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Murray Valley encephalitis virus surveillance and control initiatives in Australia

a report on behalf of the National Arbovirus Advisory Committee of the Communicable Diseases Network Australia

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Abstract

Mechanisms for monitoring Murray Valley encephalitis (MVE) virus activity include surveillance of human cases, surveillance for activity in sentinel animals, monitoring of mosquito vectors and monitoring of weather conditions. The monitoring of human cases is only one possible trigger for public health action and the additional surveillance systems are used in concert to signal the risk of human disease, often before the appearance of human cases. Mosquito vector surveillance includes mosquito trapping for speciation and enumeration of mosquitoes to monitor population sizes and relative composition. Virus isolation from mosquitoes can also be undertaken. Monitoring of weather conditions and vector surveillance determines whether there is a potential for MVE activity to occur. Virus isolation from trapped mosquitoes is necessary to define whether MVE is actually present, but is difficult to deliver in a timely fashion in some jurisdictions. Monitoring of sentinel animals indicates whether MVE transmission to vertebrates is actually occurring. Meteorological surveillance can assist in the prediction of potential MVE virus activity by signalling conditions that have been associated with outbreaks of Murray Valley encephalitis in humans in the past. Predictive models of MVE virus activity for south-eastern Australia have been developed, but due to the infrequency of outbreaks, are yet to be demonstrated as useful for the forecasting of major outbreaks. Surveillance mechanisms vary across the jurisdictions. Surveillance of human disease occurs in all States and Territories by reporting of cases to health authorities. Sentinel flocks of chickens are maintained in 4 jurisdictions (Western Australia, the Northern Territory, Victoria and New South Wales) with collaborations between Western Australia and the Northern Territory. Mosquito monitoring complements the surveillance of sentinel animals in these jurisdictions. In addition, other mosquito monitoring programs exist in other States (including South Australia and Queensland). Public health control measures may include advice to the general public and mosquito management programs to reduce the numbers of both mosquito larvae and adult vectors. Strategic plans for public health action in the event of MVE virus activity are currently developed or being developed in New South Wales, the Northern Territory, South Australia, Western Australia and Victoria. A southern tri-State agreement exists between health departments of New South Wales, Victoria and South Australia and the Commonwealth Department of Health and Aged Care. All partners have agreed to co-operate and provide assistance in predicting and combatting outbreaks of mosquito-borne disease in south-eastern Australia. The newly formed National Arbovirus Advisory Committee is a working party providing advice to the Communicable Diseases Network Australia on arbovirus surveillance and control. Recommendations for further enhancement of national surveillance for Murray Valley encephalitis are described. *Commun Dis Intell* 2001;25:33-47.

Keywords: Murray Valley encephalitis, Kunjin virus, flavivirus, arbovirus, mosquito control

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Prologue

Arboviruses (arthropod-borne viruses) of public health importance in Australia include flaviviruses and alphaviruses. Within the flavivirus group the important human pathogens include Murray Valley encephalitis (MVE), Kunjin (KUN), Japanese encephalitis (JE) and Dengue (DEN) viruses. Alphaviruses causing human disease include Ross River (RR) and Barmah Forest (BF) viruses. Other arboviruses including Sindbis (SIN), Alfuy (ALF), Edge Hill (EH), Kokobera (KOK), Gan Gan (GAN), Trubanaman (TRU) and Stratford (STR) virus cause only mild or inapparent infections.[1]

Of all the arbovirus infections, Murray Valley encephalitis causes the most severe disease. In the last 2 years MVE virus activity has increased with record levels of cases reported in Western Australia and widespread activity in the Northern Territory. In early 2001, 2 cases of Murray Valley encephalitis acquired in the Alice Springs area of the Northern Territory were reported, and a further case was detected in Mt. Isa, Queensland (see case reports in this issue of *Communicable Diseases Intelligence*). At the same time sentinel chickens in New South Wales showed seroconversions to MVE virus for the first time since the last national outbreak of Murray Valley encephalitis in 1974 (for sentinel chicken results see report in this issue of *Communicable Diseases Intelligence*).

In response to the increased MVE virus activity, the National Arbovirus Advisory Committee (NAAC), a working party of the Communicable Diseases Network Australia (CDNA) proposed that a scoping study of all current and possible surveillance mechanisms for Murray Valley encephalitis be undertaken. The collation of the information would facilitate, in the event of a national outbreak, rapid identification and co-ordination of these surveillance systems. The call for the review also reflected the perceived need to address cross-border issues that may arise during outbreaks and identify gaps in the current surveillance mechanisms. This document provides a summary of surveillance mechanisms and vectorborne disease control initiatives for MVE virus in Australia. Existing systems are described and other surveillance systems that may be utilised in the event of a widespread outbreak are discussed. Specific recommendations for the improvement of national MVE virus surveillance are proposed.

Background

Epidemiology of Murray Valley encephalitis

MVE virus is enzootic in the Kimberley region of Western Australia and the Top End of the Northern Territory. The virus is epizootic in the Pilbara and regions further south in Western Australia and the southern half of the Northern Territory. The situation in Queensland is less well understood due to the dearth of data over the past three decades. However, human cases occur sporadically throughout the State, including southern Queensland.[1] Since 1974, however, nearly all cases of arboviral encephalitis due to MVE virus have been reported from Western Australia and the Northern Territory,[2,3,4] with MVE activity and human disease occurring in most years. Virus activity occurs in the wet season, with human cases being infected between February and July.

Prior to 1974 only 1 case of encephalitis due to MVE virus had been reported from Western Australia, and none from the Northern Territory. Strong circumstantial evidence has indicated that ecological and environmental changes resulting from damming the Ord River and establishing the irrigation area in the north-east Kimberley may have provided conditions conducive to increased MVE virus activity and endemicity.[3] Any future changes to the waterways in the north of Australia may further change the ecology of the flaviviruses.

The history of severe epidemics of encephalitis in south-eastern Australia (particularly in the Murray/Darling River system) and the subsequent identification of MVE virus has been previously described.[4] These outbreaks started in December/January, peaked in February/March and declined in the cooler months. They occurred at irregular intervals, the last being in 1974 and involving approximately 58 cases, 13 of whom died.[5,6,7]

The 1974 outbreak spread to all mainland States of Australia and led to the introduction of the term 'Australian encephalitis (AE)'. The term AE has subsequently been used to refer to encephalitis due to either MVE or KUN infection,[8,9] and has led to considerable confusion. It is recommended that this term no longer be used and the terms MVE encephalitis and KUN encephalitis replace this nomenclature. If the infecting flavivirus cannot be differentiated, MVE/KUN encephalitis should be used. While surveillance mechanisms for KUN virus may be similar to that for MVE virus, the focus of this paper will be MVE virus.

Clinical aspects

It has been estimated that 1 in approximately 1,000-2,000 persons infected with MVE virus will develop severe encephalitis.[10,11] A larger proportion will develop a milder illness[12] but the vast majority remain asymptomatic. However, estimations of the case:infection ratios are not based on prospective data and are potentially inaccurate. It is likely that the rates will be higher during epidemics or in those at higher risk of severe disease.[13]

Murray Valley encephalitis is a potentially serious infection, with symptoms that include headache, neck stiffness, fever, tremor, weakness, confusion, fitting, and sometimes coma and death. Burrow and colleagues describe specific clinical features.[9] These signs may not be immediately associated with Murray Valley encephalitis by physicians when cases occur in non-enzootic areas. Persisting fevers and seizures are common in children. Cerebellar signs, brainstem features (e.g. cranial nerve palsies such as facial palsies and ophthalmoplegias) and spinal cord involvement (pseudopolio) are seen in more severe cases, often with an associated tremor. Computerised tomography scans are usually normal, but abnormalities on magnetic resonance imaging may be dramatic (e.g. thalamic lesions). Examination of the cerebral spinal fluid (CSF) usually shows a lymphocytic pleocytosis and samples should be sent to a reference laboratory for culture, serology and Polymerase Chain Reaction (PCR).

The case fatality rate for Murray Valley encephalitis is 20 per cent and approximately 40 per cent of survivors will be left with permanent neurological damage.[6,12] Young Aboriginal children in Western Australia have a particularly poor outcome.[13] The incubation period has not been well defined due to the difficulty in defining exact exposure episodes. No primary sources of data provide information regarding the

MVE virus-specific incubation period, other than from a single case report from the 1974 outbreak, with an incubation period of 28 days.[5] This exceeds the range reported for other arboviruses (5 to 15 days)[14] and at the moment the best estimate of the incubation period is 5-28 days.

MVE virus life cycle

There is a complex relationship between humans, vertebrate hosts, mosquito vectors and the environment involved in the ecology of most arboviruses. A range of factors may be involved in establishing and maintaining the MVE virus life cycle (Table 1).

MVE virus is transmitted to humans by mosquitoes and there is no direct transmission from person to person. The most common vector is the fresh water mosquito *Culex annulirostris*,[1,7] although other possible vectors have been identified.[1,11] The possibility of survival of MVE virus in arid areas via desiccation resistant *Aedes tremulus* eggs has been described.[15]

Both field and experimental infection studies have been used to investigate a number of vertebrate species as potential hosts for MVE virus. A comprehensive review of studies has been previously published.[7] Investigation of wild and domestic animals around the time of the 1974 outbreak revealed infections in domestic fowls,[16] wild birds and horses.[17] While serological field studies do confirm that particular species can be infected with MVE virus, they do not indicate what viral titres are achieved during infection and how long these infections are maintained. Both of these factors influence whether a particular vertebrate species is likely to be a major host in the MVE virus life cycle. Experimental studies have been undertaken to address these issues. Following experimental infection, wild birds, including herons and egrets, have been shown to develop viraemias of 3 to 5 days duration. Maximal titres were obtained in younger birds.[18] Studies have shown that domesticated animals such as fowl, pigs, cattle and horses may also be experimentally infected[19] but the role of these species in natural transmission cycles is not believed to be important. On the basis of laboratory and field experiments, wild birds, particularly wading water birds are thought to be important in the life cycle of MVE virus. The rufous night heron (*Nycticorax caledonicus*, also known as the nankeen night heron) is recognised as a major vertebrate host of MVE virus.[7]

Meteorological events such as rainfall, temperature and humidity also play a major role in the transmission of MVE virus.[20] Mosquito abundance is affected by the availability of aquatic breeding habitats. Other factors such as temperature, wind speed and wind direction affect their distribution and life cycle. Outbreaks of Murray Valley encephalitis may occur after unusually heavy and persistent rainfall and subsequent flooding. Abnormal rainfall may increase the numbers of mosquitoes and lead to movement of infected birds from enzootic regions to epizootic regions.[5,21] The mechanisms by which outbreaks in south-eastern Australia commence are unclear. One possibility is that MVE virus may be enzootic in south-eastern Australia in cryptic foci that are not detected by vector and vertebrate surveillance mechanisms in the intervening periods between outbreaks, but this seems to be an unlikely explanation given the extensive surveillance efforts. A more plausible explanation is that the virus is reintroduced by birds from the northern latitudes following periods of extreme rainfall and flooding. Indeed, genetic evidence demonstrates a lack of independent divergence of Australian MVE lineages, which strongly

Table 1. Factors that may affect establishment and maintenance of MVE virus life cycle

Virus	Enzootic presence or reintroduction into the environment
	Inter-epidemic survival
Vector	Density, fecundity and longevity
	Feeding patterns and feeding preferences
	Oviposition and over-wintering
	Distribution of vectors
	Natural predators of vectors
	Control mechanisms by humans
Vertebrate host	Range of potential hosts species
	Viral titre and duration of viraemia
	Host species density and breeding
	Prior exposure to the MVE virus
	Movement and migration
	Mosquito avoidance mechanisms
Environment	Climate and weather, particularly temperature, rainfall and humidity
	Physical landscape, such as presence of waterways
	Human interventions on the environment, such as irrigation, drainage of swamps etc
Human	Prior exposure to the MVE virus
	Population distribution
	Lifestyle factors
	Use of preventative measures to avoid being bitten by mosquitoes

supports the re-introduction of MVE virus rather than the presence of cryptic foci.[22]

Surveillance mechanisms

Mechanisms for monitoring MVE virus activity include surveillance of human cases, surveillance of MVE virus activity in vertebrate hosts, monitoring of mosquito vectors for abundance, virus isolation from mosquitoes and climate surveillance.[23] In some jurisdictions monitoring of human cases alone is insufficient for public health action, particularly when there are alternative surveillance mechanisms which may trigger action prior to the detection of human cases, and when the outcomes of infection can be so severe. If viewed in purely economic terms, the financial costs of these additional surveillance systems must be weighed against the cost of preventing human disease. The prevention of 1 human case with permanent neurological damage would make these systems cost effective. In the United States of America (US) it has been estimated that the community cost of a patient with permanent neurological damage is $US3 million. No equivalent costings are available in Australia, although it is likely that the value may be less than that estimated for the US.

The additional surveillance systems are used in concert to signal the risk of human disease. Monitoring sentinel animals for MVE virus activity provides information regarding whether MVE transmission to vertebrates is actually occurring. Sentinel chickens flocks maintained in a number of states and territories provide information on MVE virus activity.[24] Vector surveillance includes mosquito trapping for speciation and enumeration of mosquitoes to monitor population sizes and composition. Monitoring of weather conditions and vector surveillance determines whether there is potential for MVE activity to occur. Virus isolation from trapped mosquitoes is necessary to define whether MVE is actually present, but is difficult to deliver in a timely fashion in some jurisdictions.

The trapping of live vector collections does require the use of CDC/EVS CO_2 baited traps, which may have logistical constraints for remote regions of Australia, such as the Kimberley region of Western Australia. The development of PCR assays for detection of MVE virus in pools of mosquitoes would allow more timely reporting of the detection of virus in mosquitoes and may obviate the need for live mosquito collections. It would be advantageous if these methods could be applied to mosquitoes that have been collected in traps for up to a week. These methods remain in the developmental stage in a number of laboratories across Australia until technical problems are overcome. These problems include the storage of trapped mosquitoes, prevention of fungal growth, viral RNA degradation and the presence of PCR inhibitors in large pools of mosquitoes. Optimal pooling sizes must be determined before PCR is a cost-effective and timely replacement for virus isolation using current cell culture techniques.

Meteorological surveillance is used in the prediction of MVE virus activity by signalling conditions that have been associated with outbreaks of Murray Valley encephalitis in humans in the past. Examination of climate meteorologic information including rainfall, temperature and the Southern Oscillation (SO) may assist the prediction of risk situations. The SO is an inter-annual oscillation in tropical sea level pressure between eastern and western regions of the Pacific Ocean. The Southern Oscillation Index (SOI) is calculated from the monthly or seasonal fluctuations in the air pressure difference between Tahiti and Darwin. Positive SOI values suggest that rainfall will be above average across eastern Australia, while negative values suggests that rainfall will be below average. The SOI predicts rainfall to a lesser extent in central and western Australian states.

There are 2 models for the prediction of Murray Valley encephalitis activity for south-eastern Australia using climatic information. The Forbes model[2] utilises rainfall patterns and provides a quantitative approach to predicting outbreaks of Murray Valley encephalitis. The model relies on rainfall patterns in the preceding and current season of MVE virus activity. The model predicts MVE amplification where there has been above average rainfall in the current and preceding summers, with the underlying hypothesis that abnormal rainfall enhances breeding of both wading birds and mosquitoes. The Nicholls model[25] suggests a qualitative association between Darwin atmospheric pressure (a measure of SO) in autumn, winter and spring of the preceding season and Murray Valley encephalitis activity.

A mathematical model based on host and vector factors, has been developed[26] for the rural amplification of MVE virus in southern Australia during the 1951 and 1974 outbreaks. This model predicted the likely duration of the rural amplification phase, estimated to have commenced in October of the year prior to an outbreak. Thus it appears that seeding of the south-eastern areas of Australia in the previous year is important for the establishment of an outbreak in the following season.

Evidence supporting the use of animal, vector and climate surveillance mechanisms to predict disease in humans

The complex MVE virus life cycle means that a number of surveillance mechanisms can be utilised to predict MVE virus activity. Whether this activity heralds human disease is enhanced by drawing together of data from a number of surveillance systems and evaluating their predictive ability in light of human cases. The appropriate surveillance tools for monitoring MVE virus activity may vary from jurisdiction to jurisdiction. Factors such as whether the virus is enzootic or epizootic, the frequency of human disease, the geography and climate, the availability of laboratory facilities and other infrastructure, competing public health concerns and the availability of public health resources will affect what surveillance mechanisms are appropriate. The ability to evaluate the use of animal, vector and climate surveillance is affected by the frequency of human disease, indeed, it is difficult to evaluate the use of surveillance mechanisms in the Murray Valley region given the last human cases occurred in 1974.

Some of the best evidence for the use of non-human surveillance mechanisms for predicting MVE activity comes from Western Australia. The State has marked climate variability and encompasses enzootic and epizootic regions, as well as regions where no MVE virus activity has ever been detected. Due to the logistical difficulties associated with mosquito monitoring, sentinel chicken and climate surveillance are the key elements for predicting MVE virus activity in Western Australia. Large outbreaks (e.g. in 1993 and 2000) have been associated with abnormal weather patterns in Western Australia. Data collected over the last 10 years of the sentinel chicken program indicate that

seroconversions in sentinel chickens have preceded likely dates of exposure of the first human cases by 2 to 18 weeks in all but one situation during that period.[27]

While it has been shown that large outbreaks of Murray Valley encephalitis are associated with abnormal weather patterns in Western Australia, the use of climate surveillance to predict outbreaks in south-eastern Australia remains controversial, and should be used in conjunction with other surveillance data. The Nicholls model provides more timely prediction of the risk of Murray Valley encephalitis activity compared with the Forbes model as it does not rely on collection of data during the current season. The Forbes model, however, has proved more accurate in recent years. This model suggested there would be MVE virus activity in south-eastern Australia during the 1999/2000 season when there were a number of cases of Murray Valley encephalitis in the Alice Springs area of the Northern Territory and a single case in the north of South Australia. The Forbes model again predicted activity for the 2000/2001 season, which did occur. In comparison, the Nicholls model suggested activity was unlikely in both seasons (personal communication, S Doggett).

One of the difficulties with both the Forbes and the Nicholls models is that they were based on major outbreaks of arboviral encephalitis in south-eastern Australia, which have not occurred since 1974. Both models were developed using very small data sets and neither incorporates observed activity in sentinel animals or addresses subclinical infections. Neither scheme takes into account the impact of other factors such as the breeding and movements of vertebrate hosts and vectors, or the influence of human activities on the natural landscape, such as irrigation and land development. Similarly, public health messages regarding the risk of arbovirus infection and widespread vector management programs may have reduced the incidence of human disease despite the presence of weather conditions that have been associated with outbreaks in the past.

Laboratory testing

Testing for MVE virus in humans

Virus isolation from blood is only possible in the very early acute phase of the illness prior to the appearance of antibodies. MVE virus has only been isolated from a small number of human cases, and none since 1974. While detection of viral RNA in CSF[8] or blood[28] using PCR has a higher yield, most infections are diagnosed serologically. Due to high levels of background flavivirus infection in endemic areas, and the long-term persistence of IgM, it is important to demonstrate rising titres of IgG or to have a positive viral detection test (culture or PCR) to confirm acute infection. If confirmatory laboratory evidence is unavailable or inconclusive, then a detailed exposure and clinical assessment is required to determine the likelihood of recent infection. As there is broad cross-reactivity in antibodies to the flaviviruses, assigning a particular virus as the cause based on serology requires a test that is sufficiently specific. Diagnostic and reporting guidelines for MVE (and other

Table 2. Arbovirus research laboratories in Australia providing testing for vertebrate and vector surveillance systems for MVE virus*

Location	Laboratory	Institute	Testing provided
Western Australia	Western Australian Arbovirus Surveillance and Research Laboratory	University of Western Australia	Serological testing of vertebrate hosts Mosquito collection and identification Virus isolation from mosquitoes
Northern Territory	AL Rose Virology Laboratory	Department of Primary Industry and Fisheries	Serological testing of vertebrate hosts Mosquito collection and identification
	Medical Entomology Branch	Territory Health Services	Mosquito collection and identification
New South Wales	NSW Arbovirus Laboratory	Institute of Clinical Pathology and Medical Research, Westmead	Serological testing of vertebrate host Mosquito collection and identification Virus isolation from mosquitoes
Victoria	Victorian Institute of Animal Sciences	Department of Natural Resources and Environment	Serological testing of vertebrate hosts Mosquito collection and identification Virus isolation from mosquitoes
	Australian Animal Health Laboratory	Commonwealth Scientific and Industrial Research Organisation	Serological testing of vertebrate hosts
Queensland	Queensland Health Scientific Services	Queensland Health	Mosquito collection and identification
	Arbovirus and Emerging Diseases Laboratory	University of Queensland	Serological testing of vertebrate hosts Mosquito collection and identification Virus isolation from mosquitoes
	Tropical Public Health Unit	Queensland Health	Mosquito collection and identification
South Australia	Mosquito Research Laboratory	University of South Australia	Mosquito collection and identification

* excluding opportunistic testing

arboviral diseases) have been developed[29] and have subsequently been refined in the Public Health Laboratory Network case definitions.[30]

Vertebrates and vectors

There is a network of laboratories across Australia that provides a range of testing for MVE virus activity in vertebrates and vectors, including serological testing, mosquito identification and viral isolation from mosquitoes (Table 2). In addition, there are laboratories that provide such services, but are not currently contributing to surveillance systems. These laboratories may provide services on an *ad hoc* basis for research purposes, for example, for the opportunistic testing of domestic animals. Local councils in some jurisdictions may also undertake mosquito identification.

Public health action

Public health action is determined by assessing data from the various surveillance mechanisms. It is impossible to fully eliminate mosquito breeding, therefore, it is important to warn the general public of the risk of Murray Valley encephalitis once conditions are optimal for virus transmission. Advice on personal protection and reducing risk behaviour are the major public health messages. Such warnings can be developed specifically to target the lifestyles and literacy levels of at-risk communities. Mosquito control programs may reduce the numbers of both mosquito larvae and adult vectors in certain circumstances. However, as there is no specific treatment for Murray Valley encephalitis, prevention remains the most important strategy for averting disease.

While it has not been possible to formally evaluate their effectiveness, targeted public health campaigns, drawing on evidence from animal, vector and climate surveillance, are believed to be more effective than general warnings. Data from these additional surveillance mechanisms can be used to stimulate public awareness prior to the detection of human cases.

