B (unspecified)[2]	4	352	0	79	35	2	211	64	747	7,791
Hepatitis C (incident)	1	3	0	-	3	1	1	3	12	449
Hepatitis C (unspecified)[2]	15	594	25	274	91	32	364	128	1,523	19,174
Hepatitis D	0	0	0	0	0	0	1	0	1	25
Gastrointestinal										
Botulism	0	0	1	0	0	0	0	0	1	2
Campylobacterosis[3]	43	-	23	285	181	72	563	186	1,353	12,541
Haemolytic uraemic syndrome	0	0	0	2	0	0	0	0	2	9
Hepatitis A	1	15	1	14	3	0	4	11	49	798
Hepatitis E	0	0	0	0	0	1	0	NN	1	1
Listeriosis	0	2	0	0	1	0	1	0	4	60
Salmonellosis	9	96	25	144	28	15	86	65	468	5,664
Shigellosis[3]	0	-	15	3	1	0	9	2	30	436
SLTEC, VTEC[4]	0	0	0	NN	1	0	0	NN	1	34
Typhoid	0	0	0	0	0	0	0	0	0	63
Yersiniosis[3]	0	-	0	3	0	0	0	1	4	68
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]	19	337	92	454	166	25	248	198	1,539	16,370
Donovanosis	0	0	1	0	NN	0	0	0	1	13
Gonococcal infection[6]	1	58	94	75	10	0	67	77	382	5,726
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0
Syphilis[7]	3	61	14	93	0	1	0	7	179	1833
Vaccine preventable										
Diphtheria	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	2	0	0	0	0	3	0	5	28
Measles	0	7	0	2	0	0	1	0	10	103
Mumps	0	5	1	0	2	0	2	3	13	204
Pertussis	7	454	0	62	73	6	65	15	682	5,289
Poliomyelitis	0	0	0	0	0	0	0	0	0	0
Rubella[8]	1	47	0	5	0	0	2	2	57	283
Tetanus	0	0	0	0	0	0	1	0	1	8
Vectorborne										
Arbovirus infection NEC	0	0	0	1	0	0	0	0	1	65
Barmah Forest virus infection	0	12	0	37	7	0	3	1	60	570
Dengue	0	0	0	1	1	0	0	1	3	230
Malaria	0	11	6	17	2	1	11	0	48	920
Ross River virus infection	0	13	3	70	37	0	6	12	141	4,129

Table 3 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 30 November 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	2	0	0	0	0	2	22
Hydatid infection	0	NN	0	0	0	0	3	1	4	26
Leptospirosis	0	10	3	5	3	0	9	0	30	227
Ornithosis	0	NN	0	NN	2	0	10	0	12	87
Q fever	0	8	0	35	0	0	1	1	45	501
Other										
Legionellosis	1	4	0	3	10	0	10	6	34	443
Leprosy	0	1	0	0	0	0	0	0	1	5
Meningococcal infection	0	15	1	5	0	1	21	4	47	554
Tuberculosis	2	37	8	11	3	1	29	14	105	961
Total	107	2,149	314	1,685	663	163	1,738	805	7,624	86,093

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 30 November 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	109	173
	New Children's Hospital, Westmead	61	142
	Repatriation General Hospital, Concord	-	-
	Royal Prince Alfred Hospital, Camperdown	51	45
	South West Area Pathology Service, Liverpool	-	-
Queensland	Queensland Medical Laboratory, West End	331	354
	Townsville General Hospital	7	8
South Australia	Institute of Medical and Veterinary Science, Adelaide	533	593
Tasmania	Northern Tasmanian Pathology Service, Launceston	8	8
	Royal Hobart Hospital, Hobart	-	-
Victoria	Monash Medical Centre, Melbourne	19	164
	Royal Children's Hospital, Melbourne	67	73
	Victorian Infectious Diseases Reference Laboratory, Fairfield	138	211
Western Australia	PathCentre Virology, Perth	-	-
	Princess Margaret Hospital, Perth	74	53
	Western Diagnostic Pathology	-	27
Total		1,398	1,851