Surveillance mechanisms in Australia

Surveillance of human cases

State and territory notifications

MVE virus is enzootic in the Northern Territory and cases of Murray Valley encephalitis have been reported in a number of years since the 1974 outbreak.[10] In recent years, members of CDNA agreed that notifiable diseases should be reported by the jurisdiction in which the case is diagnosed, rather than the likely place of infection. Infection may be acquired as people travel through regions with MVE virus activity, but diagnosis may be undertaken elsewhere, when travellers return home or when severe cases are transferred for medical treatment. The regional location of acquisition of cases of Murray Valley encephalitis notified by the Northern Territory is shown in Table 3. A presumptive case of MVE acquired in Alice Springs was identified in 1997, but could not be confirmed due to the death of the patient.[31]

Forty-one cases of Murray Valley encephalitis acquired in Western Australia have been notified since the 1974 outbreak[2,8,12,32] (regional location, of where infections were acquired are given in Table 4). While regular activity has been confined to the Kimberley, epidemic activity extending

Table 3. Confirmed Murray Valley encephalitis cases notified by the Northern Territory, 1974-2001 to date, by regional location of acquisition

Year	Region	Cases (deaths)
1974	Katherine	1
	Barkly	2
	Alice Springs	2
1981	East Arnhem	1
1987	Darwin	1
1988	Arnhem Land	2
	Darwin - Rural	1
1991	Darwin	1(1)
	Barkly	1
1993	Katherine	5(1)
	Acquired in WA	1
2000	Darwin - Rural/Katherine	1
	Alice Springs	3
	Acquired in WA	2(1)
	Acquired in SA	1
2001	Alice Springs	2

Table 4. Confirmed Murray Valley encephalitis cases notified by Western Australia, 1974-2001 to date, by regional location of acquisition

Year	Region	Cases (deaths)
1974	Kimberley	1
1978	Kimberley	2
	Pilbara	2
1979	Kimberley	1
1981	Kimberley	3
	Pilbara	3
	Gascoyne	1
1984	Kimberley	2
1986	Kimberley	1
1989	Kimberley	1(1)
1990	Kimberley	1(1)
1991	Kimberley	2(1)
1993	Kimberley	9(4)
1997	Kimberley	1
	Gascoyne	1
1998	Kimberley	1
2000	Kimberley*	1(1)
	Pilbara	2
	Mid-west/Kimberley*	1
	Mid-west	3
	Gascoyne	1
	Murchison	1

* Diagnosed and notified nationally by the Northern Territory.

further south causes occasional outbreaks outside this region. In 2000 there was a new southerly extension of MVE virus activity with cases occurring as far south as the Mid-west region, coming within 300 km of the metropolitan area.[12]

As well as a recent case in Mt Isa in 2001, 4 cases of Murray Valley encephalitis have been reported in Queensland since the 1974 outbreak, including 2 in 1991[33] (only 1 of which was a confirmed case), one in 1994[34] and 1 in 1997 (personal communication, J Hanna). The latter case was thought to have contracted the disease in the Northern Territory, and subsequently died. Ten cases scattered throughout Queensland were recorded in the 1974 outbreak. As shown in Table 3, one case of MVE acquired in the north of South Australia was reported by the Northern Territory in 2000. South Australia, New South Wales and Victoria have not recorded a case of Murray Valley encephalitis acquired within the Murray Valley region, since 1974. No Murray Valley encephalitis cases have ever been reported from the Australian Capital Territory or Tasmania.

National surveillance of human cases

All States and Territories report arbovirus infection to the National Notifiable Disease Surveillance System (NNDSS) maintained at the Commonwealth Department of Health and Aged Care. From 1991 to 2000 flavivirus infections were classified as either 'Dengue virus' or 'Arboviruses: not elsewhere classified (NEC)'. From 1996 onwards the latter included infections with MVE, KUN, JE, KOK and STR viruses. It has not been possible to determine the number of MVE notifications at a national level, from NNDSS for these years. NNDSS is currently in the process of a major revision and from 2001 onward it will be possible to distinguish the different arboviruses at a national level.

There is some variation between the case definition for MVE infection used by State and Territory health departments. With the exception of the Northern Territory and Western Australia, all jurisdictions use the 1994 National Health and Medical Research Council (NHMRC) case definition[35] that includes serological identification of all infections with MVE virus, whether encephalitis associated or not. In the Northern Territory the case definition requires the presence of a clinically compatible illness with features of encephalitis. In Western Australia a laboratory diagnosis is supplemented with a clinically compatible illness, however, this need not be encephalitis. Therefore, in Western Australia, encephalitic and non-encephalitic clinical cases are reported, but asymptomatic cases are not. In the Northern Territory, only Murray Valley encephalitis cases are reported. All other jurisdictions report all MVE virus infections.

The Laboratory Virology and Serology Reporting Scheme (LabVISE) is an additional surveillance tool maintained by the Commonwealth Department of Health and Aged Care. LabVISE is a voluntary passive reporting scheme to which sentinel virology and serology laboratories around Australia, contribute. MVE virus is one of the infectious agents reported by this system, however, the number of reports may overestimate the true number of cases of Murray Valley encephalitis as the total number will represent both encephalitic and non-encephalitic infections. State and Territory notifications may not truly represent the location of disease acquisition and duplicates may also occur, due to cross border testing and the transfer of patients for interstate clinical management. IgM positive cases may also be automatically notified even if they have not been shown to be recent infections.

Animal, vector and climate surveillance in States and Territories

Sentinel chicken surveillance programs are active in 4 jurisdictions; Western Australia, the Northern Territory, New South Wales and Victoria and mosquito monitoring complements the surveillance of sentinel animals in these jurisdictions. In addition, other mosquito monitoring programs exist in South Australia and Queensland. No surveillance mechanisms for monitoring MVE virus activity in vectors or vertebrate hosts are operational in the Australian Capital Territory or Tasmania.

New South Wales

Surveillance mechanisms in New South Wales include mosquito-monitoring, virus isolation from mosquitoes and sentinel chicken surveillance. The NSW Department of Health (NSW Health) co-ordinates the New South Wales Arbovirus Surveillance and Vector Monitoring Program. Laboratory work for this program is currently contracted to the Institute of Clinical Pathology and Medical Research (ICPMR), Westmead. Comprehensive reporting from the program is available on the Internet at:
http://www.arbovirus.health.nsw.gov.au.

The New South Wales arbovirus surveillance program has sentinel chickens located across inland areas of the State (Figure 1). Flocks of 15 chickens are located at 12 sites. The flocks are bled at weekly intervals and tested for antibodies to flaviviruses, including MVE and KUN viruses. All chickens are replaced annually in October and additional birds may be included mid season if a large number seroconvert. The program also involves mosquito collection at locations throughout the State (Figure 1). The trapping program operates from mid-spring to mid-autumn (November to April) to cover the period for natural activity and transmission of arboviruses. Mosquitoes are collected weekly for vector identification and quantitation and are processed for isolation of MVE, KUN, EH, ALF, STR, KOK, SIN, RR and BF viruses.

Data on the Southern Oscillation Index, rainfall and temperature are obtained from the Bureau of Meteorology Website (http://www.bom.gov.au). These data are used by the members of the monitoring program to predict mosquito-breeding capabilities. Climatic data are used to predict potential Murray Valley encephalitis outbreaks using both the Forbes and Nicholls models.

Northern Territory

Surveillance for MVE virus activity in the Northern Territory consists of sentinel surveillance of virus antibodies in sentinel chickens and virus isolation from mosquitoes. Surveillance of sentinel chicken flocks for flavivirus activity is a combined program between the Northern Territory Department of Primary Industry and Fisheries (DPI&F), Territory Health Services (THS), the University of Western Australia (UWA) and volunteers. The program is designed to detect flavivirus activity (including the enzootic arboviruses MVE and KUN viruses and exotic arboviruses such as JE), in the Northern Territory. Sentinel chicken flocks are maintained at 9 sites (Figure 2). Flocks are usually bled once a month and the samples are sent to the Arbovirus Surveillance and Research Laboratory, UWA, for specific testing for MVE and KUN viruses. When the majority of chickens in a flock

Figure 1. Monitoring locations in New South Wales

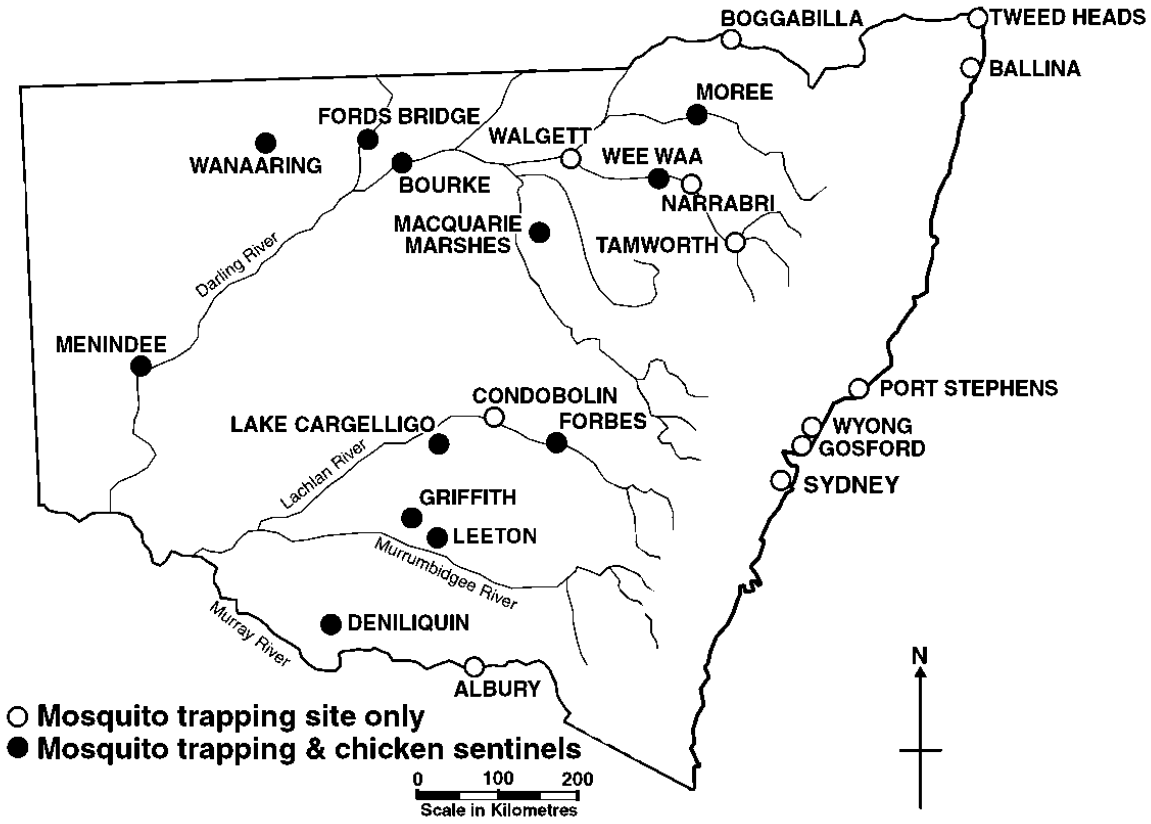
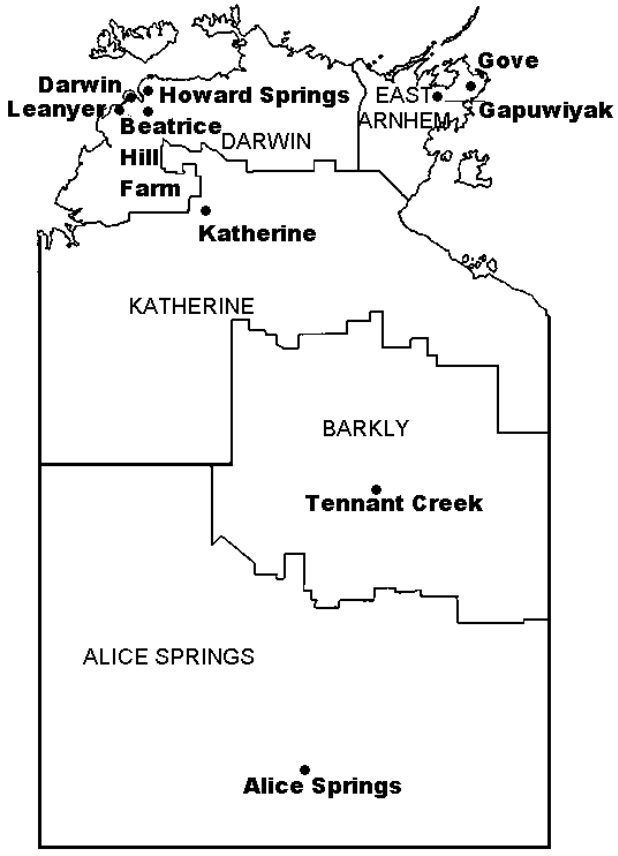


Figure 2. Sentinel chicken flocks in the Northern Territory



seroconvert in a season, the flock is replaced. As the majority seroconvert each year, replacement can occur mid-season. Most flocks are replaced annually and all are replaced within 2 years.

The Northern Territory Mosquito Borne Disease Control Program assists regional authorities with mosquito monitoring and provides advice and funding for direct mosquito control for some of the major towns in the Northern Territory (Darwin, Jabiru, Nhulunbuy on the Gove Peninsula, Katherine, Tennant Creek, Alice Springs, and at Alyangula on Groote Eylandt). Monitoring and control operations are usually carried out by town councils or other local authorities through local environmental health officers, with the identification of all mosquitoes carried out by the Medical Entomology Branch (MEB) of Territory Health Services.

The routine mosquito-monitoring program in Darwin consists of 18 trapping sites throughout the Darwin urban area. Although now discontinued, until recently mosquitoes were routinely collected for virus isolation at Middle Point, near Beatrice Hill. The Middle Point virus isolation collections were part of a combined project with DPI&F and THS. Mosquito trapping near sentinel chicken flocks aims to correlate antibodies and virus isolates in the animals with vector activity. Collection of mosquitoes for virus isolation is currently on an *ad hoc* basis, during actual outbreaks or periods of potential disease activity.

Four additional routine trapping sites are located in Jabiru, 5 in Gove, 3 in Tennant Creek, 4 in Katherine and 3 sites at Alyangula on Groote Eylandt. The mosquito-monitoring program in Alice Springs is a co-operative program between the Alice Springs Town Council and the MEB in Darwin. There are 6 regular mosquito-monitoring sites located in

Alice Springs. Environmental health officers from the Alice Springs Town Council collect mosquitoes from these sites on a weekly basis.

Information from the Bureau of Meteorology is used in conjunction with animal and vector surveillance. Monthly weather reviews are obtained from the Bureau of Meteorology and rainfall patterns and daily rainfall records are used to predict mosquito activity.

Queensland

Queensland has no State-wide surveillance system for monitoring MVE virus activity in vertebrate hosts or vectors and does not maintain sentinel chicken flocks. Mosquito-monitoring is performed by local councils. While there are no regular isolation programs, virus isolations from mosquitoes or animals have been carried out by the University of Queensland, the Tropical Public Health Unit Network (TPHUN) within Queensland Health, the Queensland Health Scientific Services and the Queensland Institute of Medical Research. These research programs are funded in part by Queensland Health and the NHMRC.

Three State health department entomologists are located in Queensland, one in Brisbane and two in Cairns at the Tropical Public Health Unit. Staff from the TPHUN in Cairns and Townsville perform reactive monitoring on demand. Extensive mosquito trapping for monitoring mosquito abundance and arboviral isolation was carried out in a number of sites in the Mt. Isa region in February to March 2001 in response to the human case of Murray Valley encephalitis detected in February, 2001. Research based activities are also carried out by the TPHUN. Mosquito trapping is carried out in the Torres Strait, the Gulf of Carpenteria and Western Cape York by the TPHUN, in collaboration with the University of Queensland. While the trapping is primarily to monitor potential for JE activity and isolate the virus, the program also investigates the presence of novel vectors in the region.

South Australia

Arbovirus surveillance in South Australia is co-ordinated by the Department of Human Services, South Australia, and consists of mosquito trapping in the Murray Riverland area and virus isolation when required. South Australia local councils perform mosquito surveillance and control in areas other than the Torrens Island environs. Several councils contract mosquito surveillance to the Mosquito Research Laboratory at the University of South Australia. Seasonal monitoring of the mosquito population is undertaken along the Murray River. Live collections of mosquitoes for virus isolation are sampled in response to high vector numbers and are sent to Victoria for virus isolation. This has occurred on several occasions in 2001.

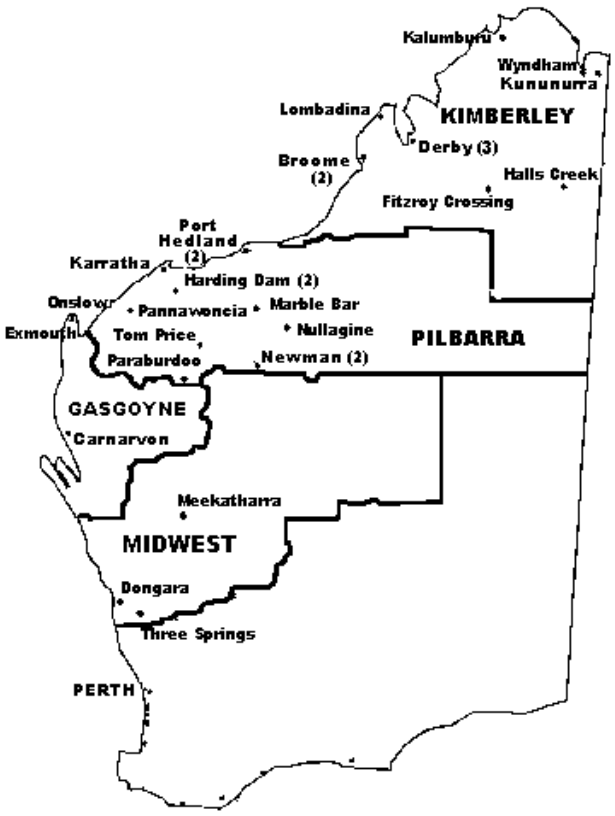
Western Australia

The Western Australia Arbovirus Surveillance and Research Laboratory, UWA, is funded by the Health Department of Western Australia (HDWA) to co-ordinate a sentinel chicken program and mosquito surveillance in Western Australia, as well as providing confirmatory serological testing for other sentinel chicken programs in Australia. Sentinel chicken sites may vary from year to year, depending on virus activity. In 2001, sentinel chicken flocks were maintained at 11 sites in the Kimberley, 13 sites in the Pilbara, at 2 sites in the Gascoyne and at 3 sites in the Mid-west (Figure 3). Twelve birds are maintained at each site. The flocks are bled fortnightly from November to May and monthly at other times during the year. Samples are tested for MVE virus specific antibody activity using an epitope-blocking enzyme immunoassay. Seropositive chickens are replaced once half the chickens in the flock have seroconverted. In addition, all flocks are replaced annually during the dry season.

In the more remote regions of Western Australia it is not logistically or financially feasible to undertake routine mosquito surveillance, and experience has shown that surveillance using sentinel chickens provides adequate warning of increases in the activity of MVE virus. An annual program of mosquito trapping is undertaken towards the end of the wet season when MVE virus activity is usually highest. Field collections are undertaken at all major towns in the Kimberley as well as at some Aboriginal communities. The mosquitoes are collected over a 3 to 4 week period and then subsequently tested in the Arbovirus Surveillance and Research Laboratory, a process that takes several months to complete. While not prospective, this program provides important data on size and species composition of mosquito populations, vector species and virus infection rates. It also assists in matching specific meteorological conditions to breeding and infection rates of vector mosquito species.

Opportunistic field collections are carried out in response to seroconversions in sentinel chickens and extreme weather events in a number of other areas of the State. The University of Western Australia and the HDWA also undertake field surveillance for incursion of exotic vector mosquitoes and viruses, including surveillance at specific new or proposed developments such as dams, irrigation projects or mine sites. Local councils may also undertake

Figure 3. Sentinel chicken flocks in Western Australia



mosquito trapping for species identification, enumeration and evaluation of their mosquito management programs.

Climate surveillance also provides vital information for the prediction of arbovirus activity using meteorological data obtained weekly from the Bureau of Meteorology Website. Graphs of weather patterns in all towns from the Kimberley to Esperance are produced and used to predict arbovirus activity. The SOI is used to predict rainfall, although this measure is not as accurate at predicting rainfall in Western Australia as it is in the south-eastern regions of Australia. Therefore, modelling of Murray Valley encephalitis activity using the Forbes and Nicholls models is not as reliable in Western Australia.

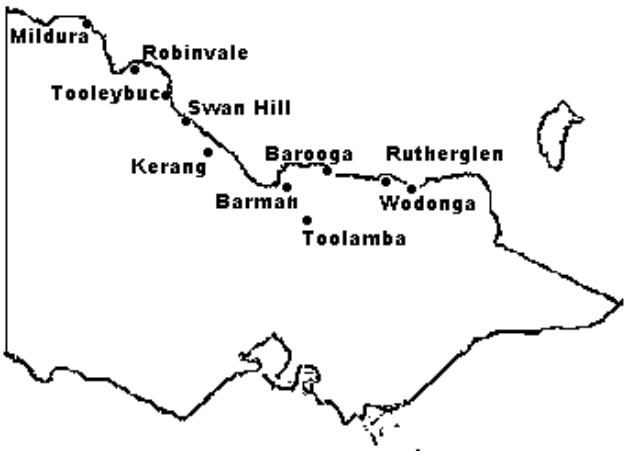
Victoria

The Department of Human Services (DHS) contract the Victorian Institute of Animal Science (VIAS) to conduct sentinel chicken surveillance from November to April. Chicken flocks of 20 birds are located at 10 locations (Figure 4). Flocks are bled weekly from mid-October to April, although surveillance may be extended in periods of MVE virus activity. Flocks are replaced annually. Blood samples are tested for flaviviruses by enzyme linked immunosorbent assay (ELISA). Cross-reactivity with members of the flavivirus family is investigated with further tests to identify the infecting virus.

Six municipal councils in Victoria, including Greater Shepparton, Mildura Rural, Moira, Swan Hill Rural, Wellington and Wodonga Shire Councils, undertake mosquito surveillance. Four traps are placed in each area and mosquitoes are collected weekly and sent live to the VIAS for identification, enumeration and virus isolation.

Adult mosquito abundance and distribution are assessed in combination with climatic information such as temperature, wind direction and wind speed. The Victorian Arbovirus Task Force examines the risk of outbreaks of Murray Valley encephalitis using meteorological surveillance data such as SOI and rainfall deciles using both the Forbes and Nicholls models.

Figure 4. Sentinel chicken flocks in Victoria



Other surveillance mechanisms

Further surveillance mechanisms may include monitoring of other flaviviruses in animals, opportunistic testing of domestic and non-domestic animals, seroprevalence studies in humans and vector monitoring.

Surveillance mechanisms for other flaviviruses in animals

Currently there are programs in operation which monitor other arbovirus activity, including human flaviviruses such as JE, or arboviruses of importance to the livestock industry, such as bluetongue. These systems have the potential to provide additional information about the activity of MVE virus or other flaviviruses. Collections of sera from these programs could be tested for MVE virus activity, although the value of testing of other sentinel animals for MVE virus activity is yet to be fully determined. Both the Australian Animal Health Laboratory (AAHL) and the State veterinary laboratories are involved in surveillance programs.

The National Arbovirus Monitoring Program

The National Arbovirus Monitoring Program deals primarily with bluetongue viruses but also with bovine ephemeral fever and akabane viruses. The program involves approximately 70 sentinel cattle herds of 10 to 15 animals each, distributed around Australia. Sentinel cattle are monitored serologically and by virus isolation. Insect trapping for *Culicoides,* the major vector of bluetongue, is conducted in conjunction with the sentinel herds. The Berrimah Veterinary Laboratories in Darwin undertake virus isolation from weekly collections from sentinel cattle at Beatrice Hill Farm in the Northern Territory.

The Northern Australian Quarantine Strategy

The Commonwealth Government through the Northern Australian Quarantine Strategy (NAQS) of the Australian Quarantine Inspection Service (AQIS) runs a monitoring program for JE. At the start of each wet season, groups of young sero-negative pigs are placed at strategic sites in the Torres Strait Islands, Cape York Peninsula and in the Northern Territory. Animals from Queensland sites are tested by AAHL and Queensland Health Scientific Services. AAHL screens these sentinel pig sera with a competitive ELISA for JE, which detects cross reactions with endemic Australian flaviviruses. Reactive sera are then tested by a plaque reduction neutralisation test for neutralising antibodies to JE, MVE and KUN viruses. The sentinel pigs surveillance system operating in the Northern Territory is co-ordinated by the DPI&F. Pigs are maintained at each site at Berrimah and Beatrice Hill on the Adelaide River flood plain. The animals are bled monthly and tested for broad flavivirus activity by the AL Rose Virology Laboratory at the DPI&F. Positive test results are further investigated with serum neutralisation tests specific for MVE, KUN and JE viruses.

Surveys of feral animals including pigs, cattle, donkeys, goats, and deer are undertaken as part of NAQS. Domestic animals are surveyed in the Torres Strait and in the Northern Peninsular area. A NAQS team periodically samples wild migratory birds in the Torres Strait. The Arbovirus Surveillance and Research Laboratory at UWA tests sera provided by NAQS from cattle herds in the Kimberley for antibody to MVE, KUN and JE viruses. Sera from a wide range of other vertebrates have been tested for flavivirus antibodies since the first detection of JE virus in the Torres Strait in 1995.

Opportunistic testing of animals

Serological surveys provide evidence of past or recent infection with arboviruses in domestic and non-domestic animals, but their use as an early warning system is limited. Survey data are unreliable indicators of recent virus activity, unless undertaken in young animals, and do not take into account issues such as animal migration. They do, however, provide data for hypothesis generation regarding the range of vertebrate hosts of the virus and define regions of virus activity.

Non-domestic animals

Regular testing of sera from birds and animals from both enzootic and epizootic regions of MVE virus activity is undertaken in Western Australia. Serological reactivity to flaviviruses has been investigated in western grey kangaroos opportunistically bled during culling exercises in South Australia (personal communication, M Kokkinn). In January 2001, NSW Health and the Commonwealth Scientific and Industrial Research Organisation (CSIRO) undertook opportunistic sampling of a breeding colony of rufous (nankeen) night herons along the Murray River. In Victoria, wildlife sera collected following culls of kangaroos and possums, are opportunistically tested for MVE virus.

Domestic animals

Opportunistic testing of domestic chickens in regions where sentinel chickens are not located has been used in Western Australia for many years to provide additional information about activity of MVE virus in epizootic regions, particularly to define the spread of MVE virus activity. It was used very successfully, for example, in 2000 to demonstrate the southern and eastern limits of activity of MVE virus during a major outbreak in humans. Results were subsequently used to determine the most appropriate location for new flocks of sentinel chickens in the Mid-west, Murchison and Goldfields regions.

Testing of domestic chickens was recently undertaken in New South Wales in response to sero-conversions in sentinel flocks, targeting areas with large human populations surrounding the sites of sentinel chicken activity. Areas further north were also included to assess whether the virus was moving in a North-South direction. The NSW Department of Agriculture recently tested a range of domestic animals to determine the spatial distribution of MVE virus activity and to compare the usefulness of dogs, cattle, horses, and domestic chickens as sentinels for MVE virus, in and away from major human population centres. In South Australia, DHS co-ordinated the testing of domestic chicken flocks at Paringa in February 2001 in response to recent seroconversions in sentinel chickens in New South Wales and Alice Springs. There are currently plans for testing domestic cattle in the Yunta area of South Australia to further investigate MVE virus activity. Opportunistic testing of cattle has been undertaken in Victoria in response to the recent MVE virus seroconversions in sentinel chickens.

The evidence that arboviruses affecting human health cause disease in animals is controversial. Animals (particularly horses) presenting with symptoms of encephalitis (ataxia) or arthritis (stiffness or swollen joints) may be tested for RR, KUN and MVE viruses. Methods for diagnosing recent arbovirus infection in animals have only lately become available and are not fully validated. Rising KUN IgG titres and RR IgM reactivity have been occasionally demonstrated in animals showing signs of disease, where more common causes of disease have been excluded (personal communication, P Ellis). Further research is required to confirm whether these are coincidental infections or the causative agents. No surveillance mechanisms are active to monitor these diagnoses. The National Animal Health Information System is an analogous system to NNDSS, but does not currently record animal infections with arboviruses that affect human health.

Seroprevalence studies in humans

Seroprevalence studies in humans are useful for determining who is susceptible within a given community and provide information on the epidemiology of MVE virus. When undertaken following an outbreak, it is possible to determine the ratio of symptomatic to subclinical cases. As with studies in animals, seroprevalence studies in humans are not useful for early warning of MVE virus activity. Some of the early seroprevalence studies investigating MVE virus infections in humans[36,37,38,39] are difficult to interpret due to cross-reactions of haemagglutination-inhibition antibodies with other flaviviruses. Studies monitoring the sero-prevalence of antibodies to MVE virus using more specific ELISA have been reported.[9,40,41] These investigations provide an indication of the level and patterns of MVE virus exposure within a community.

Vector monitoring by AQIS

A vector-monitoring program is run by AQIS for the surveillance of exotic mosquito species. Surveillance is conducted at each international airport in Australia (currently 16 locations) and each seaport (currently 47 locations) within a 400 metre radius of the port. The vector monitoring program is supported by the AQIS First Port Airport Disinsection Program and the First Port Seaport Mosquito Control Program with the aim of maintaining an exotic vector free status around airports and seaports. While the focus of this work is not MVE virus surveillance, endemic vector species are trapped frequently. In the event of an outbreak this surveillance mechanism could provide details on vector abundance at the various sites.

Geographical Information Systems

Geographical Information Systems (GIS) have the potential for surveillance of mosquito vector habitats. Dale and colleagues have published a non-technical review of the use of GIS for this purpose.[42] Further investigations may be required to assess the utility of this method of surveillance of vector habitats in relation to MVE virus activity. GIS can overlay data from multiple sources and could be used to map the distribution of human cases, virus activity, climatic and environmental information.

Control mechanisms

Mosquito management

Local government is the main agent of mosquito management in most jurisdictions, including New South Wales, the Northern Territory, Queensland, South Australia, Western Australia and Victoria. Effective mosquito management incorporates a range of practices, with emphasis on an integrated approach, focussing on source reduction, appropriate town planning and other preventative measures as well as vector management using chemicals.[43]

Management programs vary according to regional requirements, as not all procedures are appropriate in all situations. The practicality, logistics and cost of such measures in enzootic areas needs careful consideration. Mosquito control is not feasible in many remote areas of northern Western Australia, Queensland and the Northern Territory where there are large and widespread natural wetlands. Instead, local governments tend to carry out community based procedures. While not applicable in all situations, both larviciding and adulticiding can be used to decrease vector numbers in or near residential areas. Aerial larvicide applications are used in some areas (e.g. in the Northern Territory) when surveillance programs determine that health-driven mosquito management is required. Adulticiding using fogging may be used if an outbreak is indicated, but this measure only temporarily decreases the number of mosquitoes. As the use of adulticiding remains controversial, its use can be restricted to barrier fogging of residential areas in times of very high vector abundance or disease risk and to protect communities where application of larvicides is logistically impossible. There is an emphasis on the use of environmentally acceptable chemicals for mosquito management.

Changes in agricultural practice (e.g. clearing vegetation in waterways and avoiding excessive irrigation), may also affect vector numbers. Land management to control breeding sites through engineering and drainage has been instituted. In addition to monitoring natural mosquito-breeding sites, local councils liaise with town planners to monitor and prevent mosquito breeding in waste-water treatment and disposal facilities, public works and land development projects such as residential, aquaculture, industrial and mining developments.

State and Territory health authorities assist regional authorities with mosquito monitoring. They also:

- provide funding, technical expertise, notification data, educational materials and training programs for local government personnel and other public and private parties who develop and operate mosquito management systems;
- provide funding for the establishment of new mosquito management programs;
- provide emergency mosquito management in the advent of an arbovirus disease outbreak or when there is a risk of disease transmission to humans;
- collaborate with industry towards the development of new mosquito management products and methods; and
- provide research funding for studies on prevention and management of arboviral disease.

Media campaigns

Most jurisdictions have developed educational material on mosquito avoidance and arbovirus disease. Media alerts are distributed in New South Wales, the Northern Territory, Queensland, South Australia and Victoria in response to MVE virus activity. The public is made aware of the health risks associated with mosquito bites and that mosquito bites and domestic breeding sites can be avoided or reduced. Self-protection mechanisms include the use of screens, knockdown aerosol sprays, residual insecticides and mosquito repellents. Information for the public is also available on ways to prevent the contamination of ground pools with organic matter generated from overflowing septic tanks or other waste-water. Human movement into areas of high breeding, for work or recreational purposes increases risk. The public is also advised of measures to avoid being bitten, for example, avoiding fishing at dusk and dawn, and avoiding camping in risk areas during months when the virus is likely to be present. Public access to high-risk areas may be temporarily closed.

Some jurisdictions have developed additional campaigns. As a result of recent activity, NSW Health is developing an emergency management plan for MVE that includes media releases and fact sheets for General Practitioners and the general public. In the Northern Territory a 'mozzie sickness alert' poster is distributed to communities, the Northern Territory tourist bureau, and is displayed in roadhouses. A Murray Valley encephalitis pamphlet is sent to the Northern Territory Tourism association for distribution to the general public. Annual media campaigns including mosquito and disease awareness advertisements are conducted by the THS through the MEB. In South Australia media releases from DHS regarding the risk of vector borne disease are sent out to the public at specific times of the year, such as prior to the Easter and Christmas holidays. In Western Australia a 'beat the bite' campaign is currently being developed to promote education regarding mosquito bites, particularly to school aged children. In addition, a culturally appropriate pictorial health alert has been developed by the UWA and the Kimberley Public Health Unit. This is issued to Aboriginal communities in the Kimberley, Gascoyne and Pilbara regions in association with the wider HDWA public warnings. These have been made available to other states in 2001. Mosquito awareness campaigns have been undertaken in primary schools in Western Australia and Victoria. In response to MVE activity in western Queensland in 2001, the TPHUN used newspaper bulletins and a poster to warn the public of risk and how to protect themselves from mosquitoes.

State-based strategic plans for MVE virus activity

Since 1990 a southern tri-State agreement has existed between the Health Departments of New South Wales, Victoria and South Australia and the Commonwealth Department of Health and Aged Care. All partners have agreed to co-operate and provide assistance in predicting and combatting outbreaks of mosquito-borne disease in south-eastern Australia. In addition, several jurisdictions have developed specific state-based strategic plans.

New South Wales

The New South Wales Arbovirus Disease Control Advisory Group provides advice on arbovirus disease issues and makes recommendations pertaining to surveillance and management activities to NSW Health. Membership of this group includes representatives from NSW Health, the New South Wales Department of Agriculture, local government, the Australian Institute of Environmental Health, the New South Wales Arbovirus Surveillance Program, infectious disease and virology experts and medical entomologists. The committee is chaired by the Director of Health Protection of NSW Health. A strategic plan for arbovirus disease control is being developed[44] and is available at: http://www.health.nsw.gov.au/health-public-affairs/greenpaper/index.html.

Through the New South Wales Arbovirus Surveillance Website (www.arbovirus.health.nsw.gov.au), the Department of Medical Entomology, ICPMR, has provided a comprehensive and readily available resource to the

general public on mosquitoes, mosquito management and mosquito-borne diseases.

Northern Territory

Contingency plans for mosquito control have been developed in the Northern Territory. Due to the vast areas of land affected by water during the wet season, it is impractical to control vectors over large areas or around many of the smaller towns and communities. Strategic plans to control mosquitoes during mosquito-borne disease outbreaks are restricted to high priority sites such as sewerage treatment facilities in smaller communities and swamps adjacent to large urban areas in Darwin and other major towns. Public awareness campaigns are important features in these strategies.

In the event of a mosquito-borne epidemic in Darwin, the Mosquito Control Advisory Committee meets and discusses control and public awareness measures. This committee consists of representatives of the Communicable Diseases Branch, the MEB, the Darwin City Council, a general practitioner and members representing the public and other interested groups. The contingency plan includes information on the organisation, cost and initiation of contingency measures. Additionally, a counter-disaster sub-plan provides further details regarding the control of mosquito vectors in the event of natural disasters, which may be associated with vector borne disease. There is also a Zoonosis Committee chaired by the Centre for Disease Control, Darwin, with representatives from MEB, DPI&F, NAQS, the Royal Darwin Hospital (laboratory and clinical services) and the Parks and Wildlife Commission.

South Australia

The Department of Human Services has convened a special working party to develop a strategic plan for mosquito control. This document has been drafted and is currently being distributed to key stakeholders (particularly local government bodies) for comment.

Western Australia

The State Arbovirus Control Committee (which includes representatives from HDWA, UWA, PathCentre, the Australian Defence Force, Agriculture WA and other national and international experts as required) has developed a contingency plan for Murray Valley encephalitis in Western Australia. The committee developed protocols to reduce exposure of humans to arboviral disease, to be implemented when surveillance systems provide an indication of activity of MVE virus. The protocols include the surveillance systems themselves, a notification procedure, the timing, severity and area to be covered by public warnings and control measures to be implemented. Control measures involve public education at several levels, source reduction and chemical control.

Victoria

A contingency plan for control of arbovirus disease has been developed by the Victorian Arbovirus Task Force.[45] The task force consists of representatives from DHS, the Department of Natural Resources and Environment, the Victorian Infectious Diseases Reference Laboratory and local government. The contingency plan details notification procedures for human cases, mosquito management procedures, surveillance in animals and a response strategy. The latter is based on prediction of epidemic years, based on both the Forbes and the Nicholls models. The plan is a step-by-step guide for personnel involved in an outbreak of arbo-encephalitis as well as detailing procedures used to predict MVE virus activity in the region.

National Strategies

The National Arbovirus Advisory Committee

The National Arbovirus Advisory Committee includes representatives from the Commonwealth Department of Health and Aged Care, State and Territory health departments, CSIRO, AQIS and academia. Laboratories involved with the diagnosis of both human and animal disease, epidemiologists, clinicians and entomologists are also represented on the committee. The NAAC is funded by the Commonwealth Department of Health and Aged Care, which also provides the secretariat. The NAAC is to report and make recommendations to the Communicable Diseases Network Australia on arbovirus surveillance and control.

Conclusions

This scoping exercise has identified a number of surveillance activities operating within Australia that provide information on MVE virus activity and drive public health action. The collation of information regarding these surveillance systems represents the first step in the process of building on our current strengths by recognising opportunities for collaboration and identifying gaps in the current approach. Further steps include addressing these gaps, strengthening old and building new collaborations and developing national approaches. While Murray Valley encephalitis is a relatively rare disease, its increasing incidence and the severity of the condition means we must be pro-active in our approach to ensure we have timely and effective mechanisms for detecting virus activity and delivering warning of this risk to the general public.

Strengthening national surveillance of MVE virus activity is a first step in ensuring that Australia is prepared to rapidly detect, contain or mitigate new or emergent arboviral diseases. The 1999 outbreak of encephalitis associated with West Nile virus in New York City[46] provides a number of valuable lessons regarding the management of arbovirus disease outbreaks, including the need to enhance awareness and training of clinicians, build public health resources and expertise, strengthen laboratory capacity, and improve communication between human and animal health authorities. The very nature of the life cycle of MVE virus and other arbovirus diseases requires that our prevention strategies are built on strong inter-sectorial communication between all stakeholders who have access to timely information on the complex ecology of arboviruses. The use of Web-based information and surveillance systems, including Geographical Information Systems, the further development of predictive models, and the development of comprehensive response plans will assist our ability to assess and manage the risk of important diseases.

Recommendations

Following the review of national surveillance and control mechanisms for Murray Valley encephalitis, the following recommendations can be made.

1. Evaluation and possible expansion of current sentinel animal surveillance mechanisms

This review of national surveillance and control mechanisms for Murray Valley encephalitis has allowed the identification of gaps in our current systems with the outlook to close these gaps in the future. It was not the aim of the current exercise to formally evaluate the existing surveillance mechanisms. All schemes may benefit from a formal appraisal, which may be carried out using the framework for evaluations published by the Centers for Disease Control, Atlanta.[47] Current sentinel animal systems for surveillance of MVE virus activity do not cover all areas where cases of Murray Valley encephalitis have been detected. National surveillance of MVE virus activity could be enhanced by development of sentinel chicken programs in these areas if, after review, these are deemed necessary.

2. Development of national reporting of animal and vector surveillance data

The development of a system for collation of national animal and vector surveillance data would ensure timely reporting and allow cross border comparisons. Development of a Website may represent a feasible option for such a system. This system should be co-ordinated and developed in consultation with key stakeholders, including a range of Commonwealth bodies, State and Territory health departments, virologists, clinicians, epidemiologists, entomologists, veterinarians and other animal health specialists.

3. Development of national human case reporting

Limited data on cases of Murray Valley encephalitis are currently collected on a national basis. While the development of the NNDSS will provide additional data, an enhanced data set could be developed for Murray Valley encephalitis with the flexibility to include other arboviruses. Nationally consistent case definitions, data fields and reporting procedures are important components of standardised case reporting. The surveillance system for human cases could be linked to the animal and vector surveillance system. Issues of confidentiality and security need to be addressed.

4. Establishment of a national strategic approach for Murray Valley encephalitis disease management and control

While some jurisdictions have developed strategic plans for outbreaks of Murray Valley encephalitis at the State and Territory level, it would be beneficial for a national body to develop a framework document providing guidance on the essential elements of response plans and identify multi-jurisdictional issues. Such strategic approaches could encompass all aspects of disease surveillance management and control and would consolidate and establish further inter-sectorial communication between key stakeholders. Strategies should address the issue of new or emerging arboviral disease.

5. Development of laboratory capacity and building public health resources

There is a perceived need to have a better quality assurance programme, and particularly a need to standardise reagents for MVE virus diagnosis. This should also extend to a comparison of the efficacy, sensitivity and specificity of various tests in use by different laboratories. Development of PCR based assays for the detection of MVE virus in pools of mosquitoes would provide more timely and cost effective surveillance, particularly if the methods can be developed to detect viruses in mosquitoes without the need for live collections.

Good communication between all stakeholders is essential. Systems to enhance clinicians' awareness of the clinical features of Murray Valley encephalitis should be explored, and could be incorporated into messages regarding the identification of other emerging arbovirus diseases. Methods for summarising and facilitating rapid and accurate communication of information to those who need to know should be explored. Such mechanisms may include disease modelling, GIS, electronic data transmission and Web-based reporting.

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Murray Valley encephalitis in Mt Isa, north Queensland

Susan Hills, on behalf of the Tropical Public Health Unit Network, Queensland Health

Case history

On 15 February 2001 a 3-year-old Caucasian boy was admitted to Mt Isa Base Hospital with a history of an acute febrile illness of about 2 days' duration and 2 febrile convulsions. He remained febrile over the following 2 days, had further brief generalised tonic-clonic seizures and went on to develop a left hemiparesis, agitation, confusion and subsequently coma.

His cerebrospinal fluid (CSF) showed a white cell count of 200 x 10^6/L (39% mononuclear cells). A full blood count and serum electrolytes were unremarkable. A provisional diagnosis of acute encephalitis was made, and he was commenced on broad-spectrum antibiotics and acyclovir. The child was ventilated in an Intensive Care Unit for just over a week.

Tests were carried out to determine the aetiology of the encephalitis, including serological and other tests for the encephalitic flaviviruses. Polymerase chain reaction tests on CSF were negative and attempts at virus isolation were unsuccessful. However, serum collected on day 4 (and again on days 6 and 13) was strongly reactive by enzyme immunoassay for IgM to the encephalitic flavivirus group, but not reactive to any other flaviviruses. Haemagglutination inhibition (HAI) tests on sera showed a rise in titre to Murray Valley encephalitis (MVE) virus from non-reactive to re-active at a dilution of 1:20, between two specimens collected on days 4 and 6 of illness. The reactive sample from day 6 was analysed by ultracentrifugation, fractionation and HAI and reacted specifically and strongly against MVE. The serological results were therefore strongly suggestive of a recent infection with MVE virus.

Two months after the onset he has persisting major neurological sequelae; he remains semi-comatose with a spastic quadriplegia.

Public health investigations and actions

A history was taken from the child's parents to assess possible sites of exposure to infected mosquitoes. Environmental investigations and mosquito trapping were also undertaken.

During the presumed exposure period, the child had been resident in Mt Isa. On two occasions the child and his family had visited Lake Moondarra, a popular recreational lake approximately 15 km north-east of the city. On one of these occasions, the visit had lasted until mid-evening.

The boy's parents did not recall mosquito bites occurring at any specific times during the exposure period, although they had seen mosquitoes in the vicinity of their home and had seen other people applying insect repellent while at Lake Moondarra. Mosquito trapping suggested numbers of *Culex annulirostris* were very low within the city itself but moderately high, and in some areas very high, at the lake. Virus isolation results from mosquitoes are not available at the present time. Some targeted fogging to kill adult mosquitoes was undertaken, but because of the extensive mosquito breeding sites in and around Mt Isa, it was obvious that fogging would be of very limited benefit.

As soon as the diagnosis was made, a public awareness campaign was initiated in the Mt Isa and Gulf region. This included media alerts and the distribution of information and posters alerting residents and travellers of the necessary precautions to avoid mosquito bites.

Comment

The case described above was the first notified from Mt Isa in at least 26 years. Only 3 other cases have been notified from north Queensland in the last 10 years. The other cases' infections were acquired in Cape York (1991), near Burketown (1994) and either at Karumba or Mt Surprise (1997).

The nearest Australian encephalitis sentinel chicken flock to Mt Isa is situated in Tennant Creek. Tennant Creek is assumed to be proximal to Mt Isa during any seasonal southern extension of MVE from the endemic regions of the northern parts of Western Australia and the Northern Territory. The child had already been infected before the first indication of MVE seropositivity in the sentinel chicken flock at Tennant Creek.[1]

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Central Australian MVE update, 2001

Alex Brown,[1] Vicki Krause[2]

The Central Australian public health network became aware of 2 cases of Murray Valley encephalitis during February to March, 2001. Both cases were noted following reports of MVE virus activity in sentinel chicken flocks throughout the Kimberley and Pilbara regions of Western Australia, similar activity in western New South Wales and evidence of sequential north-south spread of virus activity in sentinel chicken flocks throughout the Northern Territory.

Additionally, prior to the detection of human cases of Murray Valley encephalitis, unusually high rainfall had been experienced throughout much of the Northern Territory including in Central Australia, which is commonly referred to as the 'central desert'. Tennant Creek and Alice Springs mosquito monitoring also demonstrated exceedingly high numbers of *Culex annulirostris* mosquitoes well known to transmit MVE virus.

On 22 February 2001 the Centre for Disease Control (CDC), Alice Springs received notification from the Emergency Department of the Alice Springs Hospital, of a suspected case of Murray Valley encephalitis in a 49-year-old female resident of Alice Springs. The second case was notified to CDC, Alice Springs on 3 March 2001, following the diagnosis of Murray Valley encephalitis in a 59-year-old Alice Springs man who had been transferred to the Royal Adelaide Hospital on 28 February 2001. The following are reports of these two cases.

Case 1 first developed symptoms on 19 February 2001. She presented with a 72 hour history of constant frontal headaches and fevers up to 40^{O}C, 48 hours of nausea and vomiting, 24 hours of slurred speech, expressive dysphasia, disorientation and generalised weakness. Within 12 hours of being hospitalised at Alice Springs Hospital the patient demonstrated progressive generalised weakness, respiratory failure and her deteriorating level of consciousness required intubation and ventilation.

Results from serological and cellular investigations of the cerebrospinal fluid (CSF) and serum for Case 1 are listed in the Table. Examination of the initial CSF specimen demonstrated 20 polymorphonuclear cells, 800 monocytes, 20 red blood cells and a raised protein level. Case 1 seroconverted to MVE virus, becoming IgM positive after the initial specimen was collected. A recent infection was supported by the demonstration of rising MVE virus specific IgG titres during follow up. CSF collected from the patient was positive for MVE RNA by polymerase chain reaction (PCR). The patient did not show any serological reactivity to Kunjin virus.

The CSF from Case 1 tested negative for herpes simplex virus, varicella zoster virus, acid fast bacilli, cryptococcus and listeria. Blood cultures were negative and serological testing ruled out infection with Japanese encephalitis, Ross River virus, Barmah Forest virus, Lyme disease or rickettsia.