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 30 November 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												
Measles virus	1	2	-	-	-	-	-	-	3	1	42	169
Rubella virus	-	-	-	1	-	-	1	-	2	11	41	140
Hepatitis viruses										-	-	-
Hepatitis A virus	-	-	1	5	1	-	-	-	7	33	133	351
Hepatitis D virus	-	-	-	-	-	-	2	-	2	-	8	7
Arboviruses												
Ross River virus	-	-	3	10	27	-	2	-	42	55	1,181	1,294
Barmah Forest virus	-	1	-	14	-	-	-	-	15	17	144	158
Flavivirus (unspecified)	-	-	-	-	-	-	1	-	1		40	22
Adenoviruses												
Adenovirus type 3	-	-	-	-	-	-	1	-	1	3	18	26
Adenovirus type 5	-	-	-	-	1	-	-	-	1	-	8	6
Adenovirus not typed/pending	1	13	-	-	27	1	7	6	59	129	889	1,006
Herpes viruses												
Cytomegalovirus	1	19	-	10	50	7	25	4	117	156	1,159	1,071
Varicella-zoster virus	1	3	-	4	14	2	39	-	63	167	1,107	1,484
Epstein-Barr virus	-	2	-	26	77	-	11	-	116	286	1,728	1,989
Other DNA viruses												
Parvovirus	-	-	-	20	5	-	1	-	26	34	314	412
Picornavirus family												
Coxsackievirus A9	-	1	-	-	-	-	-	-	1	1	10	9
Coxsackievirus B4	-	-	-	-	-	-	4	-	4	-	11	1
Echovirus type 30	-	-	-	-	-	-	2	-	2	-	117	10
Poliovirus type 1 (uncharacterised)	1	2	-	-	-	-	-	-	3	4	15	26
Poliovirus type 2 (uncharacterised)	-	1	-	-	-	-	-	-	1	1	7	14
Poliovirus type 3 (uncharacterised)	-	1	-	-	-	-	-	-	1	-	8	8
Rhinovirus (all types)	-	30	-	-	-	-	3	-	33	74	340	450
Enterovirus type 71 (BCR)	-	1	-	-	-	-	-	-	1	2	1	11
Enterovirus not typed/pending	-	-	-	1	-	1	7	-	9	56	681	704
Ortho/paramyxoviruses												
Influenza A virus	5	2	-	4	54	-	2	8	76	69	1,317	1,828
Influenza B virus	2	7	-	3	17	-	1	5	36	9	522	275
Parainfluenza virus type 1	-	-	-	-	6	-	-	-	6	2	229	38
Parainfluenza virus type 2	-	-	-	-	1	-	-	-	1	9	35	107
Parainfluenza virus type 3	-	10	-	6	14	-	1	27	62	115	388	741
Respiratory syncytial virus	-	19	-	2	8	-	2	2	35	129	2,645	2,977
Other RNA viruses												
Rotavirus	1	35	-	-	70	2	12	14	138	270	1,553	2,077
Norwalk agent	-	-	-	-	3	-	1	-	4	1	57	55
Other												
Chlamydia trachomatis not typed	5	53	13	80	63	1	5	8	228	338	2,684	2,967
Chlamydia psittaci	-	-	-	-	-	2	14	-	16	5	93	73
Mycoplasma pneumoniae	-	9	2	24	23	1	11	-	71	128	577	1,033
Mycoplasma hominis	-	1	-	-	-	-	-	-	1	-	8	5
Coxiella burnetii (Q fever)	-	1	-	7	1	-	4	-	13	15	80	207
Rickettsia australis	-	-	-	-	-	-	1	-	1	-	2	2
Rickettsia - Spotted fever group	-	-	-	-	-	1	-	-	1	-	2	1
Streptococcus group A	-	1	3	23	-	-	8	-	35	58	329	321