At 2 months post admission the patient remains in ICU with persistent flaccid quadreparesis requiring ongoing respiratory support. There have been no significant neurological improvements to date.

Case 2 first developed symptoms on 23 February 2001. The patient presented to Alice Springs Hospital on 25 February 2001 with 48 hours of fevers (up to 39.5^{O}C), nausea and vomiting and epigastric abdominal pain. Four episodes of haematemesis (vomiting of blood) were recorded, and the presumptive diagnosis was of a Mallory-Weiss tear.

Over the next 12 hours the patient deteriorated on the ward, where he was found to be delirious with a temperature of 40.5^{O}C. The initial septic screen failed to find a source of sepsis. The patient deteriorated further over the next 24 hours, requiring transfer to a tertiary referral centre. On arrival at the Royal Adelaide Hospital he was noted to have a Glasgow Coma Score of 11, with hypotension, right-sided weakness and right-sided facial droop.

Results from serological and cellular investigations of the CSF and serum for Case 2 are listed in the Table. Initial CSF examination demonstrated 3 polymorphs, 2 red blood cells and 170 monocytes. Case 2 was diagnosed by the presence of MVE virus specific IgM in CSF and serum in combination with a clinical picture compatible with Murray Valley encephalitis. The patient also demonstrated serological reactivity to Kunjin virus. A further convalescent serum sample is required to further investigate whether MVE virus alone, or an infection in conjunction with Kunjin virus was the cause of the patient's encephalitis, although blocking antibody tests suggest that MVE is the likely cause. Insufficient CSF was available for Case 2 for further pathogen testing.

The patient remains in hospital, but is gradually improving. He demonstrates some generalised weakness, but with no specific focal neurological signs.

As of 23 April 2001, there have been no further confirmed cases of Murray Valley encephalitis among Central Australian residents. Falling daily temperatures and dry weather has meant the risk of MVE virus activity has lessened, however, sentinel chickens bled in early April 2001 continued to show MVE activity.

These cases represent the second consecutive year of MVE virus activity and human cases in Central Australia. There has only ever been one documented outbreak of Murray Valley encephalitis in the region prior to the cases demonstrated in 2000. This highlights the need for heightened clinical and public health surveillance in the coming years, particularly in the event of widespread summer rainfall throughout the region.

Acknowledgements

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2. Centre for Disease Control, Territory Health Services, Darwin

Table. CSF and serology results for Murray Valley encephalitis cases acquired in Central Australia, 2001

Case No	Date	Sample	Serum MVE HI titre	Serum MVE IgM	Serum MVE IgG-IFA	Serum Kunjin HI titre	Serum Kunjin IgM	Serum MVE PCR	CSF PMN's x10^6/L	CSF Monos x10^6/L	CSF Protein g/L
1	22/2/01	serum	<10	neg	-	<10	neg	neg			
	22/2/01	CSF	-	-	-	-	-	pos			
	22/2/01	CSF							20	800	1.09
	26/2/01	serum	<10	pos	<10	<10	neg				
	22/3/01	serum	<10	pos	640	<10	neg				
2	28/2/01	CSF	-	pos	-	-	-	neg	3	170	-
	01/3/01	serum	80	pos	640	80	equiv				
	14/3/01	serum	80	pos	1280	40	equiv				

CSF cerebrospinal fluid
HI haemagglutination inhibition
PMN polymorphonucleocytes
PCR polymerase chain reaction
Monos monocytes
pos positive
neg negative
equiv equivocal

Australian encephalitis: Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease encephalitis, in humans. Currently 30 flocks are maintained in the north of Western Australia, 9 in the Northern Territory, 12 in New South Wales and 10 in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information and details of the location of sentinel chicken sites see Commun Dis Intell *2000;24:8-9.*

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January/April 2001

Sentinel chicken serology was carried out for 29 of the 30 flocks in Western Australia in January and February 2001. There were 5 seroconversions to flaviviruses in January, 3 from the Kimberley and 2 from Ophthalmia Dam (near Newman) in the Pilbara. In February the number of seroconversions increased, particularly in the Kimberley region. There were 29 seroconversions in the Kimberley and one from the town of Newman in the Pilbara. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 1. As a result of these findings the Health Department of Western Australia issued a second health warning to residents living in these areas warning of the increased risk of infection with MVE virus. The Kimberley Public Health Unit has issued similar warnings to Kimberley Aboriginal communities.

Sentinel chicken serology was carried out for 28 of the 30 flocks in Western Australia in March and April 2001. There were 17 seroconversions to flaviviruses in March from the Kimberley and 33 from the Pilbara. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 2. As a result of these findings the Health Department of Western Australia issued a third health warning to residents living in these areas warning of the increased risk of infection with MVE virus. The Kimberley, Pilbara and Gascoyne Public Health Units have issued similar warnings to Aboriginal communities. In April activity decreased significantly and there were 3 seroconversions in the Kimberley (2 MVE, 1 FLAVI) and 4 seroconversions to MVE from Ophthalmia and the Harding dams in the Pilbara. There have been no cases of MVE reported from Western Australia.

The Western Australian sentinel chicken program is funded by the Health Department of Western Australia.

Serum samples from all of the 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in January and February 2001. There was one new seroconversion to Kunjin virus in January from Katherine. In February there was a total of 14 new seroconversions to flaviviruses. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 3. In addition there

Table 1. Flavivirus seroconversions in Western Australian sentinel chicken flocks, January to February 2001

	January 2001			February 2001		
Location	MVE	KUN	FLAVI	MVE	KUN	MVE/KUN
Kimberley						
Wyndham				3		4
Kununurra	1	1		4*	2	1*
Halls Creek				4		1
Fitzroy Crossing				5	1	2
Derby	1			1		
Broome				1		
Pilbara						
Newman (town)				1		
Ophthalmia Dam		1	1			

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
* some results not yet confirmed

Table 2. Flavivirus seroconversions in Western Australian sentinel chicken flocks, March to April 2001

	March 2001			April 2001		
Location	MVE	KUN	MVE/KUN	FLAVI	MVE	FLAVI
Kimberley						
Kalumburu						1#
Wyndham					1#	
Halls Creek	3				1	
Fitzroy Crossing	1		1			
Derby			1	1		
Broome	8		1			
Lombadina	1					
Pilbara						
Port/South Hedland	6		1			
Karratha	2					
Harding Dam	5				1#	
Marble Bar	1					
Pannawonica	2					
Tom Price	8					
Ophthalmia Dam	2	1			2	
Newman town	1		1			
Exmouth	3					

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
some results not yet confirmed

were 2 suspected cases (subsequently confirmed) of encephalitis caused by MVE virus from Alice Springs. Health warnings have been issued by the Territory Health Department.

Serum samples from all of the 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in March and April 2001. There were 11 new seroconversions to flaviviruses in March and 5 In April. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 4. A new case of MVE, with a date of onset in March 2001, was reported and the patient's travel history suggests the infection was acquired in the Northern Territory.

For the first time since 1974 there have been seroconversions to MVE and Kunjin viruses in the New South Wales sentinel chicken flocks. Flavivirus activity was detected in 7 of the 12 flocks with 8 seroconversions occurring in January 2001 and 5 in February 2001. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 5. To date there have been no cases of encephalitis caused by MVE virus reported from the region.

Flavivirus activity was again detected in New South Wales in March 2001 but not in April. MVE virus antibodies were detected in 2 of the 12 flocks and Kunjin virus antibodies in 8 of the 12 flocks. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 5. To date there have been no cases of disease caused by MVE or Kunjin viruses reported from the region.

In addition to the activity in New South Wales there have also been 12 seroconversions to Kunjin virus in 4 of the 10 Victorian sentinel chicken flocks in February 2001. Kunjin virus antibodies were detected in 4 chickens at Mildura, 5 chickens at Tooleybuc, 2 chickens at Barmah and 1 chicken at Barooga. The last Kunjin virus seroconversions in Victoria prior to this season were reported in March 1998 from the Mildura flock.

There were 7 new seroconversions, all to Kunjin virus, reported from the Victorian sentinel chicken flocks in March 2001. Kunjin virus antibodies were detected in 3 chickens at Mildura, 3 at Tooleybuc and 1 at Kerang. Two further seroconversions to Kunjin virus were detected from Kerang in late April. There have been no cases of disease caused by either MVE or Kunjin viruses reported from the region. The sentinel chicken surveillance programs in both Victoria and New South Wales have been extended until May 2001.

Details of the locations of all chicken flocks are given in Spencer JD, Broom AK, Buick TD, Daniels PW, Doggett SL, Hapgood GD, et al. Murray Valley encephalitis virus surveillance and control initiatives in Australia. *Commun Dis Intell* 2001;25:33-48.

Table 3. Flavivirus seroconversions in the Northern Territory sentinel chicken flocks, January to February 2001

	January 2001		February 2001		
Location	KUN	MVE	KUN	MVE/KUN	FLAVI
Howard Springs				1*	
Katherine	1		1*		1*
Tennant Creek		2		2	
Alice Springs		3*	1*	2*	1*

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
* some results not yet confirmed

Table 4. Flavivirus seroconversions in the Northern Territory sentinel chicken flocks in March and April 2001

	March 2001			April 2001		
Location	MVE	KUN	MVE/KUN	FLAVI	MVE	FLAVI
Howard Springs		1				
Leanyer						1
Coastal Plains					1	
Katherine		1	1	2		
Tennant Creek	6				2#	
Alice Springs					1#	

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
some results not yet confirmed

Table 5. Flavivirus seroconversions in New South Wales sentinel chicken flocks, January to March 2001

	January 2001		February 2001			March 2001		
Location	MVE	KUN	MVE/KUN	MVE	KUN	MVE	KUN	MVE/KUN
Menindee	2	1	1					
Macquarie Marshes	2			1		1	5	
Wanaaring	1		1				1	
Griffith					1		1	
Bourke					1	1	5	1
Ford's Bridge					2		1	
Deniliquin							1	
Lake Cargelligo							1	
Leeton							9	

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA

In case you missed it

New England Journal Of Medicine 2001;344:1294-1303

Global trends in resistance to anti-tuberculosis drugs

Researchers from the World Health Organization (WHO) and International Union against Tuberculosis and Lung Disease (IUATLD) expanded a WHO-IUATLD global survey to assess trends in resistance to anti-tuberculosis drugs by surveying patients in 58 geographic sites between 1996 and 1999. They found that among patients with newly diagnosed tuberculosis (TB), the frequency of resistance to at least one anti-TB drug ranged from 1.7 per cent in Uruguay to 36.9 per cent in Estonia. The prevalence of multi-drug resistance among new cases ranged from 0 per cent in 8 sites to 14.1 per cent in Estonia. It was also high in Henan Province, China (10.8%), Latvia (9.0%), the Russian oblasts of Ivanovo (9.0%) and Tomsk (6.5%), and Iran (5.0%). Among countries that had data available for at least 2 years, the prevalence of resistance to any drug among new cases significantly increased in Estonia, Denmark, Peru, New Zealand, and Germany. Significant decreases were observed in Spain, Switzerland, France, and the United States. The authors state that multi-drug-resistant TB continues to be a serious problem in countries of Eastern Europe as well as China and Iran, and is likely a result of inadequate TB control strategies.

Editorial comment: Australia continues to have very low rates of resistance to anti-tuberculosis drugs (National TB Advisory Committee. Tuberculosis notifications in Australia, 1998. *Commun Dis Intell* 2001;25:1-8.)

Report of the Australian National Polio Reference Laboratory, 1 July to 31 December 2000

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Abstract

The Australian National Polio Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory (VIDRL) is responsible for processing and testing samples for poliovirus from all Australian patients with acute flaccid paralysis and for identifying and characterising polioviruses recovered from untyped enteroviruses submitted from Australian laboratories. From 1 July to 31 December 2000, a total of 12 specimens from 7 patients with AFP were referred to the NPRL. Poliovirus type 3 Sabin-like was isolated from samples from 2 patients with suspected vaccine- associated paralytic poliomyelitis. No viruses were isolated from samples from the remaining 5 patients. Since 1995 a total of 1325 isolates have been referred for testing from laboratories throughout Australia. Seven hundred (53%) were confirmed as Sabin vaccine-like polioviruses, 542 (41%) were non-polio enteroviruses and 82 (6%) yielded no virus or viruses other than enteroviruses. At Kyoto, Japan in October 2000, the Western Pacific Region of the World Health Organization was declared wild polio-free. This represents a significant step towards the global eradication of poliovirus with one quarter of the world's population free of endemic infections from wild poliovirus. Surveillance of AFP and containment of wild polioviruses has been coordinated at the VIDRL. Since February 2000, Australia has been developing and implementing a plan for the containment of wild poliovirus stocks and potentially infectious materials. *Commun Dis Intell* 2001;25:54-58.

Keywords: poliomyelitis, acute flaccid paralysis, enteroviruses, polio certification, VAPP

Introduction

Poliomyelitis is a nationally notifiable disease and should be reported by all States and Territories to the National Notifiable Diseases Surveillance System.[1] Enterovirus surveillance of acute flaccid paralysis (AFP) cases is important in confirming that cases are not polio.

In 1988, the World Health Organization (WHO) made a commitment to the global eradication of poliomyelitis by the year 2000. An international laboratory network[2] has been progressively established and now consists of a three-tiered system incorporating specialised, regional and national or sub-national laboratories.

The Commonwealth health department supported the establishment of the Australian National Polio Reference Laboratory (NPRL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL) in 1994. The laboratory has served as a regional reference laboratory for the Western Pacific Region (WPR) of WHO since 1990. Surveillance of AFP was initiated in March 1995 and is currently coordinated at the VIDRL in collaboration with the Australian Paediatric Surveillance Unit (APSU) and the NPRL. AFP surveillance is a highly sensitive monitor to ensure that every possible case of paralytic poliomyelitis is detected, investigated and characterised.[3] Based on figures of other non-endemic countries, Australia should be detecting at least one case of AFP per 100,000 children below the age of 15 years each year.

The WHO Western Pacific Region, of which Australia is a member nation, was declared free of wild poliovirus in October 2000. Although significant progress has been made to eradicate wild poliovirus worldwide, there are some countries remaining that have yet to reach this target. For this reason, high quality surveillance of AFP needs to be maintained as part of an ongoing process for the detection of wild poliovirus importations.

Whilst Sabin oral polio vaccine (OPV) remains part of the Australian Standard Vaccination Schedule,[4] incidental polioviruses will continue to be isolated. Until global certification for poliovirus is reached, even countries like Australia that have been certified wild-poliovirus free, remain susceptible to importation. An additional mechanism to detect wild poliovirus in the community is screening all poliovirus and untyped enterovirus isolates nationwide, regardless of the source of the original sample. Further virological studies are performed in order to identify all polioviruses and subsequently characterise them as Sabin or wild-type.

This report describes the functions of the NPRL and its activities during the second half of 2000. Earlier reports of the laboratory's activities have been published.[5,6,7,8] The report of the activities of the laboratory from January to December 1998[5] includes its terms of reference and their implementation.

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Methods

Samples from AFP patients

As part of the WHO poliovirus-free certification process, every case of AFP in children under 15 years in Australia must be investigated for clinical and laboratory information and followed-up for residual paralysis at 60 days to ensure that the AFP case was not poliomyelitis.[9] Paediatricians are requested to promptly report all cases of AFP to the APSU and the AFP surveillance coordinator. Arrangements are then made for 2 stool samples to be collected 24 to 48 hours apart, within 14 days of onset of paralysis. The samples are kept at 4-8^{o}C and transported to the NPRL within 3 days of collection. If transportation of samples to the NPRL is delayed, samples must be frozen at -20^{o}C and shipped frozen when suitable arrangements can be made.

Referred specimens from non-AFP patients

Samples for non-AFP patients can be referred to NPRL to exclude poliomyelitis. Five faecal samples from 3 patients with non-AFP were transported to VIDRL for testing.

Characterisation of referred entero/polioviruses

Since 1995 the various state virology laboratories have supported the VIDRL in its task to characterise all polioviruses isolated from non-AFP patients to ensure they are Sabin vaccine-like. Incidental poliovirus islolates recovered from referred entero and poliovirus isolates are characterised by nucleic acid probe hybridisation (NAPH).

Characterisation of polioviruses

A comprehensive network of laboratories for enterovirus surveillance supplements the AFP surveillance system. All polio and untyped enterovirus isolates from Australian laboratories should be referred to the NPRL for identification and intratypic differentiation. The staff of the Virology and Serology Laboratory Reporting Scheme send reminders to all laboratories reporting polio and untyped enteroviruses to refer these strains to the NPRL. This scheme ensures that no wild polioviruses should go undetected within the Australian community.

Results

Samples from AFP patients

During the second half of 2000, a total of 12 stool samples from 7 patients with AFP were referred to the NPRL (Table 1). Two patients were from Queensland and New South Wales and one each from Victoria, South Australia and the Northern Territory.

Onset dates were available for 6 patients, of whom 5 had stool samples collected within 14 days. Six samples were transported to VIDRL within 3 days of collection.

Faecal samples from 5 patients failed to yield any viruses. Sabin-like poliovirus type 3 was isolated from faecal samples from 2 patients with suspected vaccine-associated paralytic poliomyelitis (VAPP).

One 5-month-old boy from Queensland who had received a second dose of Sabin OPV 18 days before the onset of encephalomyelitis and AFP (Table 1, Case 1), had 2 stool samples collected 5 and 7 days after onset of paralysis. The referring hospital reported the detection of enteroviral ribonucleic acid by polymerase chain reaction (PCR). The NPRL identified poliovirus type 3 from the first faecal sample. The isolate was characterised as poliovirus type 3 (Sabin) by NAPH and enzyme immunoassay (EIA). The second sample failed to yield a virus. Testing of paired sera collected 2 and 20 days after onset of paralysis suggested the patient had antibodies to all 3 poliovirus strains but no significant rise in antibody titres was observed. Both faecal samples were referred to the Women's and Children's Hospital in Adelaide. Culture results confirmed *Clostridium botulinum* type A. Assay for toxin detection could not be performed due to insufficient residual volume of the 2 samples. The case was considered as non-polio by the Polio Expert Committee[9] and subsequently classified clinically as infant botulism due to the patient's improved condition.

Table 1. Results of specimens tested for enteroviruses from patients with AFP, Australia, 1 July to 31 December 2000

State/ Territory	Specimen date (stool)	Result	Paralysis onset date	Date of last OPV	Serum collection date	Serum titre result		
						P1	P2	P3
Case 1 (Qld)	6 Sep 2000	P3 Sabin	1 Sep 2000	14 Aug 2000	3 Sep 2000	256	>724	181
	8 Sep 2000	NV14			21 Sep 2000	228	>724	144
Case 2 (Qld)	7 Sep 2000	NV14	1 Sep 2000	>3 weeks				
	8 Sep 2000	NV14						
Case 3 (Vic)	19 Sep 2000	P3 Sabin	14 Sep 2000	7 Sep 2000	19 Sep 2000	>724	>724	81
Case 4 (SA)	15 Oct 2000	NV14	8 Oct 2000	>3 weeks				
	17 Oct 2000	NV14						
Case 5 (NSW)	23 Oct 2000	NV14	3 Oct 2000	>3 weeks				
	24 Oct 2000	NV14						
Case 6 (NT)	29 Oct 2000	NV14	23 Oct 2000	16 Oct 2000				
Case 7(NSW)	8 Dec 2000	NV14	INP	INP				
	12 Dec 2000	NV14						

NV14.No virus isolated after 14 days incubation in cell culture.

INP. Information not provided.

One 5-month-old female from Victoria (Case 3) with decreased movement and poor attachment to the breast had received OPV 7 days earlier. The stool and serum samples collected from the patient 5 days after onset of paralysis were referred to the VIDRL. Initial testing identified an enterovirus by PCR. The poliovirus type 3 strain isolated from the patient's stool sample was characterised as Sabin-like using NAPH and EIA. Neutralisation tests performed on the serum sample suggested that the patient had antibodies to all 3 poliovirus strains (Table 1). Further investigation concluded infant botulism as a final diagnosis. A faecal sample sent to the Women's and Children's Hospital in Adelaide yielded a *Clostridium botulinum* type B toxin. The organism could not be cultured from the faecal sample.

Referred specimens from non-AFP patients

Of 2 patients from Queensland 1 had calf muscle pain following OPV and another was diagnosed with symmetrical polyneuropathy. No viruses were isolated from these samples.

The patient from the Northern Territory was diagnosed with viral meningitis. The referring laboratory cultured and identified poliovirus type 1 from the patient's cerebrospinal fluid but considered it to be a laboratory contaminant. The isolate was referred to the NPRL and tested positive for Poliovirus type 1 by PCR and Poliovirus type 1 Sabin vaccine-like by NAPH. Faecal samples collected 2 months later and referred to the NPRL failed to yield any viruses. A neutralisation test to detect poliovirus antibodies was performed on paired serum samples. The first and second serum samples were collected 2 days and almost 2 months respectively after the collection of the cerebrospinal fluid. Although the patient had poliovirus antibodies to all 3 serotypes, no significant rise in titres was observed.

Characterisation of referred entero/polioviruses

During July to December 2000, a total of 121 polio and enterovirus isolates from 4 states were referred to the NPRL for testing. Ninety-six (79%) were referred from Western Australia, 21 (17%) from Victoria, 2 (2%) from Tasmania and 2 (2%) from South Australia. Of these 121 isolates, 34 (28%) were characterised as Sabin vaccine-like polioviruses. Forty-four (37%) were non-polio enteroviruses, 42 (35%) could not be recovered and one (1%) isolate was identified as adenovirus type 2.

Between 1995 and December 2000, a total of 1325 isolates from within Australia have been tested at the VIDRL. Table 2 summarises the cumulative results of testing of these isolates during this period.

Discussion

Surveillance and investigation of acute flaccid paralysis

Surveillance has played an important role in certification of Australia and the other countries of the WHO Western Pacific Region as being wild poliovirus-free. As part of the polio eradication process, it is important to investigate not only the polio cases but also conditions that may resemble polio clinically.[10] The main causes of AFP in Australia in the last 5 years have been Guillain Barré Syndrome and transverse myelitis.[9] However, 2 infants recently presented with AFP following immunisation with oral polio vaccine (OPV). Poliovirus type 3 (vaccine-like) was isolated from the faecal samples of both patients, which suggested possible cases of VAPP. Further testing of the faecal samples concluded infant botulism was the cause of both patients' paralysis. Exclusion of VAPP as the differential diagnosis was made possible due to adequate faecal sample collection and extensive laboratory testing. The NPRL plans to sequence all poliovirus isolates from AFP cases and is currently performing molecular characterisation of the poliovirus type 3 isolates from the suspected VAPP cases to determine whether these viruses are true Sabin OPV or vaccine derived.

Surveillance of AFP is considered a highly sensitive indicator of wild poliovirus activity since nearly all cases of paralytic polio have AFP.[10] Based on figures in non-endemic countries, Australia should identify 40 cases of AFP in children below the age of 15 years annually,[11] or 20 cases for this reporting period.

Despite the continuous publicity and information provided to highlight the critical role of AFP surveillance, the expected number of AFP cases investigated was not reached in this reporting period. Between January to December 2000, samples from a total of 20 AFP cases (13 cases from an earlier report[8]) were referred to the NPRL. Until December 1999, there had been an increasing trend in the number of AFP cases reported, per calendar year – samples from 27 cases were referred to the NPRL in 1999. However, a decrease in the number of referred samples in 2000 may be attributed to complacency due to Australia approaching wild poliovirus-free certification. Also worth noting is the failure of hospitals to refer faecal samples collected from 4 AFP cases to the NPRL during the reported period. Although notification to the APSU had been made, these samples were tested in other laboratories for various pathogens. Samples from only one of these cases were tested for virus culture. A further 9 notifications to the APSU were made but specimens were not collected for laboratory investigation. If all notified AFP cases – 20 in total – had specimens collected and referred to the NPRL, Australia would have met the WHO criteria for AFP surveillance for this reporting period. Continuing commitment from paediatricians to report all cases of AFP, return study questionnaires and arrange collection and transportation of faecal samples in accordance with the WHO protocol to the NPRL is required. The Department of Health and Aged Care is committed to continuing AFP surveillance at an acceptable level until global certification of poliovirus-free status is achieved.