Table 5 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 30 November 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				
Bordetella pertussis	-	7	-	8	24	-	21	-	60	135	580	769
Legionella pneumophila	-	-	-	-	-	-	1	-	1	-	38	17
Legionella longbeachae	-	-	-	-	6	-	1	-	7	12	52	39
Cryptococcus species	-	-	-	-	1	-	-	-	1	2	15	9
Leptospira species	-	-	-	-	4	-	-	-	4	10	51	51
Treponema pallidum	-	5	19	25	37	-	-	-	86	130	762	685
Entamoeba histolytica	-	-	-	1	-	-	2	-	3	1	17	6
Toxoplasma gondii	-	-	-	-	-	-	1	-	2	2	15	8
Total									1,398	2,470	20,053	23,659

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 43 to 47, 2000

Week number	43		44		45	
Week ending on	29 October 2000		5 November 2000		12 November 2000	
Doctors reporting	61		58		59	
Total encounters	7,642		7,373		7,119	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	46	6.0	51	6.9	24	3.4
Chickenpox	8	1.0	6	0.8	10	1.4
Gastroenteritis	67	8.8	69	9.4	56	7.9
Gastroenteritis with stool culture	12	1.6	10	1.4	7	1.0
ADT immunisations	29	3.8	30	4.1	20	2.8

Table 6 (continued). Australian Sentinel Practice Research Network reports, weeks 43 to 47, 2000

Week number	46		47	
Week ending on	19 November 2000		26 November 2000	
Doctors reporting	58		60	
Total encounters	6,908		7,180	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	21	3.0	14	1.9
Chickenpox	12	1.7	17	2.4
Gastroenteritis	58	8.4	66	9.2
Gastroenteritis with stool culture	7	1.0	7	1.0
ADT immunisations	31	4.5	28	3.9

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

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 31 July 2000, as reported to 31 October 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 to 31 July 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	3	0	2	1	0	2	0	8	10	47	44
	Male	1	22	0	7	1	0	13	0	44	63	379	371
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total[1]	1	25	0	9	2	0	15	0	52	73	428	415
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	2	9	9
	Male	0	4	0	2	0	0	2	0	8	9	98	80
	Total[1]	0	4	0	2	0	0	2	0	8	11	107	89
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	5	3
	Male	0	5	0	0	0	0	2	2	9	11	64	68
	Total[1]	0	5	0	0	0	0	2	2	9	12	69	72

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 31 July 2000, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	28	616	9	160	62	5	221	119	1,220
	Male	226	11,109	108	2,031	682	78	3,955	926	19,115
	Sex not reported	0	246	0	0	0	0	24	0	270
	Total[1]	254	11,992	117	2,198	744	83	4,214	1,049	20,651
AIDS diagnoses	Female	9	188	0	49	25	3	71	26	371
	Male	87	4,688	35	832	347	45	1,647	356	8,037
	Total[1]	96	4,888	35	883	372	48	1,726	384	8,432
AIDS deaths	Female	4	114	0	32	15	2	49	17	233
	Male	66	3,219	24	571	231	29	1,281	252	5,673
	Total[1]	70	3,341	24	605	246	31	1,336	270	5,923

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

Bulletin Board

Master of Applied Epidemiology
3rd MAE Conference
Charting new directions: cutting-edge issues in applied epidemiology
1-2 April 2001
Hyatt Hotel, Canberra, Australian Capital Territory
Phone: +61 2 6249 2790
Fax: +61 2 6249 0740
E-mail MAE(DC): ros.hales@anu.edu.au
E-mail MAE(IH): elizabeth.lovell@anu.edu.au

The Communicable Diseases Network Australia New Zealand (CDNANZ)
Communicable Diseases Control Conference 2001
2-3 April 2001
Hyatt Hotel, Canberra, Australian Capital Territory
Phone: +61 2 6251 0675
Fax: +61 2 6251 0672
E-mail: diseases@consec.com.au
Website:
http://www.health.gov.au/pubhlth/cdi/cdconf.htm

International Society of Travel Medicine
7th Conference
27-31 May 2001
Innsbruck, Austria
Phone: +49 89 2180 3830.
Fax: +49 89 33 6038
E-mail: istm_eura@csi.com
Website: http://www.istm.org/istm_c7.html

Institute for Microbiology of Medical Faculty of Masaryk University & St Anna's Faculty Hospital
10th Tomasek Days
Annual conference of young microbiologists
6-8 June 2001
Brno, Czechia
Contact: Ondrej Zahradnicek
Phone: +420 5 4318309
Fax: +420 5 4318308
E-mail: ozahrad@med.muni.cz
Website: www.med.muni.cz/zahrad/strtomda.htm

International Conference on Exposure Assessment in Epidemiology and Practice
10-13 June 2001
Göteborg, Sweden
Phone: +46 31 335 4890
Fax: +46 41 40 9728
E-mail: x2001@ymk.gu.se
Website: http://www.ymk.gu.se/eng/x2001.htm

Association for Professionals in Infection Control and Epidemiology
Annual Meeting, Seattle, Washington
10-14 June 2001
Phone: +1 202 789 1890
Fax: +1 202 789 1899
E-mail: apicinfo@apic.org
Website: http://www.apic.org/

The Communicable Diseases Intelligence *bulletin board is provided as a service to readers. Every effort has been made to provide accurate information, but readers are advised to contact the relevant organisation for confirmation of details. Information about the availability of resources is included when space allows. Inclusion of a resource on the Bulletin Board does not imply endorsement of the resource by either the Communicable Diseases Network Australia New Zealand or the Commonwealth Department of Health and Aged Care.*

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

Overseas briefs

Pacific Public Health Surveillance Network

The Pacific Public Health Surveillance Network serves to disseminate information about communicable diseases in the Pacific region through Pacnet. Pacnet may be accessed, on registration, through the South Pacific Commission Website (http://www.spc.org.nc).

Cholera, Marshall Islands

Contributed by Tin Soe, Medical Director, Ministry of Health, Ebeye (edited)

Between 1 and 23 December 2000, in Ebeye Island, Kwajalein Atoll, 49 patients have been admitted, 64 patients seen at ER, and 30 patients treated in OPD. There were (5) deaths as of 24 December. Cholera diagnosis was confirmed by the Guam Laboratory.

Laboratories in Honolulu confirm that the specimen sent from Lae Atoll contains *Vibrio cholerae*. This is the first time cholera has been laboratory-confirmed outside of Ebeye Island. The Ministry strongly recommends that travel to and from Lae Atoll be restricted.

The cholera isolated from Ebeye patients is type Ogawa. This is the same type as in the FSM outbreak. Total mortality is now six.

Dengue in Palau, Caroline Islands, Micronesia

Source: Palau Ministry of Health News Release, 8 December 2000 (edited)

More than 350 cases of febrile illness have occurred in Palau since the beginning of September. Blood samples were sent to the World Health Organization (WHO) reference laboratory in Australia, and dengue fever has been confirmed as the cause of illness in a number of cases.

There are 4 viruses that can cause dengue. Infection with one virus type confers long-term immunity against the same virus type, but only temporary or partial immunity against other virus types. A subsequent infection with another dengue virus type may carry a higher risk of dengue haemorrhagic fever.

In the current outbreak, dengue type 1 has been identified in several cases. Initial interpretations of antibody levels (ie. haemagglutination inhibition antibody titres) suggested that dengue types 3 and 4 were causing the current outbreak. Since then, however, more sensitive tests (nucleic acid amplification and virus isolation) have identified only type 1 dengue in the samples, although not all confirmed cases have been analysed for virus type. Identification of the infecting dengue virus from antibody titres may be complicated by the occurrence of cross-reactive antibodies to antigenic determinants shared by all 4 dengue viruses.

Although no tourists have yet been identified among the suspected cases, travellers should be mindful of the outbreak. Visitors to Palau can reduce their own risk of infection through the use of DEET-based insect repellents, and are strongly encouraged to seek medical care if symptoms occur while in Palau or after they have returned to their home countries.

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.

E. coli O157:H7 outbreak, Spain

Contributed by Ana Martinez, Josep Maria Oliva, Gloria Hernandez-Pezzi, Pilar Soler. Source: Eurosurveillance Weekly Issue 1, 4 January 2001 (edited)

So far, a total of 181 cases have been reported in the largest outbreak of *Escherichia coli* O157:H7 infection yet identified in Spain. The cases are 150 schoolchildren at 4 schools in Barcelona and 31 household contacts. Cases became ill between 19 September and 5 November 2000.