Western Pacific Regional Certification

The Regional Commission for the Certification of Poliomyelitis Eradication in the WPR was established in 1996. Its task has been to set the standards and criteria to be followed by every country and area in the Region.[10] The Regional Certification Commission met in Kyoto, Japan, during October 2000 to review each country's final documentation for certification. Every country of the WPR including Australia, provided evidence consistent with the absence of indigenous wild poliovirus for a minimum of 3 years whilst undertaking high quality surveillance. Other criteria to be met were the validation of the certification documentation by a National Certification Committee of each country and development of plans of action to detect and respond to importation of wild poliovirus and to contain wild poliovirus and potentially infectious materials. Once the Regional Certification Commission was satisfied with the

Table 2. Cumulative summary of identification of enteroviruses and intratypic differentiation of polioviruses from Australian laboratories from 1995 to December 2000 performed at the NPRL (percentage of total in brackets)

State	Year	Polio Sabin-like	Non-polio enterovirus	Non-enterovirus/ negative	Total
Victoria	1995	9			9
	1996	17			17
	1997	5			5
	1998	7			7
	1999	19			19
	2000	24			24
Queensland	1995	41	5	8	54
	1996	99	4	9	112
	1997	41			41
	1998	8	15	2	25
	1999	2			2
	2000				
Western Australia	1995/6	133	384	5	522
	1997	32	76		108
	1998			2	2
	1999	3	9	9	21
	2000	13	44*	47†	104
Tasmania	1995	1			1
	1996	3			3
	1997	4			4
	1998	4			4
	1999	4			4
	2000	2			2
New South Wales	1994	5			5
	1995	76	5		81
	1996	35			35
	1997	39			39
	1998	30			31‡
	1999	31			31
	2000	4			4
South Australia	1997	3			3
	1998	3			3
	1999	1			1
	2000	2			2
Total	1995-2000	700 (53%)	542 (41%)	82 (6%)	1,325

* Two non-poliovirus isolates were cultured in L20B and RD cells.

† Includes one adenovirus type 2 isolate.

‡ Includes one non-Sabin poliovirus type 2.

documentation for each country in the Region, a decision for certification was reached. At the Kyoto Meeting for the Declaration of Poliomyelitis Eradication in the WPR on 29 October 2000, the Regional Director of the WPR WHO declared that the Region had achieved polio-free status.

Maintaining poliovirus-free status

The WPR is now the second region in the world to have achieved a wild poliovirus-free status. The Americas Region was certified wild polio-free in 1994. However, wild poliovirus is still circulating in countries throughout other non-certified regions. The threat of importation exists in Australia due to immigration from endemic countries. To prevent the reintroduction of wild poliovirus, it is imperative that we maintain high vaccination coverage and effective surveillance of AFP cases. The one case of poliomyelitis resulting from the importation of wild poliovirus from India into China in 1999[12] highlights the importance of high quality AFP surveillance and a national strategy to respond to importations.

An outbreak of poliomyelitis in the Caribbean between July and November 2000 was caused by a mutant form of a type 1 poliovirus from OPV.[13] The reverted strain had

characteristics of both neurovirulence and transmissibility, which the live attenuated strains in OPV do not possess. In Egypt, cases of poliomyelitis between 1988 and 1993 were associated with vaccine-derived poliovirus type 2.[14] In both areas, circulation of vaccine-derived polioviruses occurred in areas with low OPV coverage.

These examples highlight the need for Australia to remain vigilant by maintaining adequate AFP surveillance, high immunisation coverage, a plan to effectively respond to a wild-type poliovirus importation and ensuring adequate laboratory containment of wild poliovirus materials[15] until global certification has been achieved.

Abbreviations

National Polio Reference Laboratory (NPRL), Victorian Infectious Diseases Reference Laboratory (VIDRL), World Health Organization (WHO), Western Pacific Region (WPR), Australian Paediatric Surveillance Unit (APSU), Vaccine-Associated Paralytic Poliomyelitis (VAPP), Acute Flaccid Paralysis (AFP), Oral Polio Vaccine (OPV), Polymerase Chain Reaction (PCR), Nucleic Acid Probe Hybridisation (NAPH), Enzyme Immunoassay (EIA)

Acknowledgements

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Addendum

Stambos V, Brussen KA, Turnbull A et al. Report of the National Polio Reference Laboratory: 1 January to 30 June 2000. *Commun Dis Intell:* 2000;24:300-303.

The last reported case of indigenous wild poliovirus in the Western Pacific Region was in Cambodia in March 1997 and not in 1977 as reported in the discussion section. The authors apologise for any inconvenience that this may have caused.

Annual report of the Australian Gonococcal Surveillance Programme, 2000

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all States and Territories. In 2000 the *in vitro* susceptibility of 3,468 isolates of gonococci was determined by standardised methods. Antibiotic susceptibility patterns varied considerably between regions. Resistance to the penicillins was highest in larger urban centres and warrants close attention in those rural centres where treatment with the penicillins continues. Quinolone resistance in gonococci became more widespread in Australia in 2000. Endemic cycles of transmission of quinolone-resistant gonococci (QRNG) in homosexually active men continued in Victoria but declined in New South Wales. Heterosexual endemic transmission of QRNG increased substantially in New South Wales and the proportion of all gonococci represented by QRNG also increased markedly in Queensland and Western and South Australia. All isolates remained sensitive to spectinomycin, but a small number of isolates in a number of jurisdictions showed some decreased susceptibility to ceftriaxone. Strains examined in South Australia, New South Wales and Victoria were predominantly from male patients and rectal and pharyngeal isolates were common. In other centres the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2001;25:59-63.

Keywords: surveillance, Neisseria gonorrhoeae*, antimicrobial resistance, gonorrhoea, antibiotics, quinolones, penicillins, spectinomycin, cephalosporins*

Introduction

Gonorrhoea is essentially the only bacterial STI found in Australia where antibiotic resistance materially affects both individual management and public health control. *Neisseria gonorrhoeae* has developed some form of resistance to all agents used in the treatment of gonococcal disease. This resistance has led to the abandonment or modification of some antibiotic treatment regimens and hindered efforts aimed at disease control. The Australian Gonococcal Surveillance Programme (AGSP) was established to monitor the susceptibility to antibiotics of gonococci isolated throughout the country. The AGSP is a long-term collaborative programme of surveillance conducted by reference laboratories in each State and Territory. Data from this programme were published each quarter in *Communicable Diseases Intelligence* from 1981 and annual reports have been produced since 1996.

Methods

The AGSP comprises participating laboratories in each State and Territory (see acknowledgments). It is a collaborative network of laboratories which obtains isolates for examination from as wide a section of the community as possible. Both public and private sector laboratories refer isolates to regional testing centres. The sources of isolates remained relatively unchanged in 2000. However, the increasing use of non-culture based methods of diagnosis has the potential to reduce the number of cultures available for susceptibility testing.

Gonococci isolated in and referred to the participating laboratories were examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins and for high level resistance to the tetracyclines by a standardised methodology.[1] In 2000, the AGSP also conducted a programme-specific quality assurance (QA) programme.[2] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory which collated the results and also conducted the QA programme. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. The geographic source of acquisition of resistant strains was ascertained whenever possible.

Results

Numbers of isolates

There were 3,547 gonococcal isolates referred to or else isolated in AGSP laboratories in 2000 and the distribution and site of infection of these isolates are shown in the Table. Of these, 3,468 (97.8%) remained viable for susceptibility testing in 2000. One-thousand, two-hundred and fifty-five gonococci (35% of the Australian total) were isolated in New South Wales, 802 (22.6%) in Victoria, 620 (17.5%) in Queensland, 445 (12.5%) in the Northern Territory, 317 (9%) in Western Australia, and 93 (2.6%) in South Australia with small numbers in Tasmania and the Australian Capital Territory. The site of isolation and sex of some infected patients was not known.

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Table. Gonococcal isolates, Australia, 2000, by sex, site and region (excluding those from the Australian Capital Territory and Tasmania)

	Site	NSW	Vic	Qld	SA	WA	NT	Aust
Male	Urethra	892	620	413	62	224	128	2,352
	Rectal	182	91	16	12	7	0	308
	Pharynx	91	44	8	5	2	1	152
	Other/NS	22	8	22	4	20	127	203
	Total	1,187	763	459	83	253	256	3,015
Female	Cervix	57	33	151	3	55	147	447
	Other/NS	8	6	10	7	9	42	82
	Total	65	39	161	10	64	189	529
Total		1,255	802	620	93	317	445	3,547

Compared with data from the same sources in recent years, there were continuing increases in the number of isolates in Victoria (from 362 in 1997, 565 in 1998 and 744 in 1999 to 802) and Queensland (from 516 in 1998 and 589 in 1999). The number of isolates available from the Northern Territory, Western Australia and South Australia was stable. In New South Wales the number of isolates decreased for the first time in many years (from 902 in 1997, 1,386 in 1998 and 1,528 in 1999). Numbers in other centres were low.

Source of isolates

For the 3,544 cases isolated for whom data on gender were available, there were 3,015 strains from men and 529 from women, with a male:female ratio of 5.7:1. The number of strains from men decreased slightly from the 3,111 examined in 1999 but was still higher than the 2,233 in 1997 and 2,886 in 1998. Strains from women decreased further from 628 in 1999 and 697 in 1998 and the total was below the 1997 figure of 597. The male:female ratio was 3.7:1 in 1997, 4.1 in 1998 and 5.1 in 1999. The male:female ratio remained highest in Victoria (19.5:1) and New South Wales (18.3:1) where strains were obtained more from urban populations, but lower in South Australia (8.3:1). The lower ratios in Western Australia (3.9:1), Queensland (2.8:1) and the Northern Territory (1.4:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in New South Wales (23% of isolates from men) and Victoria (17.6%). This pattern is similar to that noted in recent years. About 8 per cent of isolates are shown as being isolated from 'other' sites. These included 13 cases of disseminated gonococcal infection, 11 in men and 2 in women. Not all infected sites were identified. Isolates from urine samples were regarded as genital tract isolates. Although most of the other isolates were probably from this source, they were not so specified. There were a small number of isolates from the eyes of both newborn and older infants.

Antibiotic susceptibility patterns

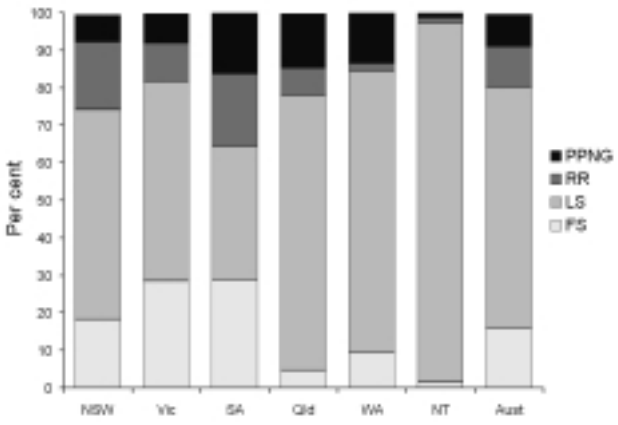
In 2000 the AGSP reference laboratories examined 3,468 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed greatly between the various States and Territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

Resistance to the penicillin group (penicillin, ampicillin, amoxycillin) may be mediated by the production of beta-lactamase (penicillinase-producing *N. gonorrhoeae* - PPNG) or by chromosomally-controlled mechanisms (CMRNG).

Chromosomal resistance is expressed as the minimal inhibitory concentration in mg/L (MIC) which is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions. The categorisation of strains in Australia in 2000 by penicillin MIC is shown in Figure 1. The MIC reflects the expression of multiple and different chromosomal changes present in an organism. These multiple changes result in incremental increases in the MIC. Strains are classified as fully sensitive (FS, MIC 0.03 mg/L), less sensitive (LS, MIC 0.06 - 0.5 mg/L) or relatively resistant (RR, MIC 1 mg/L). PPNG are a separate (resistant)

Figure 1. Penicillin resistance of gonococcal isolates, Australia, 2000, by region, (excluding those from the Australian Capital Territory and Tasmania)



FS fully sensitive to penicillin, MIC 0.03 mg/L
LS less sensitive to penicillin, MIC 0.06 – 0.5 mg/L
RR relatively resistant to penicillin, MIC 1 mg/L
PPNG penicillinase producing *N. gonorrhoeae*

category. Infections with strains in the less sensitive or fully sensitive categories usually respond to therapy with standard treatment regimens with the penicillins. Infections caused by strains which are PPNG or in the relatively resistant category (CMRNG) usually fail to respond to treatment with the penicillins.

In 2000, there were 377 isolates found to be resistant to penicillin (10.6%) by chromosomal mechanisms. This was lower than the 782 (21.8%) recorded in 1998 and the 525 (14.3%) in 1999. Strains of this type were concentrated in New South Wales (224 CMRNG, 18% of all isolates), Victoria (81 CMRNG, 10% of all isolates), South Australia (18 CMRNG, 19%) and Queensland (42 CMRNG, 7.2%). In contrast there were 6 (2%) CMRNG amongst Western Australian isolates and 6 (1.4%) in the Northern Territory strains.

PPNG again increased in 2000 both numerically (to 302 from 269 in 1999 and 206 in 1998), and as a proportion of all isolates (to 8.7% from 7.4% and 5.3% in 1999 and 1998 respectively). Again the distribution of PPNG differed significantly by region. New South Wales had the highest number, 92 (7.4%) of PPNG but the highest proportions were in South Australia (15 strains, 16%); Queensland (85, 14.6%) and Western Australia (41, 13.4%). The 62 PPNG in Victoria represented 7.8 per cent of strains. Five PPNG were found in the Northern Territory (1.1%) and there were 2 PPNG strains in Tasmania. Indonesia, the Philippines, Thailand, Vietnam and China were the most frequently nominated countries of PPNG acquisition. PPNG acquisition was also reported from contact in Korea, Singapore, Hong Kong, Brazil, Malaysia, Cambodia and Taiwan.

Ceftriaxone

A small number of strains in a number of states and territories showed a small increase in ceftriaxone MICs. This phenomenon requires continued monitoring.

Spectinomycin

All isolates of *N. gonorrhoeae* were susceptible to spectinomycin in 2000.

Quinolone antibiotics

Resistance to the quinolone antibiotics is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered resistance as an MIC of 0.06 mg/L or more. Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with this less developed resistance in about 90 per cent of cases, but lower doses of the antibiotic will more often result in treatment failure. The proportion of treatment failures increases exponentially as MICs rise. Treatment failure occurs in about 60 per cent of infections with strains with MICs of 1 mg/L or more, even when higher doses are used. Currently gonococci with MICs up to 16 and 32 mg/L are being seen in Australia. Newly released quinolone agents would not be expected to offer any significant advantage over ciprofloxacin for the treatment of gonorrhoea.

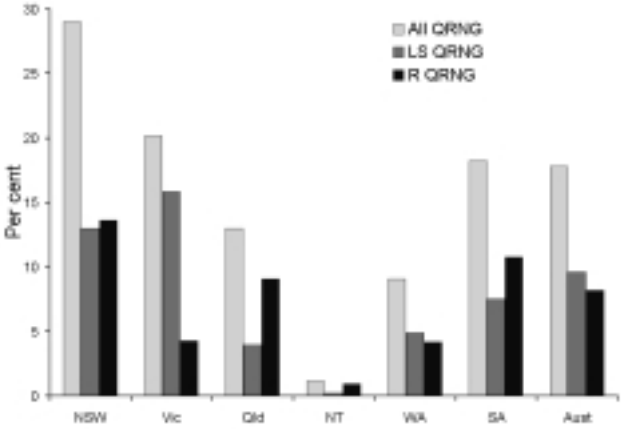
In 2000 a total of 619 gonococci (17.8%) displayed altered sensitivity to the quinolones (QRNG). This is about the same number and proportion of QRNG seen in 1999 (628, 17.2%) but more than the three times the number of QRNG seen in 1998 (186, 5.2%). The distribution of QRNG changed significantly in 2000, with larger numbers found in more states and territories, apart from a slight decline which occurred in the two larger States. In 1999 the high rate of QRNG mainly resulted from their distribution in homosexually active men (HAM) in New South Wales and Victoria. This high rate of QRNG in HAM in Victoria was maintained, but declined in New South Wales in 2000. The QRNG in HAM in these states were in the lower MIC range, namely, 0.06 – 0.5 mg/L.

Although the proportion of QRNG in HAM declined in New South Wales throughout 2000, an increasing proportion of isolates from that State were higher level QRNG (MIC 1mg/l or more). These QRNG were seen in infections in the male clients of female commercial sex workers. Rates of QRNG have been high in New South Wales since an increase in the number and proportion of QRNG in heterosexuals was noted in New South Wales in the December quarter of 1996. This rate of isolation was sustained throughout 1997 and the early part of 1998, but declined in the latter part of that year. The 332 QRNG in New South Wales represented 26.6 per cent of all isolates and similar numbers of low and higher level QRNG were noted.

In Victoria 160 QRNG accounted for 20 per cent of all isolates but 126 nearly (80%) were in the less sensitive range. The proportion of QRNG in Victoria in 1999 was 24 per cent.

About 80 per cent of all QRNG identified in Australia in 2000 were found in Victoria or New South Wales (Figure 2). QRNG were found in all centres except the Australian Capital Territory. Numbers of QRNG increased in all other states and territories. Queensland had 76 (13%) QRNG, almost twice the proportion in 1999. In South Australia there were 17 QRNG, also a substantial increase over 1999. In Western Australia there were 28 QRNG (8.8%), up from 9 (3%) in 1999. The Northern Territory recorded 5 QRNG (1.1%). The spread of QRNG in Sydney and Melbourne was mainly by local as opposed to overseas contact, but in most other centres cases were imported from overseas contact

Figure 2. Quinolone-resistant *N. gonorrhoeae*, Australia, 2000, by region, (excluding those from Tasmania)



LS QRNG Ciprofloxacin MICs 0.06 – 0.5 mg/L
R QRNG Ciprofloxacin MICs 1 mg/L

from sources similar to those described for PPNG acquisition.

High level tetracycline resistance

Three hundred and eighteen high level tetracycline resistant *N. gonorrhoeae* (TRNG, 9.1 % of isolates) were detected throughout Australia in 2000, continuing a slight upward trend (288, 7.9% in 1999). Most TRNG were found in Queensland (115, 19.8%) with a high proportion also recorded in Western Australia (38, 12.4%). There were 91 (7.3%) TRNG in New South Wales, 56 (7%) in Victoria, 9 (9.6%) in South Australia, and 7 (1.6%) in the Northern Territory. Infections with TRNG were mainly acquired overseas in Indonesia, Thailand and Singapore.

Discussion

The World Health Organization guidelines suggest that a rate of gonococcal resistance to an antibiotic of 5 per cent or more is an indication to change treatment schedules. The AGSP data has noted considerable regional variation in susceptibility patterns over many years and this was again present in 2000. This suggests that treatment regimens suited to local patterns of resistance should be adopted.

A high proportion of the gonococci isolated in urban centres has been resistant to the penicillins for many years and this trend was maintained in 2000. Rates of penicillin resistance in New South Wales, Victoria, South Australia, Queensland and Western Australia ranged between 15 and 35 per cent. Most of this resistance was chromosomally mediated and in locally acquired strains in New South Wales and Victoria, but in Western Australia and Queensland PPNG were more important. While PPNG rates had declined for a number of years prior to 1999, PPNG are now being seen in widely dispersed areas of rural Australia and the proportion of PPNG is again increasing. Although the proportion of CMRNG in the Northern Territory and Western Australia remains low, there has been a continuing shift upwards in MICs to the point where close surveillance needs to be maintained if penicillins are to remain the preferred treatment option.

There has also been considerable volatility in the proportion of QRNG seen in Australia over recent years and this continued in 2000. The pattern of a high proportion of strains less sensitive to the quinolones was maintained in Victoria. In New South Wales the less sensitive strains in HAM declined but higher level resistance was seen in another sub-population. The continuing high levels of endemic transmission of QRNG observed in these two centres indicate that use of quinolones should be discontinued. The proportion of QRNG in other centres increased significantly in 2000 suggesting that use of these agents in the treatment of gonorrhoea should also be reconsidered in these jurisdictions.

Most gonococcal isolates were fully susceptible to the third generation cephalosporin ceftriaxone, although a few strains showed a ‘shift to the right’ in terms of slightly increased MICs. All gonococci tested in Australia were susceptible to spectinomycin.

The number of TRNG increased only slightly in 2000 after several years of substantial rises. Sustained domestic transmission of TRNG was evident especially in Sydney. The spread of TRNG is examined as an epidemiological marker and tetracyclines are not a recommended treatment for gonorrhoea.

The sample of available isolates in 2000 declined slightly, but was sufficient for the purpose of susceptibility surveillance. AGSP has until now been able to confirm other findings on rates of gonococcal disease in Australia by comparing data from its sample of isolates obtained from relatively unchanging sources. Additionally, AGSP data record site of isolation that was not always available in other data sets. This had allowed the AGSP to comment on trends in gonococcal disease in Australia as a by-product of its prime role in antibiotic susceptibility surveillance. This situation has been irrevocably altered by the increasing use of non-culture based methods of diagnosis. It is not possible to determine the number of diagnoses made by nucleic acid amplification testing methods or if these were additional diagnoses made in outreach settings or simply a replacement of traditionally derived diagnoses. For these reasons no comment is made on the significance of altered numbers in the AGSP sample.

Gonorrhoea remains an important disease globally. Mucosal infection has a well documented complication and morbidity rate and deleterious consequences include decreased fertility through early trimester abortion and pelvic inflammatory disease. Neonatal and, in Australia, childhood ophthalmia, is also well recognised. Gonorrhoea also is known to significantly amplify the rate of transmission of HIV. Rates of gonorrhoea are again increasing in a number developed countries and this increase has also been reported in parts of Australia. Control of gonorrhoea is therefore essential but is a complex issue requiring a concerted and continuing effort on many fronts. One essential component of this integrated approach to control is the availability of simple but effective antibiotic treatments for the disease. However treatment is becoming more difficult as the choice of suitable therapy is increasingly restricted by antibiotic resistance. Continued monitoring of resistance patterns is therefore required to optimise treatment regimens, individual case management and disease control.

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Investigation and control of a cluster of cases of Legionnaires disease in western Sydney

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Abstract

Three cases of *Legionella pneumophila* infection were identified in Sydney's west in November 1998. Epidemiological investigations identified an association with one workplace. Environmental sampling revealed that the cooling towers in the workplace, and at 2 other premises within a 1 km radius of the workplace, were positive for *L. pneumophila* serogroup 1 (LP1) which was indistinguishable from clinical isolates of 2 of the cases on DNA fingerprinting. Our report highlights limitations of the current control program for *Legionella* in cooling towers, including the finding of unregistered cooling towers, cooling towers positive for LP1 despite satisfactory results on inspection, and cooling towers potentially linked to disease with counts of LP1 below the current protocol requirements for immediate decontamination. *Commun Dis Intell* 2001;25:63-66.