Though 6 children developed haemolytic uraemic syndrome (HUS), all recovered.

The attack rates in the 4 affected schools ranged from 4 to 56 per cent.

Preliminary enquiries suggested that the vehicle of infection was sausage served by a catering company on 18 September 2000. The catering company supplied 10 schools, one factory, and a home for elderly people.

Cases arose at schools where the sausages were not heated. Inspection of the catering company identified irregularities and the company was closed down. No food samples were investigated. *E. coli* O157:H7 was isolated from 27 cases, and 8 isolates were shown to be phage type 2.

Hand, foot and mouth disease, SE Asia: enterovirus 71 identified

Contributed by Muruga Vadivale 5 January 2001. Source: Joint Press Release by Ministries of The Environment, Health, Community Development and Sports, and Education (Singapore) (edited)

The Department of Pathology, Singapore General Hospital, has identified enterovirus 71, one of the several viruses associated with Hand, Foot and Mouth Disease (HFMD), in postmortem samples taken from the 4-year-old boy from Pontian (Malaysia) who died on 3 Jan 2001.

Altogether, 8 children ranging from 3 months to 7 years old were admitted to the KK Women's and Children's Hospital on 3 January 2001. Of these, 2 were residents of Pontian who sought medical treatment here after attending the funeral of the deceased child.

Over the last 8 weeks, there was an average of 50 cases a week. In the last 2 weeks of December 2000, there were 45 and 29 cases respectively. In the first 3 days of this year, there was an average of 10 cases a day.

Multi-State outbreak of listeriosis, United States

Source: MMWR, 21 December 2000 (edited)

Since May 2000, 29 illnesses caused by a strain of *Listeria monocytogenes* (LM) have been identified in 10 States: New York (15 cases); Georgia (3); Connecticut, Ohio, and Michigan (2 each); and California, Pennsylvania, Tennessee, Utah, and Wisconsin (one each). Dates of LM isolation ranged from 17 May through 26 November with 26 (90%) infections occurring since 15 July.

When subtyped, the LM isolates from these cases were indistinguishable by pulsed-field gel electrophoresis.

Included in the report were 8 perinatal cases and 21 non-perinatal cases. Among the 21 non-perinatal cases, the median age was 65 years (range: 29-92 years); 13 (62%) were female. The 29 cases have been associated with 4 deaths and 3 miscarriages/stillbirths.

A case-control study conducted by 5 state and 2 local health departments and CDC implicated eating deli turkey meat as the probable source of infection.

On 8 December, investigators from the Food Safety and Inspection Service, USA Department of Agriculture (USDA) began investigating the implicated establishments. On 12 December, Cargill Turkey Products, Inc. (Waco, Texas) stopped shipping ready-to-eat foods and, on 14 December, voluntarily recalled processed turkey and chicken deli meat that might have been contaminated.

Africa, precautions against malaria

Source: Xinhua News Agency, 22 December 2000 (edited)

The World Health Organization (WHO) has warned holiday travellers to Africa to take all possible precautions in order to prevent malaria.

WHO has received numerous reports in recent weeks from Spain and Germany of travellers falling ill after returning from last-minute package holidays to destinations such as Senegal and Gambia. Similar reports have come from the United Kingdom, Sweden and Denmark.

In Southern Africa, WHO said, there is a special need for vigilance over the coming months as above-normal rainfall is forecast from December 2000 to March 2001, with a corresponding increase in malaria transmission over most of the region up to May 2001.

WHO has recommended weekly mefloquine prophylaxis for most African countries. Mefloquine prophylaxis should be started 2 to 3 weeks before travel. (One week before entry is usually sufficient). For travellers who failed to start prophylaxis in time, daily doxycycline offers an alternative, as it can be started the day before travel.

To cover the incubation period of the disease, both drugs should be continued during the stay and for 4 weeks after leaving the endemic area.