Keywords: legionellosis, legionella, cooling towers, surveillance, environmental health

Introduction

Legionnaires' disease was first identified in 1976 after an outbreak occurred at an American Legion convention.[1] Legionnaires' disease is generally caused by the environmental pathogen *Legionella pneumophila* serogroup 1 (LP1) or *Legionella longbeachae*.[2] Outbreaks of LP1 have been associated with cooling towers,[3-9] evaporative condensers,[10] domestic hot water systems,[11,12] and excavation and construction activity.[13] On average one LP1 outbreak is reported annually in Australia.[2]

In western Sydney outbreaks of Legionnaires' disease occurred in 1992,[14] 1993,[8] 1994[15] and 1995.[15] We report here on a cluster of cases of Legionnaires' disease associated with a cooling tower in a workplace in Sydney's west.

Methods

Epidemiological investigation

Two cases of Legionnaires' disease[16] were notified by laboratories to the Western Sector Public Health Unit on 12 November 1998. Both cases satisfied definitions of confirmed cases in the NSW Health Department Infectious Diseases Manual.[16] When initial investigations revealed that the 2 cases were employed in the same workplace (Premises A) an outbreak was suspected and active surveillance was initiated and continued for a 10-day period. All emergency departments and intensive care units were contacted daily to ascertain whether there were any probable cases of Legionnaires' disease. Laboratories were also contacted daily to ensure test results were available as soon as possible. Additionally, we requested that the workplace alert us of staff members suffering prolonged respiratory illness.

All cases were interviewed by Public Health Unit staff using a standard questionnaire.[17] Information on the case's movements, including potential exposures to *Legionella*, during the 10-day period prior to onset of illness was obtained for each case. Where the case was unconscious, proxies were interviewed and electronic work diaries and time sheets were used as sources of information. Clinical specimens were processed using standard microbiological techniques. Putative isolates were identified as *L. pneumophila* by growth, colony characteristics and serogrouping.

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Environmental investigation

Environmental investigations commenced immediately on receipt of notifications, which was between 3 and 5 weeks after the likely exposure. Potential environmental sources of *Legionella*, such as cooling towers, at or near sites of exposure reported by cases, were identified from registers held by local government authorities.

These sources were then inspected and sampled in sequence, commencing at locations of common exposure to more than 1 case, and progressing to those where only 1 case reported potential exposure. At sites of common exposure, potential sources up to 500 m from the site were investigated. When a third case was reported with exposure 800 m from Premises A, the radius of investigation around Premises A was increased to 1 km. The Environmental Health Officers made efforts to identify any unregistered premises or cooling towers within that radius by visual inspection from suitable vantage points.

Molecular investigations

Environmental samples were tested for the presence of *Legionella* by the Legionella Reference Laboratory of the Division of Analytical Laboratories (DAL), Lidcombe, New South Wales. This testing involved the use of standard culture procedure, using heat treatment at 50 C for 30 minutes and Glycine-Vancomycin-Polymixin D-Cyclohexamide agar. Cultural confirmation involved the use of Buffer-charcoal-yeast extract and Tryptocase-soya broth agar, Gram staining and serological characterisation.

Molecular investigations were carried out by the Molecular Microbiology Laboratory of the DAL. Isolates were sent for molecular testing from cooling towers which tested positive for LP1 at premises where there was an epidemiological link to the cases. In situations where more than 1 cooling tower at the premises tested positive for LP1, samples from the cooling tower with highest count were examined. At least 2 different isolates from each cooling tower and all available clinical isolates were subjected to molecular testing. Isolates were characterised by two methods – randomly amplified polymorphic DNA[18] and restriction fragment lengths polymorphism.[19] The DNA fingerprint profiles were then visually compared.

Results

Epidemiological investigation

Three cases with onset dates 6 days apart were notified to the Public Health Unit and characterised as confirmed cases (Table 1). Active surveillance identified a fourth case that met initial probable case definition criteria, but was subsequently excluded on the basis of negative *Legionella* antibody tests in acute and convalescent sera.

All cases survived. One case developed acute renal failure. In-depth interviews (via proxy in Cases 1 and 2) revealed 3 epidemiological links between the cases. These were as follows: Cases 1 and 3 were employed at the same premises, (Premises A); Case 2 lived 800 m south of Premises A; and Cases 1 and 3 attended a day long seminar in the same premises during their incubation periods, which was 6 km north of Premises A.

Environmental investigation

Intensive investigation identified 8 premises with 30 cooling towers within 1 km of Premises A, including 3 premises with a total of 6 cooling towers which had not been registered with the local government authority. Four cooling towers in premises within 500 m of Premises A were found to be positive for LP1; 2 of these towers were in Premises A, and the remaining 2 were at Premises B located within 100 m of Premises A. All of these cooling towers had been registered and were satisfactory on inspection. (Table 2)

Table 1. Cases by date of onset, age, case criteria and risk factors

Case number	Date of onset	Age (years)	Case criteria	Risk factor
Case 1	23/10/98	44	Urine positive for *Legionella pneumophila* antigen	smoking
Case 2	25/10/98	60	*L. pneumophila* serogroup 1 isolated	smoking
Case 3	29/10/98	66	*L. pneumophila* serogroup 1 isolated	smoking

Table 2. Summary of *Legionella pneumophila* 1 (LP1) counts by the number of premises and cooling towers in each distance range

Distance from Premises 1 (metres)	Number of premises	Number of registered premises	Number of premises LP1 positive	Number of cooling towers	Number of registered cooling towers	Inspection satisfactory	Number of cooling towers LP1 positive
< 500	5	4	2	21	20	19	4
500-1000	3	1	2	9	4	5	5
1000 – 2000 *	6	6	0	10	10	8	0
> 2000 *	11	11	3	32	32	32	4
Total	25	22	7	72	66	64	13

* Only premises linked to case exposures were investigated at these ranges

A further 5 cooling towers in 2 premises © and D) between 500 and 1000 m of Premises A were found to be positive for LP1. Four of these towers were at one of the unregistered premises (Premises D) and were unsatisfactory on inspection. The remaining towers, including those at Premises C and the other unregistered premises, were satisfactory on inspection. (Table 2)

Outside this range, only cooling towers at sites reported as visited by cases were inspected and sampled. LP1 was isolated from a further 4 cooling towers at 3 premises at sites from 2 km to 20 km from Premises A; this did not include the premises attended by 2 of the cases for a seminar.

Overall 72 cooling towers at 25 premises were inspected and sampled during the investigation, and 13 cooling towers were found to be positive for LP1.

Following inspection and sampling, immediate decontamination was undertaken for all cooling towers in Premises A and B between 13 and 15 November, which was 2 and a half weeks after the onset of the last case. At other premises, action was taken in accordance with the New South Wales Code of Practice for the control of Legionnaires' disease.[20] All cooling towers that were positive for *L. pneumophila* were re-tested following decontamination.

Molecular typing was undertaken on LP1 isolates from 4 cooling towers from the 4 LP1 positive premises within 1 km of Premises A and compared with clinical isolates obtained from 2 patients (Cases 2 and 3). The DNA profiles of the 2 clinical isolates were identical and matched at least 1 isolate from 3 of the cooling towers (at Premises A, B and C, Table 3).

Discussion

We identified 3 cases of Legionnaires' disease, which we viewed as a sentinel to initiate active surveillance and a comprehensive environmental investigation. The epidemiological links among the 3 cases were supported by the finding of indistinguishable DNA profiles in the 2 cases where clinical specimens were available.

Environmental investigation identified cooling towers positive for LP1 in 4 premises which were potentially linked to the cases. Molecular typing was unable to distinguish between LP1 isolates from cooling towers at 3 premises and clinical samples from 2 cases. However, the lack of information on the frequency with which this particular DNA profile of LP1 is found in cooling towers prevents a definite conclusion on the source of the cluster.

The environmental investigation identified that 50 per cent (4/8) of the premises and 30 per cent of the cooling towers (9/30) within 1 km of Premises A were positive for LP1 on sampling. This proportion is higher than that found in a survey of registered New South Wales clubs in 1997 (6% of 126 clubs tested were positive for *Legionella* spp)[21] and higher than that found among the premises and cooling towers investigated which were distant from Premises A.

This could be due to the more intensive investigation within the 1 km radius of Premises A, which identified 3 premises and 6 cooling towers which had not been registered. Four of these cooling towers were positive for LP1 and were unsatisfactory on inspection. This suggests that action by cooling tower owners to register towers with their local councils may be associated with improved cooling tower maintenance and reduced detection of LP1. However, even if the unregistered towers are not included, the rate of isolation of LP1 in this area is high and other factors, such as the preponderance of industrial rather than commercial premises, and the time of the year, need to be considered. (Our Environmental Health Officers anecdotally report that production schedules in some industrial premises have created difficulties in adhering to regular maintenance regimes for cooling towers).

The New South Wales Code of Practice has a response protocol based on stratified counts of *L. pneumophila* and it recommends immediate shutdown and decontamination

Table 3. Cooling towers where LP1 was isolated and results of molecular typing

Location	Cooling tower*	On register	Inspection satisfactory	Legionella count†		Molecular typing done	DNA fingerprint match
		Y/N	Y/N	LP 1	Other		
< 500m	A.1	Y	Y	330	70	Y	Case 2, 3
	A.2	Y	Y	40	0	N	
	B.1	Y	Y	130	50	Y	Case 2,3
	B.2	Y	Y	40	0	N	
500 – 1000m	C.1	Y	Y	370	0	Y	Case 2,3
	D.1	N	N	40	30	Y	None
	D.2	N	N	10	0	N	
	D.3	N	N	30	10	N	
	D.4	N	N	10	0	N	
> 2000m	E	Y	Y	150	70	N	
	F.1	Y	Y	20	0	N	
	F.2	Y	Y	10	0	N	
	G	Y	Y	100	0	N	

* Cooling tower notation as premises cooling tower number.

† Colony forming units per millilitre (cfu/mL).

where counts of *L. pneumophila* in cooling tower water exceed 1000cfu/mL.[20] For counts between 100 and 999 cfu/mL a re-evaluation of maintenance procedures, including the current disinfection process is recommended.[20] None of the premises within a 1 km radius of Premises A had counts over 370 cfu/mL. Although these samples were taken 2 to 3 weeks after the onset dates for the cases, these cooling towers were associated with disease.

The response protocol based on stratified counts of *L. pneumophila* first appeared in an internal Department of Housing and Construction report in 1987.[22] The report suggested that the use of stratified *Legionella* counts should only be used as a guide and as an interim tool. The evidence for the stratified counts was based on the consensus of experts, primarily from the United States of America, rather than specific quantitative studies (personal communication, Mr Clive Broadbent (AM), Legionella Consultant, Canberra, 20 December 2000). These stratified counts were then adopted into the New South Wales Code of Practice in 1991, based on the 1987 internal Department of Housing and Construction report (personal communication, Mr Tony Burns, Senior Environmental Health Officer, Wagga Wagga, 21 December 2000). The findings of this investigation, and results of recent research in South Australia[23] which indicate that health risks from cooling towers cannot be reliably based upon single or infrequent *Legionella* tests, suggest that a review of the response protocol is required.

Seventy percent (9/13) of *L. pneumophila* positive cooling towers were assessed as compliant with the *Public Health Act 1991* at the time of inspection and sampling. The questionnaire includes a visual assessment of cleanliness, water turbidity, presence of slime, type of biocide used and recent cleaning history. We suggest that a broader questionnaire may be needed, as legislative compliance is a proxy for risk of *Legionella* contamination. The emphasis in the Code of Practice is on correct maintenance of cooling towers and occupiers are not required to sample cooling towers and test for *Legionella*.[20] Our findings suggest that either the questionnaire does not reflect the true maintenance of cooling towers or current maintenance procedures do not ensure the elimination of *Legionella* from cooling towers.

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Prevalence of tattooing and body piercing in the Australian community

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Abstract

Tattooing and body piercing are now worldwide fashion crazes. The health risks associated with these procedures are as yet unclear. This article examines the prevalence of body decoration and the associated health risks within the Australian population using a random sample survey of individuals aged 14 years and over, collected between June and September 1998 (n = 10,030). The results show that one in 10 people have had a tattoo at some point in their lives and 8 per cent some form of body piercing, excluding ear piercing. Men are more likely than women to report tattooing, while females are more likely to report body and ear piercing. Some 10 per cent of respondents report drinking alcohol or using other drugs when the procedures were undertaken. The prevalence of tattooing and body piercing is considerably higher among injecting drug users. Although the rates of transmission of bloodborne disease due to body decoration are believed to be low, the strong association with youth and with injecting drug use suggests considerable potential for transmission. *Commun Dis Intell* 2001;25:67-72.

Keywords: tattooing; body piercing; hepatitis C; HIV/AIDS; injecting drug use; prevalence

Tattooing and body piercing are the worldwide fashion craze of the 1990s. Body piercing, in particular, has become the fastest growing form of body decoration in the modern world, and is widely practised among the young.[1,2] The body decoration craze, however, has occurred following the emergence of bloodborne viruses (BBVs), notably hepatitis B, HIV, and the hepatitis C virus (HCV). Tattooing and body piercing have become potential routes of BBV transmission.

Widespread tattooing and body piercing has also coincided with an unprecedented rise in injecting drug use (IDU) in Australia. Record numbers of deaths have occurred due to overdoses,[3] while those who have injected illicit drugs in the previous year have trebled compared with a decade ago.[4,5] There is a strong association between tattooing and body piercing and injecting drug use, partly due to youth but also to cultural values. While health promotion campaigns have much reduced the risk of infection due to shared needles, tattooing and body piercing remain a major potential source of infection for this group.

Health risks from body piercing

In western countries, HIV has spread primarily through sexual contact, and specifically homosexual contact, and secondarily through injecting drug use. In Australia, 90 per cent of AIDS cases in 1994 occurred among men with a history of homosexual contact.[6] The spread of HIV through the sharing of contaminated needles has been much less prevalent. Reliable estimates are problematic, but about 2 to 5 per cent of injecting drug users are HIV positive compared to up to 27 per cent in parts of the United States of America. The risk of contracting HIV through tattooing or body piercing exists, but it is not believed to be high. In contrast to HIV, the risks of contracting HCV through some form of skin penetration are probably much greater. It has been estimated that there are 210,000 people in Australia with hepatitis C.[7]

The most important risk factor for contracting HCV is injecting drug use. Less common routes of transmission may include receipt of blood or blood products (although blood donor screening has vastly reduced this risk), needle stick injury, tattooing, body piercing, sexual activity and household contact such as blood contact and the sharing of toothbrushes and razors.[8] The epidemiological support for some of these routes of transmission remain inconclusive and it is therefore difficult to provide estimates of the risk of transmission.

Information on recent patterns of HCV transmission in Australia can be gained by examining the data on incident HCV notifications reported to the National Notifiable Diseases Surveillance System. Most incident hepatitis C notifications in 1999 were in the 15 to 29 year age range. Notifications among males outnumbered females by 1.8 to 1 in 1999.[9]

Particular subgroups have a higher risk of BBV infection, notably the prison population. The prevalence of widespread tattooing and body piercing among prison inmates using non-sterile equipment, coupled with high rates of IDU and high turnover among inmates, makes prisons a major area of potential infection.[10,11] Estimates of the prevalence of tattooing among the prison population vary from 51 per cent of females and 57 per cent of males in New South Wales in 1996[10] to 58 per cent of prisoners in Western Australia.[12] In Victoria virtually all male injecting drug users in prison have been found to possess tattoos, with 60 per cent reporting that they acquired a tattoo while in prison.[13] Studies show that the cleaning of tattooing equipment is less common than for injecting equipment, and that tattoo needles are often shared.[12]

Whether tattooing in prison is associated with acquiring HCV remains controversial, due to the overwhelming association of HCV with intravenous drug use. On

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multivariate analysis, Butler and colleagues found no association between HCV infection and tattooing. In a subset of inmates who self-reported no intravenous drug use, HCV infection was associated with greater than 11 tattoos, suggesting that tattooing may cause infections in non-IDUs. Whether these tattoos were received in prison or not was not reported. These results should be viewed with caution as the history of injecting drug use was self-reported by the inmates.

Most studies to date on HCV infection in prisons examine HCV prevalence rather than incidence. No study in Australia has estimated the risk of acquiring HCV via tattooing in prison therefore further studies are required.

Regulation of tattooing and body piercing

Tattooing and body piercing are not nationally regulated; the responsibility lies with each of the States and Territories. Regulation can either be covered by the criminal code or by health acts that regulate providers in regard to the control of infectious diseases. Skin penetration procedures can occur in both health and non-health care settings and some states and territories have established Codes of Practice, usually based on the Australian Standards and National Health and Medical Research Council guidelines, to ensure minimum standards. Victoria and the Australian Capital Territory further require that the premises conducting such procedures be formally registered.

There is no accredited system of apprenticeship for body modification in Australia, nor are there currently any technical courses offered at TAFE colleges for potential practitioners. The Professional Tattooing Association of Australia (PTAA) was established in 1994 to represent the industry; it works with the relevant health authorities to develop Codes of Practice. There is no comparable national association to the PTAA for other forms of body modification.

All States and the Australian Capital Territory have legislation stipulating a minimum age of consent for tattooing, with penalties for non-compliance. The age of consent is generally 18 years, although it is 16 years in New South Wales and 17 years in Queensland (Table 1). It is possible in 3 States for minors to be tattooed with written parental consent, but in Western Australia it must be because of cultural or religious belief. In all States the penalty includes a fine while in 4 States imprisonment is also an option. Tattooing in prison is illegal in all jurisdictions, although enforcement varies considerably.

Method

The National Drug Strategy Household Survey is the sixth in a series of surveys conducted by the Department of Health and Aged Care since 1985. The survey is designed to examine awareness, attitudes and behaviour relating to drug use and drug problems, and to assess changes in these attitudes and behaviours. The 1998 survey was conducted between June and September by the Roy Morgan Research Centre and was based on a stratified multi-stage sample. The sample was designed to provide a random sample of households within each geographical stratum.[14,15]

The survey had three separate but related samples. The first sample was a national random sample, stratified by region, of persons aged 14 years and over living in households. The second sample was collected from households included in the first sample where there was more than 1 person over the age of 14 years; respondents were the youngest persons in the household, other than the respondent included in the first sample. The third sample was collected from different, randomly selected households in the same areas as households in the first sample, but in capital cities only; these respondents were aged between 14 and 39.

The first sample used a personal interview schedule with a self-administered questionnaire for the sensitive drug use sections. The second and third samples used a self-administered questionnaire only. In total, 4,012, 1,983 and 4,035 people participated, representing effective response rates of 54.5 per cent, 48.2 per cent, and 33.5 per cent, respectively. Analyses indicated few statistically significant differences between the samples which allowed them to be pooled for analysis.[15] The samples were weighted to adjust for age, gender, stratum, and household size and type.[13] Of the total sample of 10,340 respondents, 1,200 were aged between 40 and 49 years, 806 between 50 and 59 years, and 1,134 were aged over 60 years.

Statistical tests and confidence intervals were adjusted to take account of the 'effective sample size' by dividing the total number of respondents by a 'mean design effect'.[15] Standard chi square tests were based on the assumption that the dataset was reasonably large, and that the tables were densely populated and well balanced (i.e. has asymptotic properties). When these assumptions were not met exact tests were calculated using Monte Carlo approximation.[16] The exact significance was always reliable, regardless of the size, distribution, sparseness, or balance of the data.

Table 1. Legal restrictions on tattooing in Australia

State/Territory	Age of consent	Maximum penalty
Australian Capital Territory	18 or written parental consent	$1,000 fine
New South Wales	16 or written parental consent	$22,000 fine
Northern Territory	No specific legislation	Not applicable
Queensland	17	$3,000 fine or 6 months imprisonment
South Australia	18	$1,000 fine or 3 months imprisonment
Tasmania	18	$500 fine or 6 months imprisonment
Victoria	18	$1,200 fine
Western Australia	18 or parental consent based on long standing cultural or religious belief	$400 fine, 6 months imprisonment or both

Patterns of body decoration in the population

The most common form of body decoration is ear piercing. Table 2 shows that nearly 1 in 3 of the population aged 14 years and over reported having had their ears pierced at some point in their lives. The prevalence is much higher among women than men; in 1998, 44.4 per cent of women reported having had their ears pierced, compared to less than half that proportion for men. Around 1 in 10 people have had a tattoo at some point in their lives, with more men (11.9%) than women (8.5%) falling into this category. Body piercing is the least common form of body decoration; 6.7 per cent reported undergoing this procedure at some stage in their lives.

The estimates for those who had undergone these procedures in the past year show that nearly 5 per cent of women had their ears pierced, which represents 349,564 women (95% CI: 273,648-425,675). Just under 3 per cent of men reported having acquired a tattoo in the previous 12 months, representing a population estimate of 180,974 men (95% CI: 125,856-236,245). It is also notable that nearly 2 per cent of women reported acquiring a tattoo during that period. Overall, nearly 1 in 3 Australians possess some form of body decoration, with twice as many women falling into the category as men. About 1 in every 16 people undergo some form of body piercing procedure each year.

In addition to marked gender differences in the use of body decoration, there are also significant age variations. The current popularity of tattoos means that the prevalence is much higher among the young (Figure 1). Among young men aged about 20, around 25 per cent reported having a tattoo, compared with 20 per cent of women of the same age. Thereafter, the prevalence of tattooing in the population declines significantly, particularly among those aged in their 50s and 60s, where only around 5 per cent reported a tattoo. Reflecting the prevalence of tattooing among the young, about 1 in 8 younger men said that they had acquired a tattoo in the previous 12 months, compared to 1 in 16 women. Interestingly, there is an upward trend in recent tattooing among older women.

Ear piercing is more common among women than men, but again the age trends suggest that it is becoming increasingly prevalent among younger men. Unlike tattoos, however, Figure 2 shows that ear piecing is widespread among men aged in their late 20s and 30s, as well as among those in their late teens and early 20s. Prevalence is highest among men aged in their mid-20s, where 30 per cent have had their ears pierced. Among younger women, nearly 70 per cent report having undergone the procedure, although the figures are much less for older women.

As with tattooing and ear piercing, body piercing shows the same concentration among the young, notably among younger women. Around 1 in 5 women aged about 20 report

Figure 1. Lifetime prevalence of tattooing, Australia, 1998, by age and sex

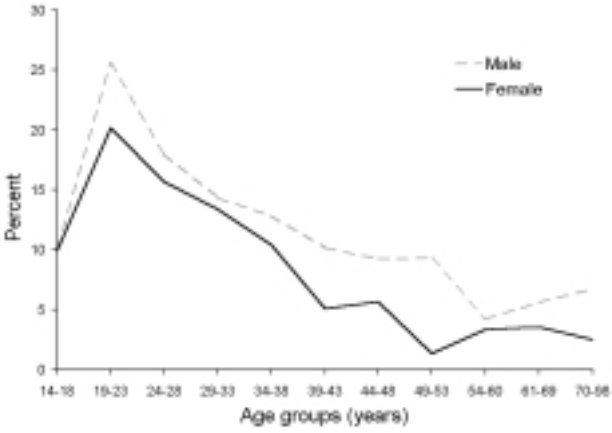

Figure 2. Lifetime prevalence of ear piercing, Australia, 1998, by age and sex

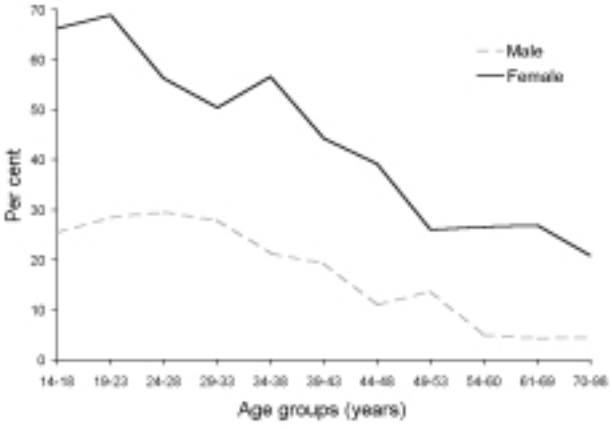

Table 2. Prevalence of body decoration, 1998, by sex

	Lifetime (%)			Past 12 months (%)		
Per cent undergoing	All	Male	Female	All	Male	Female
Tattoo	10.1	11.9	8.5*	2.2	2.6	1.8
Pierced ears	31.5	18.2	44.4*	3.8	2.7	4.8*
Pierced body	6.7	6.5	7.0	1.8	1.3	2.2
At least one of above)	(34.6)	(22.2)	(46.6*)	(6.2)	(5.2)	(7.2*)
(n)	(9,489)	(4,675)	(4,814)	(9,489)	(4,675)	(4,814)

* Males statistically significantly different from females at p <.01, two-tailed test.

Source: 1998 NDS Household Survey.

having had their bodies pierced, a figure that declines to half that proportion among those just 10 years older. Body piercing is less common among younger men, although it is notable that about 1 in 8 younger men reported having undergone the procedure and among men aged in their late 40s and early 50s, the prevalence of body piercing actually exceeded that of women. This may reflect its popularity among older homosexual men.

During the 12 months prior to the survey, more young women than men said that they had engaged in body piercing. About 7 per cent of younger women reported having had their bodies pierced in the past 12 months, as compared to around 3 per cent of males. As with tattooing, the prevalence of this activity declines with age, the major exception being older women, where a small percentage report recent body piercing.

Body decoration and drug use

The survey respondents who reported having a tattoo or having had their bodies pierced were asked if they were under the influence of alcohol or drugs while undergoing the procedure. Table 3 shows that alcohol or drugs affected significant proportions of those acquiring a tattoo at the time of the procedure. Among those who were tattooed in the past 12 months, the figure least likely to be subject to recall bias, about 10 per cent, reported having been influenced by drugs at the time. The same estimate for those undergoing body piercing is slightly less at 7 per cent.

There are significant gender patterns in these findings, with men being much more likely to report being influenced by drugs than women. For example, of those aged in their 20s who said that they were tattooed in the previous 12 months, 7 per cent of women reported being affected by alcohol or drugs at the time, compared to 19 per cent of men. Similarly, among those of the same age group who reported body piercing in the previous 12 months, 2 per cent of women said they were influenced by drugs compared to 26 per cent of men. Although self-regulation among tattooists and body piercers normally precludes a procedure being conducted on a person who is obviously intoxicated or affected by drugs, the evidence suggests that in the case of men this rule is frequently breached.

The survey included measures of whether or not the respondent reported having injected illicit drugs at any point in their lifetime and in the previous 12 months. A total of 2.1 per cent (sample n = 198) reported lifetime injecting drug use, and 0.8 per cent (sample n = 72) said that they had injected an illicit drug in the previous 12 months. Injecting drug users had substantially higher proportions that had undergone tattoo or body piercing compared to the rest of the population, and a significant minority had undergone these procedures in the past year.

Table 4 shows that lifetime injecting drug users record four times the rate of tattooing compared to the general population, and among those who have injected in the past 12 months, more than five times the population rate. Current injecting drug users are also more likely to have engaged in ear and body piercing; nine times more injecting drug users reported having had their bodies pierced in the previous year compared to the general population. The data suggest that recent experience of body decoration may be increasing, particularly in regard to body piercing among injecting drug users. For example, 16 per cent of such users said that they had had their body pierced in the previous 12 months compared with 11 per cent of lifetime injecting drug users and 2 per cent of the general population.

Discussion

Throughout history and across numerous cultures, body decoration has been used for many purposes. Western societies have often viewed it as unhealthy and have usually defined people who adopt it as deviant. Yet body decoration is 'perhaps the oldest art known to human beings, and the most ancient method of expressing personal and communal spiritual beliefs'.[17] In the past 20 years tattooing has experienced a social renaissance, embracing a better educated, more affluent population. By contrast, body piercing practices remain virtually undocumented but like tattooing there has been a noticeable shift in the demographic profile, from being a largely homosexual practice to adoption by middle class heterosexuals of both sexes.[17] This change is reflected in Australia with significantly more young people engaging in body piercing.

Table 3. Alcohol or drug influence during body procedure, 1998

	Drinking alcohol/taking drugs %			
	Yes	No	Don't know/ can't recall	Sample (n)
Tattoo				
Ever	10.9	52.8	36.3	(963)
Past 12 months	10.1	78.3	11.6	(206)
Ear piercing				
Ever	3.6	69.7	26.6	(2,990)
Past 12 months	5.0	75.6	19.3	(357)
Body piercing				
Ever	5.2	45.8	49.1	(638)
Past 12 months	7.1	71.4	21.4	(168)

All associations statistically significant at p <.01, two-tailed test.
Source: 1998 NDS Household Survey.

Table 4. Injecting drug use and body decoration, 1998

Per cent undergoing:	Injecting drug users		
	All	Lifetime	Past 12 months
Tattoo			
Ever	10.2	42.4*	54.2*
Past 12 months	2.2	9.1*	4.3
Ear piercing			
Ever	31.6	57.6*	54.2
Past 12 months	3.8	9.1*	12.5
Body piercing			
Ever	6.8	24.2*	31.8*
Past 12 months	1.8	10.6*	16.7*
Sample (n)	(9,422)	(198)	(72)

* Lifetime and previous 12 months injecting drug users were statistically significantly different from the total population at p <.01, two-tailed test.
Source: 1998 NDS Household Survey.

Although the reasons currently advanced for body decoration vary, three common themes emerge: the importance of pain; the confirmation of identity; and self-stigmatisation. Withstanding pain is important to gender constructions and is seen as a rite of passage into adulthood, particularly for young males. It may signal initiation into a particular gang or social group and demonstrate loyalty. Higher rates of participation in risk behaviours, including criminal behaviour, may partly account for the higher rates of body decoration among men. These activities can be social events and may explain the link between intoxication and body decoration noted above. Body decoration has also been recognised as a major concern in prisons, where self-mutilation is common.

For women, body decoration can symbolise gender rebellion, blurring the boundaries between male and female roles. This may account for its increasing prevalence among younger women, particularly with respect to body piercing. As body decoration is often frowned upon in Western societies, women tend to choose adornments that are not publicly visible which probably accounts for a slightly lower rate of overall participation in body modification practices. Women do exceed men in the rates of ear piercing, but this practice has been widely accepted for women and only recently for men.

Regulatory control of those who provide tattooing and body piercing services is essential to the control of infectious diseases. Infections can occur when needles and other sharp instruments are not thoroughly cleaned and sterilised. But infections can also be transmitted when other materials are not clean or handled and used unhygienically, and when operators, their premises and their sterilisation equipment are not clean. Given that these data indicate increasing prevalence of body decoration practices, particularly among the young, the regulation of the industry is necessary to ensure that body modification does not become a major source of the transmission of bloodborne viruses.

A 1998 review of Australia's response to HCV found that the implementation of infection control programs was not fully developed in even a majority of states and territories. More importantly, few jurisdictions reported that their current programs were fully effective.[9] The necessity for training skin penetration practitioners is exemplified by the 10 per cent of respondents who reported being influenced by alcohol or drugs when they obtained a tattoo and the 8 per cent who reported being under their influence when they engaged in body piercing in the past 12 months. As some of these activities are social events, it is also important that individuals and friends make informed decisions. Education strategies informing young people about the risks in body modification should be a high priority.

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An outbreak of *Salmonella* Typhimurium phage type 29 linked to a noodle restaurant in South Australia

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In January 2001, a cluster of 24 cases of salmonellosis was investigated by the Communicable Disease Control Branch, Department of Human Services, South Australia. Hypothesis generating interviews sought demographic, illness, food purchasing practices, food consumption, social activities, and animal contact information for the 7 day period prior to the onset of symptoms. Eight (7 female, 1 male, age range 11 to 40 years) cases reported eating at an Adelaide noodle restaurant between 17 and 22 December 2000. The predominant symptoms were diarrhoea (100%) with 37.5 per cent reporting bloody diarrhoea, abdominal pain (87.5%), fever (75%) and nausea (75%). The median incubation period from eating at the restaurant to illness was 4 days (range: 2 to 6 days). Where obtained, the median duration of symptoms was 9 days (range 6 to 17 days). Five stool specimens obtained from cases were positive for *Salmonella* Typhimurium phage type 29 and a further 3 stool specimens were positive for *Salmonella* Typhimurium phage type untypable. A polymerase chain reaction based test showed the relatedness of the untypable isolates with the phage type 29 isolates. In addition, there were 3 reports of gastrointestinal illness in restaurant employees. All 3 employees reported the onset of gastrointestinal illness between 17 and 18 December 2000. One symptomatic employee reported working in various food preparation and delivery roles from 18 to 22 December 2000.

With the exception of individual serves of Vietnamese cold rolls (2 patrons); spring rolls; and prawn crackers, heated noodle dishes were consumed by all cases. Of the 3 employees who experienced gastrointestinal illness, all reported eating Vietnamese cold rolls on the day prior to the onset of symptoms. An environmental investigation conducted 3 weeks after the exposures found food handling procedures were satisfactory. However, several environmental concerns related to the maintenance and cleanliness of the premises, adequate hand washing facilities, and the protection of food from external contamination were identified. All environmental and food sampling were negative for *Salmonella* sp.

Although the source for this outbreak was not established, a microbiological and epidemiological investigation identified a link between infection with *Salmonella* Typhimurium phage type 29 and the consumption of prepared food at the restaurant. Additionally, 3 employees experienced gastrointestinal illness following the consumption of prepared food at the restaurant. Of these, 1 employee reported working while symptomatic. The possibility that an infected food handler contaminated foods that required no subsequent cooking (e.g. garnishes) cannot be excluded. However, the employee has not been confirmed as the index case. Likewise, unrecognised cross-contamination in the kitchen may have occurred resulting in a range of contaminated dishes. Nonetheless, this outbreak highlights the need for food handlers to be excluded from the direct handling of foods while experiencing gastrointestinal illness. Further follow-up inspections and corrective action were coordinated by local government with the co-operation of the restaurant owners. No further cases of *Salmonella* Typhimurium phage type 29 infection reported dining at this noodle restaurant.

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An outbreak of *Salmonella* Typhimurium phage type 126 linked to a cake shop in South Australia

Adriana Milazzo,[1] Nick Rose[2]

On 7 March 2001, the Australian Salmonella Reference Centre in Adelaide notified the Communicable Disease Control Branch (CDCB), South Australian Department of Human Services, of 5 cases of infection with *Salmonella* Typhimurium phage type 126. *Salmonella* Typhimurium phage type 126 is an uncommon isolate in South Australia. Between 1993 and 2000 CDCB received only 27 notifications, an average of 3 cases per year.

Hypothesis generating interviews with the initial cases identified the consumption of cakes as a feature in common, in particular, the consumption of custard filled pastries. Cases reported consuming the custard filled pastries at commercially catered functions or purchasing them direct from a cake shop, cafes and delicatessens. A trace-back investigation from the point of purchase to the supplier identified a local Adelaide cake maker. This manufacturer distributes its products to 59 outlets in metropolitan Adelaide, and also supplies commercial caterers. The public is able to purchase direct from the cake maker.

On 14 March a further 5 cases were notified to CDCB. Of the 9 cases (one was excluded from the investigation because it was a secondary case), 6 were female and 3 were male. Ages ranged from 4 to 64 years (median age 32 years). The cases were distributed throughout metropolitan Adelaide. The predominant symptoms were diarrhoea (9 cases, 100%), abdominal pain (9 cases, 100%), headache (8 cases, 89%), fever (7 cases, 78%) and nausea (6 cases, 67%). The median incubation period where known, from consumption of items purchased from the cake shop to onset of symptoms, was 2 days (range 1 to 4 days) and the median duration of illness was 9 days (range 5 to 15 days). One case was hospitalised.

A case control study (9 cases and 27 controls matched by age, sex and postcode of residence) was conducted on 14 March 2001. A case was defined as a person who had an onset of illness between 19 February and 4 March and developed microbiologically confirmed *Salmonella* Typhimurium phage type 126 infection and was not a secondary case. Analysis demonstrated a statistically significant association between consumption of a pastry filled custard tart with strawberries and a jelly glaze, and illness (odds ratio 52.00, 95% confidence interval 3.57—1726.72). In total, 6 (67%) of the 9 cases reported the consumption of a pastry filled custard tart with strawberries and a jelly glaze.

An environmental investigation conducted on 14 March 2001 identified an inadequate level of premise maintenance and sanitation. A paintbrush used to apply the gel coating to the tarts was not cleaned after each day's production but left to sit in an aluminium saucepan until required. Similarly, unused custard from piping bags was returned to the initial custard container and retained for subsequent use. Commercially available ingredients were used in the production of the tarts, and raw eggs and milk were not used in making the custard. Employees had not experienced gastrointestinal illness in the month prior to the environmental investigation. Environmental and food samples were negative for *Salmonella* sp.

The original source of the outbreak remains unknown. An inadequate understanding of the risks associated with time temperature abuse of ready to eat products resulted in poor food handling practices. These practices had the potential to create a reservoir of contamination in the production process and for the cross contamination of subsequent batches. The owners of the cake shop took remedial action in collaboration with local government. One further case of *Salmonella* Typhimurium phage type 126 has been notified and reported the consumption of a sweet pastry linked to the cake shop.

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

Communicable Diseases Surveillance

Presentation of NNDSS data

With the move to a quarterly reporting system in *Communicable Diseases Intelligence*, the summary tables have changed to fall in line with a quarterly report. Table 2 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 4 presents the crude incidence of diseases by State or Territory for the current reporting quarter.

Table 2 now includes the following summary columns: total current quarter data, totals for previous quarter; total for same quarter in previous year; a 5-year mean for the same quarter and the ratio of the current quarter to the mean of the first quarter for the last 5 years.

Notifiable diseases 2001

The Communicable Diseases Network Australia has revised the list of diseases that are reportable to the NNDSS. From 2001, the following diseases are notifiable. All jurisdictions are working towards reporting against the new national list. Dates of first transmission of a dataset consistent with the new list will vary across Australia depending on changes to public health legislation and IT system development. The following new diseases have been added to the NNDSS database: anthrax, Murray Valley encephalitis, Kunjin virus infection, cryptosporidiosis, influenza (laboratory-confirmed), Australian bat lyssavirus infection and invasive pneumococcal disease (laboratory-confirmed). Data on the following diseases will no longer be collected: chancroid infection, hydatid disease, lymphogranuloma venereum, and yersiniosis.

Anthrax is an acute bacterial disease of the skin and rarely an inhalation disease affecting the oropharynx, mediastinum or intestinal tract. Since this is a serious disease and has been developed as a potential biological weapon, the surveillance of anthrax is increasingly important. Anthrax is acquired primarily by contact with livestock and occurred at very low levels in Australia (less than 8 notifications per 10 million per year) between 1917 and 1990.

Arboviruses: From 1996 to 2000, the category ‘Arboviruses: Not Elsewhere Classified (NEC)’ included Murray Valley encephalitis (MVE), Kunjin, Japanese encephalitis (JE), Kokobera and Stratford viruses. From 2001, MVE, Kunjin and JE will be notified separately.

Both MVE and Kunjin viruses are flaviviruses transmitted by mosquitoes.

Murray Valley encephalitis: While only 1 in 1,000 people infected with MVE virus will develop encephalitis, the case fatality rate is about 20 per cent, with a further 40 per cent surviving with residual permanent neurological damage. MVE virus is enzootic in the Kimberley region of Western Australia and the top end of the Northern Territory. Cases occur sporadically in Queensland. Disease outbreaks in more southerly regions of Australia are rare, with the last major outbreak occurring in 1974. The MVE virus is a flavivirus, transmitted by mosquitoes. Avian hosts play an important role in the life cycle of the virus and are useful for sentinel surveillance.

Kunjin. Infection with Kunjin virus occasionally causes encephalitis, but less frequently than the MVE virus. While both viruses share vertebrate and vector hosts, the epidemiology of Kunjin virus differs from that of MVE virus. Kunjin virus appears to be more widely distributed geographically in Australia and is closely related to the West Nile virus.

Cryptosporidiosis is a protozoal infection of the gastrointestinal tract. The main route of transmission is faecal-oral and up to 5 per cent of the human population excretes the oocytes in their faeces. Cryptosporidiosis is a significant infection in people affected by HIV/AIDS.

Continuous monitoring of influenza is a critical public health issue in the 21st century, due to the possibility of rapidly evolving pandemics causing widespread morbidity and mortality, particularly among the elderly and immuno-compromised. National laboratory-based influenza surveillance will link with ASPREN and State-based influenza surveillance schemes.

Australian bat lyssavirus infections are notifiable from this quarter. Two deaths in Queensland (in 1996 and 1998) associated with lyssavirus infection dramatised the risk of this disease for people exposed to bats. A prevalence of lyssavirus infection in bats in southern Queensland of 6 per cent has been reported (McCall, 2000).

Invasive laboratory-confirmed pneumococcal disease will also be notifiable. This will build on existing pneumococcal surveillance in the Northern Territory, New South Wales and Western Australia. These infections will be important to monitor as a new conjugate vaccine is introduced in Australia in 2001.

Highlights for 1st quarter, 2001

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, 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'.*

Figure 9 shows the changes in disease notifications compared with the 5-year first quarter mean. Disease notifications above or below the 5-year mean plus- or minus-two standard deviations are marked with an asterisk. These, and other disease trends are described below.

Bloodborne diseases

There is a continuing increase in the numbers of incident cases of hepatitis B and C being reported to the NNDSS. This may reflect changes in surveillance procedures over the past 5 years, particularly with the introduction of enhanced surveillance for incident hepatitis C virus infections in some jurisdictions.

Gastrointestinal disease

Botulism

A case of infant botulism was reported from Victoria during the first quarter of 2001 (see National Polio Reference Laboratory report p.54, this issue). This is only the fourth case of botulism in Australia since 1996. All cases have been in infants aged less than one year. Infant (or intestinal) botulism cases arise from ingestion of *Clostridium botulinum* spores, which germinate in the intestine. Sources of spores are multiple and include foods such as honey and dust. In this case, a 5-month-old infant was hospitalised after a 3-day history of poor feeding, constipation, ptosis, difficulty in swallowing, weakness and loss of head control. Although there were various environmental exposures, including dust, no source for the child's infection could be determined.

Hepatitis A

There were significantly fewer hepatitis A notifications in the first quarter 2001, than for the same quarter last year and when compared with the 5-year mean for first quarters. This continues a steep decline in the notification rate of hepatitis A in Australia, from a peak of 16.6 per 100,000 in 1997 to a projected rate of 2.0 per 100,000 in 2001.

SLTEC

Increases in the notifications of Shiga-like- and verotoxin producing- *E. coli* infections from South Australia (12 of 16 notifications) increased the notifications of this disease above the 5-year mean. However, this needs to be interpreted with caution, as these infections have not been notifiable in all jurisdictions for all of the past 5 years.

Typhoid

Thirty-three cases of typhoid were reported, including 12 from New South Wales and 10 from Western Australia. Four of the typhoid cases in Western Australia were at a detention centre for unauthorised entrants. Typing data and epidemiological investigations of these cases suggested that all were acquired outside Australia.

Quarantinable diseases

No quarantinable diseases were reported in Australia in the first quarter of 2001.

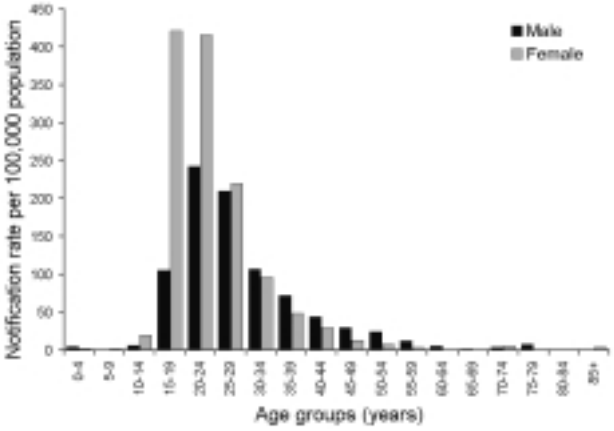
Sexually transmitted infections

STI data in Victoria is currently under review, therefore notifications of chlamydia and syphilis for the first quarter of 2001 should be interpreted with caution. For further information please contact the Communicable Diseases Section of the Victorian Department of Human Services.

Chlamydial infections

A total of 4,696 notifications in the first quarter gave a national notification rate of 98.1/100,000 population. Seventy-six per cent of all notifications were in the 15 to 29 year age range; 70 per cent of the notifications in the 15 to 24 year age range were women (Figure 1). The male to female ratio for chlamydial infections was 0.68:1.

Figure 1. Notification rates for chlamydial infections, first quarter 2001, by age group and sex



Syphilis

There were 278 notifications of syphilis reported (calculated incidence of 5.8/100,000). This is less than the 5-year mean and this quarter's results continue the decline in the notification of this disease in Australia.

Donovanosis

Two cases of donovanosis were reported; one each from the Northern Territory and Queensland.

Other

No chancroid or lymphogranuloma venereum cases were reported. Both these conditions have become very rare in Australia. The last reported case of chancroid was in 1998 and the last case of lymphogranuloma venereum was in 1995. The State and Territory health departments will no longer report these cases to the NNDSS with effect from 2001.

Vaccine preventable diseases

A single case of diphtheria in a 52-year-old man was reported from the Northern Territory. The infection was cutaneous and a toxigenic strain was isolated (*Corynebacterium diphtheriae* var. *mitis*). The patient acquired the disease in East Timor and had an uncertain vaccination history. This is the first case of toxigenic diphtheria reported in Australia since 1993.

Five cases of *Haemophilus influenzae* type b infection were reported in the first quarter 2001. Only 1 case was in an infant (1 month old), 2 cases were in children (4 years and 7 years) and 2 were adults. Vaccination information was not available for any case at a national level.

There were 70 cases of measles reported in the first quarter of 2001. Of these, 54 (77%) of the cases were reported from Victoria. A single outbreak involved 50 laboratory confirmed cases and one epidemiologically linked case, all of whom were associated with a single index case of laboratory confirmed measles. The index case appeared to have acquired measles in India. Four clear waves of transmission were identified. Among the secondary cases, 4 cases aged more than 14 years had a history of vaccination at 12 months. The trends in the incidence of measles is shown in Figure 2.

The Australian Capital Territory, the Northern Territory and South Australia reported no measles cases in this quarter. Indigenous transmission of the disease is now rare in Australia and most cases are among overseas travellers. In this quarter, the number of notifications among young adults aged more than 20 years was greater than the number of notifications among children aged 0 to 4 years (Figure 3).

Reports of pertussis (1,212) were less than the 5-year mean. South Australia had the highest rate (43.8/100,000) and the national rate was 25.4/100,000. The notification rate was highest in the 10 to 14 year age group. The number of notifications of pertussis in children aged less than 10 years of age were similar to those in adults (Figure 4). This continues the trend seen since 1999 and is the probable result of the introduction of a fifth dose of pertussis vaccine in 1995.

A single case of tetanus was reported in an elderly man in Tasmania.