Moderator's comment. Whether the increased number of reports also represent an increased number of cases is not known, except that the previous dispatch from the UK indicated that the MRC in The Gambia had experienced an increased number of expatriates with malaria which was more often resistant to chloroquine than expected. There is also no doubt that malaria in South Africa towards the border with Mozambique is on the increase, but there are few actual numbers from the South African authorities. Some travellers from the Kruger National Park have reported that the border area between South Africa and Mozambique was previously sprayed with insecticides regularly, but that this activity ceased completely a few years ago.

The WHO message mentions that lariam (mefloquine) should be started 2 to 3 weeks before entry into the malaria endemic area. The standard recommendation is one week before, but it is common practice to start lariam 3 weeks before if the traveller is concerned about possible side effects. The WHO message does not mention malarone (atovaquone/proguanil) as an alternative although it is registered in the USA and Denmark. Malarone is highly efficient without the occasional neuropsychiatric adverse reactions seen under lariam use. Malarone is especially efficient against the liver stage and can therefore be used from the day before entry and for only 7 days after departure from the malarious area.

Editorial note: Malarone (atovaquone plus proguanil) is registered in Australia for treatment of *P. falciparum* malaria in those aged 3 years or older. According to reports in the Brisbane Courier Mail (28 December 2000) a novel antimalarial developed by the US military (tafenoquine) has been trialled by Australian troops serving in East Timor .

Legionnaire's disease, Paris

Source: AP Online 30 December, 2000 11 (edited)

A Paris hospital has banned showers and ordered water pipes disinfected after 4 people were diagnosed with Legionnaire's disease; all 4 caught the disease in the past month.

The ultramodern 750-bed hospital in south-western Paris has had a series of setbacks since opening its doors in July. Officials suspect that *Legionella* developed in unused sections of water pipes in the hospital, which is only partially occupied by about 250 patients. The stagnation of hot water could explain the epidemic they said.

Hepatitis C, nosocomial transmission

Source: New York Times, Associated Press Report, 20 December 2000 (edited)

A medical technician with a cut on his finger accidentally infected 5 hospital patients with hepatitis C virus in the first documented case of its kind, German researchers say. The researchers would not identify the hospital nor where it was located.

The case involving the technician is the first documented instance of hepatitis C virus being transmitted to patients by medical personnel who are not physicians [surgeons or anaesthetists] said Dr R Stefan Ross of the University of Essen. He and colleagues reported the case in Ross RS et al. New England Journal of Medicine, vol 343(25): 1851-1854. The researchers blamed a technician, whose job was to assist the anaesthetist. They said he probably passed the virus from a cut on his finger. He normally did not wear gloves, saying that they diminished the sense of touch he needed for his work.

In the German case, the technician himself apparently contracted the virus from a patient during surgery in 1998. Within 6 weeks, he helped administer anaesthesia to 5 other patients, all of whom developed hepatitis C. Genotypic analysis confirmed that the technician was the source of the virus. Ross estimated the chances of such infection at 140 for every 1 million invasive procedures. He said that patients should not be concerned about having any medical treatments because such events are very rare.

Meningitis, Angola

Contributed by M Cosgriff, 14 December 2000. Source: Panafrican News Agency (edited)

Meningitis has killed at least 70 people in Angola's central Huambo province, since January 2000; the main victims were people under age 45 years. Over the same period, several medical centres in the province treated 376 cases of the epidemic. Caala municipality, some 23 km from Huambo, the province's chief town, is the worst affected, recording about 75 per cent of the afflictions.

Moderator's comment: Angola is outside the 'meningitis belt' of sub-Saharan Africa. Further information about the laboratory characterisation of the etiological agent of this outbreak would be helpful.

Ebola suspects in fourth Ugandan town

Contributed by Monte Bawden 9 December 2000. Source: United Press International (edited)

Three people suffering from Ebola haemorrhagic fever-like symptoms have been found in Uganda's second-largest town, Jinja, for the first time, raising new fears on 8 December 2000 that the lethal disease is spreading. Tests were under way to determine whether the patients were suffering from Ebola haemorrhagic fever.

To date, the Ebola outbreak has been confined to Gulu town, 360 km north of Kampala, Masindi town 300 km north-west of Kampala, and Mbarara town, 280 km south-west of Kampala. Ebola fever has killed 156 people, including 14 health workers, in Uganda since September.

In Rwanda, Uganda's neighbour to the south-west, a 16-year-old boy died on 4 December 2000 of symptoms similar to those of Ebola fever. Blood samples from the boy would be taken to the World Health Organization laboratory in Gulu to test for Ebola virus. Uganda's neighbours Kenya and Tanzania have taken strict precautions against the Ebola outbreak, and there has been no report so far of any Ebola virus infection within their territories. The strain of Ebola virus in Uganda is similar to the one first identified in Sudan in the late 1970s.

Avian influenza virus, diagnostic kits

Contributed by M Cosgriff, 8 January 2001. Source: South China Morning Post (edited).

Following the discovery that the lethal H5N1 avian influenza virus of 1997 was the product of genome sub-unit reassortment between 3 avian influenza viruses found in quail and geese, the World Health Organization (WHO) has developed additional diagnostic kits for 3 influenza virus hemagglutinin genes (H5, H9, and H6); these are now being used internationally to monitor new isolates. Hong Kong virologists found that an early form of H5N1 influenza virus from geese exchanged genes with H9N2 and H6N1 viruses in quail and poultry fowls to create a H5N1 influenza virus more pathogenic for chickens, spreading throughout their organs. Contact with contaminated chicken wastes, or organs sold in the markets, subsequently passed the virus to humans.

Moderator's comment: In 1997, 18 cases of influenza (bird flu) in the Hong Kong Special Administrative Region (SAR) caused by a novel H5N1 (chicken) virus resulted in the deaths of 6 individuals and raised the spectre of a potentially devastating influenza pandemic. Previously it had been believed that the introduction of novel avian influenza virus genes into human influenza viruses (and the generation of new pandemic strains by sub-unit reassortment) required co-infection of an intermediate host (the pig). Slaughter of the poultry in the live bird markets of the Hong Kong SAR removed the source of infection and no further human cases of H5N1 infection have occurred.

In March 1999, however, a new pandemic threat appeared when influenza A H9N2 viruses infected 2 children in Hong Kong. These 2 virus isolates are similar to an H9N2 virus isolated from a quail in Hong Kong in late 1997. Although differing in their surface hemagglutinin and neuraminidase components, a notable feature of these H9N2 viruses is that the 6 genes encoding the internal components of the virus are similar to those of the 1997 H5N1 human and avian isolates.

This common feature emphasises the apparent propensity of avian viruses with this genetic complement to infect humans and highlights the potential for the emergence of a novel human pathogen. This is illustrated through the H9N2 virus, which appears to have provided the 'replicating' genes for the H5N1 virus and which has since been isolated in the SAR from poultry, pigs and humans, highlighting its propensity for inter-species transmission.

These events in the Hong Kong SAR have confirmed the role of avian hosts as a source of pandemic human influenza viruses and offer the prospect of improved forecasting of human pandemics in the future. These new reagents will facilitate surveillance.

West Nile fever - New York

Contributed by Marjorie P. Pollack , 11 January 2001. Source: AP Online (edited)

An 87-year-old woman who died in December was the second person in the United States last year to die after contracting West Nile virus infection. She had been hospitalised in a coma since August and died in December. An 82-year-old New Jersey man died from the virus in September 2000. A total of 14 New York and 5 New Jersey residents tested positive for the virus in 2000.

In 1999, 7 people died and 55 others were infected in the New York area during the first known appearance of the virus in the Western Hemisphere. The latest death serves as a reminder that potentially West Nile virus can cause serious illness, particularly in the elderly.

Moderator's comment: The number of human cases recorded during the year 2000 West Nile virus outbreak in the USA is now 20 with 2 deaths. However, one of the 20 cases is a woman resident in Connecticut who experienced headache only and does not meet the full clinical case definition established by the Centers for Disease Control and Prevention.

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CDI reviewers 2000

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Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereeing process. **Instructions to authors can be found in *Commun Dis Intell* 2000;24:5**.

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