Figure 2. Notification rate of measles, Australia, 1991 to 2001

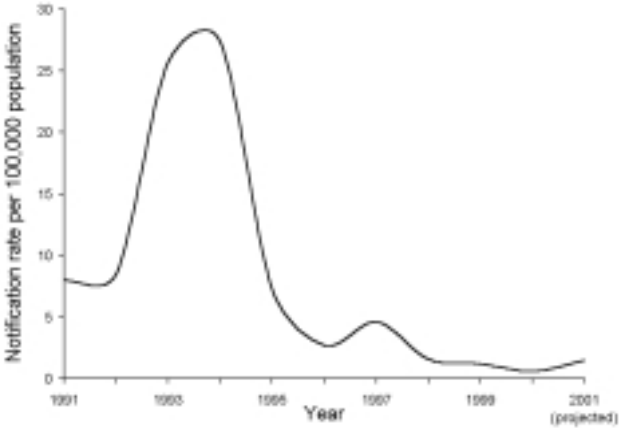


Figure 3. Notification rate of measles, first quarter 2001, by age group and sex

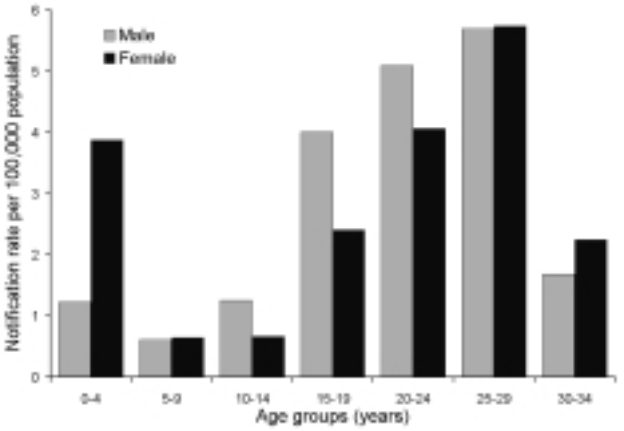
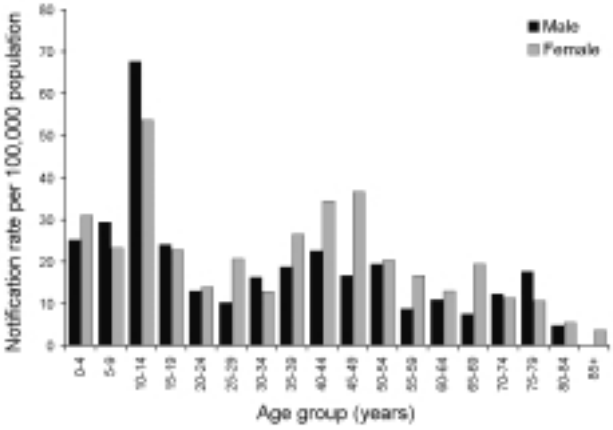


Figure 4. Notification rate of pertussis, first quarter 2001, by age group and sex



Vectorborne diseases

There was a small increase in the number of reports of Barmah Forest virus infection (n=324), particularly from Queensland. This may reflect increased transmission due to heavier than average rainfall in Queensland and northern New South Wales during this first quarter, which may increase vector numbers.

Ross River virus infections were increased in South Australia (n=100) in the lower Murray River and West coast regions in the latter part of 2000 and in the early part of the first quarter 2001. However, overall national notifications (n=1,577) were lower than previous years.

Murray Valley encephalitis (MVE) and Kunjin virus infections became separately notifiable to the NNDSS with effect from January 2001. Prior to this, notifications were reported as 'Arboviruses (NEC)'. Data are not yet available from all States and Territories. A confirmed case of MVE in a 3-year-old male child was reported from Queensland and another in a 60-year-old male from Western Australia. There were no reports of Kunjin virus or Japanese encephalitis virus infection in this quarter.

Zoonoses

There was an increased number of reports of Q fever, especially from Queensland, in the first quarter of 2001. The number of Q fever notifications (169 reports) received nationally was above the range of notifications for this disease in the past 5 years. This may be due to increased awareness and testing for this disease ahead of the implementation of the National Q Fever Management Program.

Other diseases

Legionellosis

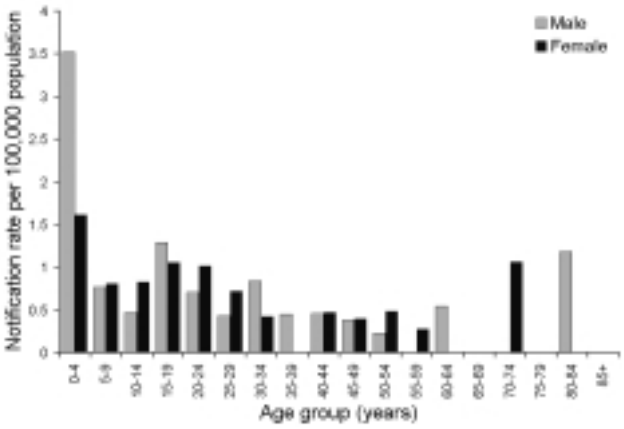
An outbreak of legionellosis in Victoria contributed 38 of the 65 cases reported to the NNDSS in this reporting period. Five of these cases came from a small outbreak in the Melbourne Central Business District in February and March 2001. There were 2 deaths associated with this outbreak.

Of the 38 cases in Victoria, 89 per cent of the cases were confirmed as *Legionella pneumophila* serogroup 1. Nationally, legionellosis was only reported in people aged 20 years or more with 65 per cent of the notifications from those aged 50 years or more. There was a preponderance of males in the reports with a male to female ratio of more than 4:1.

Meningococcal disease

In the first quarter 2001, 128 cases of meningococcal disease were reported in Australia. This was more than the upper limit of the 5-year range for this disease. Of the total 128 cases, 61 (47%) were reported from New South Wales. Figure 5 shows there was a predominance of meningococcal cases among children under 5 years, with a secondary peak of notifications among 15 to 19 years olds. Serogroup typing was available for 54 cases: these were serogroup B (30 cases), serogroup C (23 cases) and 1 case of serotype W135.

Figure 5. Notification rate of meningococcal disease, first quarter, 2001, by age group and sex



Data from the National Neisseria Network indicate there have been 101 laboratory confirmed cases of invasive meningococcal disease in 2001 up to 11 April (Table 1). Of the 101 cases, 54 (53%) were diagnosed by culturing the organism while 47 (47%) cases were diagnosed using non-culture techniques. Of the culture negative diagnoses, 20 (43% of all culture negative cases and 20% of total diagnoses) were diagnosed using PCR, while the remaining 27 (57% of all culture negative cases and 27% of total diagnoses) were confirmed by serology. Subtyping was possible for the majority of culture and PCR confirmed cases, with serogroup B being the most common subtype. Approximately half of all cases were diagnosed in New South Wales (Table 1).

Across Australia, *Neisseria meningitidis* serogroup B was the dominant serogroup. The ratio of serogroup B to serogroup C disease was 1.8:1, 6:1, 2:1, 4:0 and 1:0 in New South Wales, Queensland, South Australia, Western Australia and the Northern Territory respectively. Victoria recorded a preponderance of serogroup C disease with a serogroup B to serogroup C ratio of 8.8:1.

Tuberculosis

Tuberculosis notifications in the first quarter 2001 were significantly down compared with the 5-year mean and lower than the 5-year range (ratio 0.6:1).

Table 1. Data on laboratory confirmed cases of invasive meningococcal disease, Australia, 2001

Culture positive	NSW	Qld	SA	Vic	WA	ACT	NT	Tas	Australia (%of subtotal)
Serogroup B	12	6	2	6	4	0	1	0	31 (57%)
Serogroup C	9	1	1	8	0	0	0	0	19 (35%)
Serogroup W135	1	1	0	0	0	0	0	0	2 (4%)
Serogroup Y	0	1	0	0	0	0	0	0	1 (2%)
Serogroup to follow	0	0	0	0	0	0	0	1	1 (2%)
Subtotal	22	9	3	14	4	0	1	1	54
Culture negative									
Serogroup B	6	4	0	3	1	0	0	0	10 (50%)
Serogroup C	1	0	0	3	0	0	0	0	4 (20%)
Other/ND	1	0	0	1	0	0	0	0	6 (30%)
Subtotal	8	4	0	7	1	0	0	0	20
Subtotal	22	2	1	0	0	2	0	0	27
Total	52	15	4	21	5	2	1	1	101

LabVISE

The Laboratory Virology and Serology (LabVISE) reporting scheme is a passive surveillance scheme of voluntary reports of infectious agents contributed by sentinel laboratories around Australia, to the Commonwealth Department of Health and Aged Care.

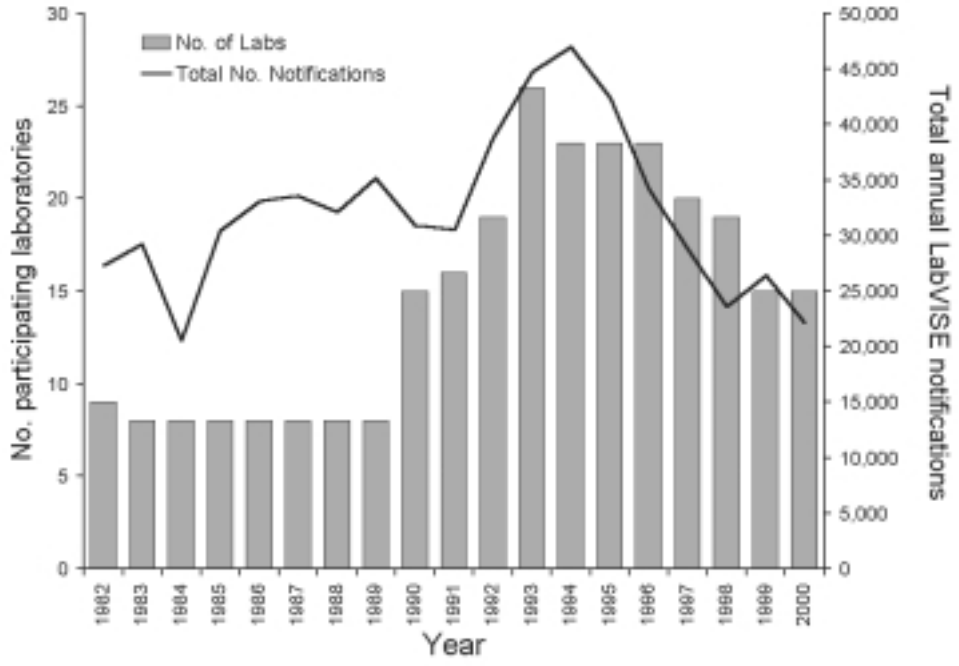
LabVISE provides information on a number of notifiable and non-notifiable viruses and other infectious agents (bacteria, parasites and fungi) of potential public health importance. Data include the demographic characteristics of infected persons that are not reported by other surveillance schemes. The scheme currently holds over 500,000 records collected since 1982.

There has been a decline in the number of notifications and participating laboratories in LabVISE (Figure 6), since a peak in the early 1990s. Currently 11 laboratories reporting on samples from all States and Territories contribute data regularly to LabVISE.

LabVISE has an essential role in providing data for the certification of Australia as a wild polio-free country and in providing data on influenza and isolates for characterisation and formulation of vaccines.

LabVISE has the potential to provide essential subtyping and antibiotic susceptibility data on important pathogens and to monitor new notifiable diseases and exotic emerging pathogens in Australia. The scheme needs to develop a more comprehensive network of laboratories to be representative of the patterns of disease in Australia, to provide more timely and well-documented data to national surveillance systems and to focus data collection on

Figure 6. Trends in reporting and participating laboratories, LabVISE, 1982-2000



pathogens of public health significance. These issues will be addressed in a review of LabVISE in 2001.

Comments on first quarter 2001 LabVISE data

In the first quarter of this year, 2,499 reports were received from 11 laboratories compared with 5,425 reports from 14 in first quarter of 2000. Table 5 shows the totals for the first quarter 2001 compared with the number of isolates in first quarter 2000. Species for which there were no isolates in 2001 were excluded from the list. Contributing laboratories are shown in Table 6.

As in previous years, the majority of pathogens reported to LabVISE were viruses (1,523, 61%, Figure 7). The majority of viral reports belonged to the Herpes virus family (42%), including cytomegalovirus, varicella-zoster virus and Epstein Barr virus. Arboviruses made up 19 per cent of virus reports (n=290). Significantly fewer cases of Ross River virus were reported in the first quarter 2001 (n=228) compared with the first quarter 2000 (n=573). This reflects a smaller number of total reports of Ross River virus to the NNDSS (Table 2) in this quarter (1,393) compared with 2,097 reports of Ross River virus in the first quarter of 2000. This may be due to no reports being received from PathCentre Virology, Perth, during the first quarter 2001 (Table 6).

Among the 976 non-viral pathogens (Figure 8), 454 (46%) were identified as *Chlamydia.* The most commonly identified species was *Chlamydia trachomatis* (442 isolates) accounting for 97 per cent of the total chlamydial reports. There were 163 *Treponema pallidum* reports to LabVISE in this quarter, representing 17 per cent of the non-viral results.

Figure 7. Viral infections detected, first quarter 2001, LabVISE

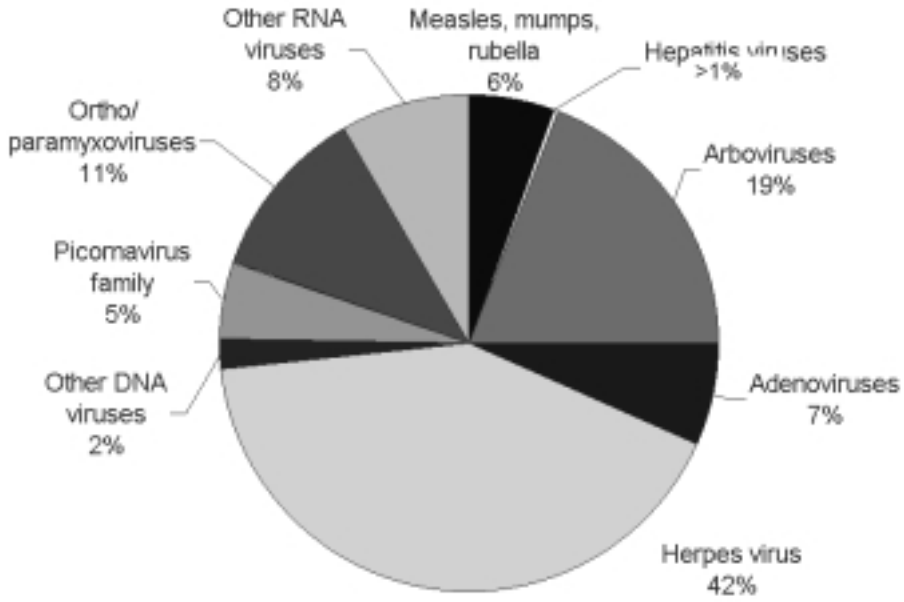
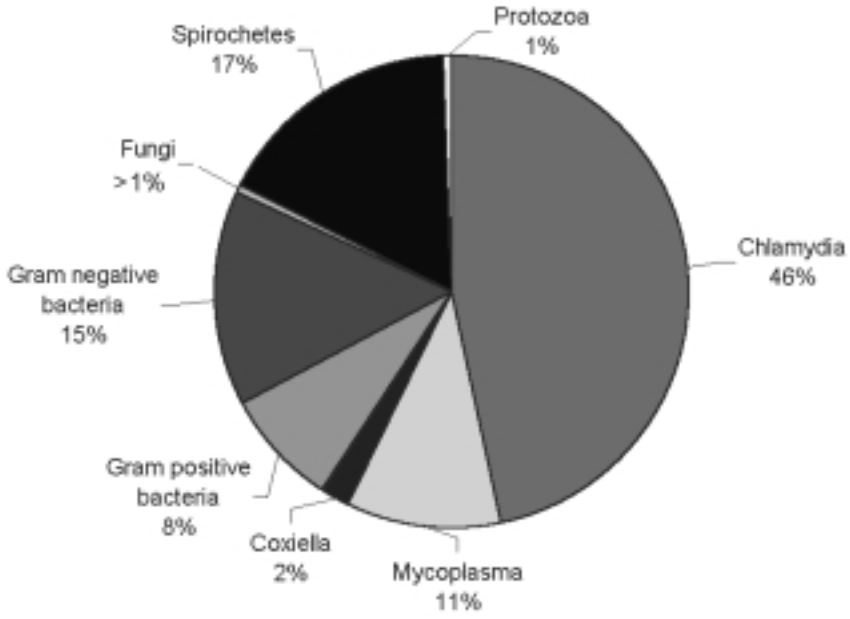


Figure 8. Non-viral isolates, first quarter 2001, LabVISE



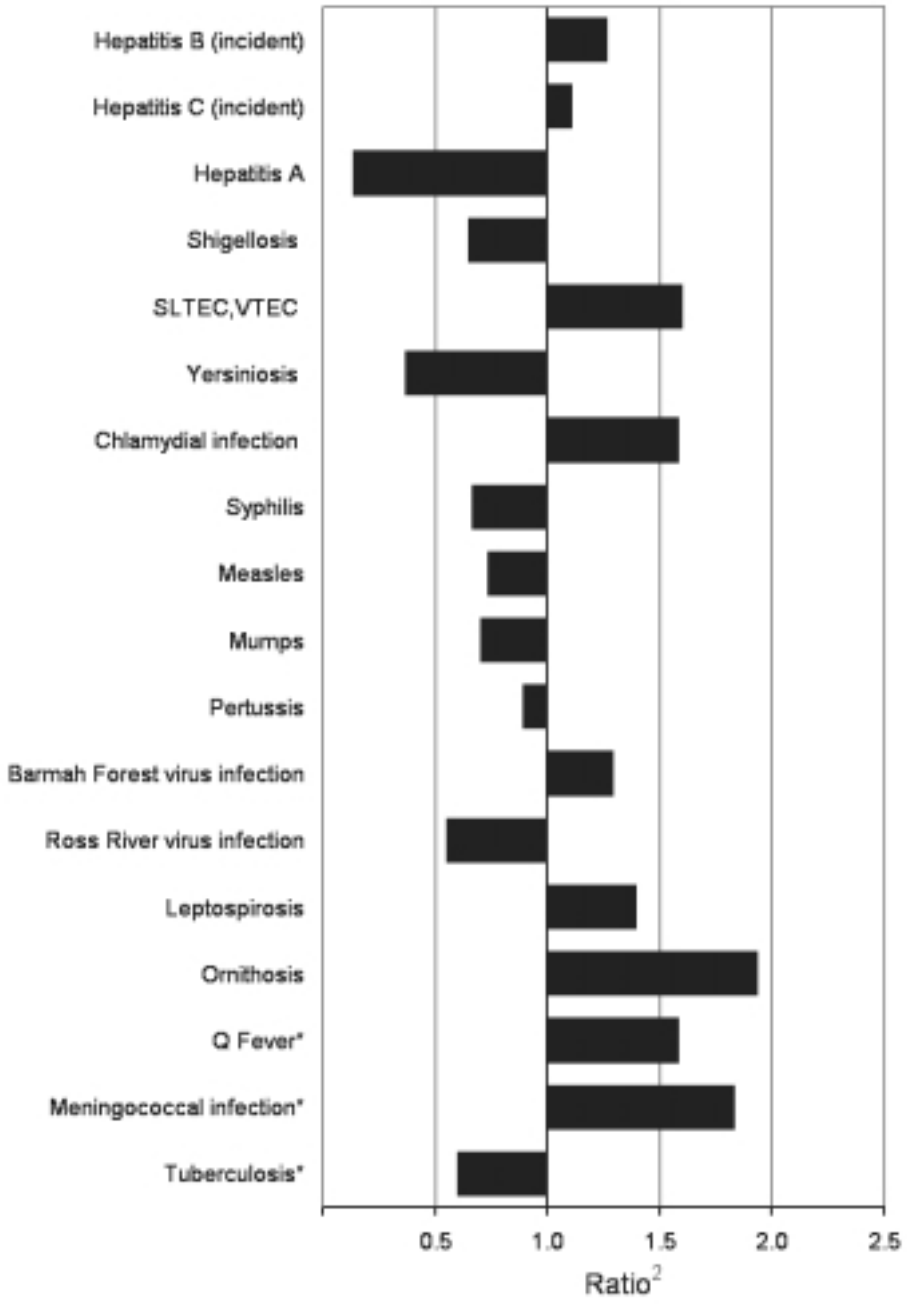
Tables

There were 22,741notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2001 (Table 2). Figure 9 illustrates, for selected diseases, the first quarter 2001 totals as ratios to the mean of the first quarters for the previous 5 years. A summary of diseases currently being reported by each jurisdiction is provided in Table 3. The crude incidence of diseases per 100,000 population for each State or Territory is presented in Table 4.

There were 2,499 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2001 (Tables 5 and 6).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 1-4 to 9-12, ending 25 March 2001, are included in this issue of *Communicable Diseases Intelligence* (Table 7).

Figure 9. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2001 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 January to March data for the previous five years

* Notifications above or below the 5-year mean plus- or minus- two standard deviations

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2001, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2001[1]	Total 4th quarter 2000[1]	Total 1st quarter 2000[1]	Last 5 years mean 1st quarter	Ratio*
Bloodborne													
Hepatitis B (incident)	0	25	2	6	24	4	29	6	96	87	89	76	1.3
Hepatitis B (unspecified)[2]	5	870	NDR	40	52	13	441	66	1,487	2,292	2,020	1,786	0.8
Hepatitis C (incident)	4	29	0	0	29	1	14	5	82	115	160	74	1.1
Hepatitis C (unspecified)[2]	45	1,610	57	858	191	105	1,323	120	4,309	4,615	5,855	4,662	0.9
Hepatitis D	0	4	0	1	0	0	0	0	5	12	4	4	1.3
Gastrointestinal													
Botulism	0	0	0	0	0	0	1	0	1	0	0	0	0.0
Campylobacterosis[3]	94	-	67	866	481	138	1,228	520	3,394	3,843	3,198	3,086	1.1
Cryptosporidiosis	5	NDR	112	101	19	0	NDR	18	255	NDR	NDR	n/a	n/a
Haemolytic Uraemic Syndrome	0	1	0	0	0	0	1	0	2	8	5	n/a	n/a
Hepatitis A	2	29	6	24	3	3	22	7	96	133	305	704	0.1
Hepatitis E	0	0	0	0	0	0	0	1	1	1	0	1	0.0
Listeriosis	0	6	0	7	1	1	6	0	21	15	26	21	1.0
Salmonellosis	29	514	136	705	177	71	343	205	2,180	1,507	2,087	2,466	1.0
Shigellosis[3]	1	-	36	37	10	2	24	6	116	128	125	184	0.6
SLTEC,VTEC[4]	0	0	0	1	12	0	3	0	16	6	17	n/a	n/a
Typhoid	0	12	0	3	0	1	7	10	33	14	22	28	1.2
Quarantinable													
Cholera	0	0	0	0	0	0	0	0	0	0	1	1	n/a
Plague	0	0	0	0	0	0	0	0	0	0	0	0	n/a
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	n/a
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	n/a
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	n/a
Sexually transmissible[‡]													
Chlamydial infection[5]	71	961	304	1,398	628	101	795	438	4,696	4,661	4,283	2,982	1.6
Donovanosis	0	0	1	1	NDR	0	0	0	2	2	5	9	0.2
Gonococcal infection[6]	6	261	311	281	104	4	227	263	1,457	1,239	1,654	1,335	1.1
Syphilis[7]	2	185	39	36	0	2	0	14	278	490	430	419	0.7

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2001, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2001[1]	Total 4th quarter 2000[1]	Total 1st quarter 2000[1]	Last 5 years mean 1st quarter	Ratio*
Vaccine preventable													
Diphtheria	0	0	1	0	0	0	0	0	1	0	0	0	0.0
Haemophilus influenzae type b	0	0	1	2	0	0	2	0	5	9	3	9	0.6
Influenza	2	NDR	7	0	NDR	0	NDR	3	12	NDR	NDR	n/a	n/a
Measles	0	10	0	4	0	1	54	1	70	26	28	95	0.7
Mumps	1	4	0	0	4	0	17	5	31	35	46	44	0.7
Pertussis	20	638	6	219	164	5	138	22	1,217	1,841	939	1,367	0.9
Invasive pneumococcal disease	0	NDR	16	57	NDR	0	NDR	13	87	NDR	NDR	n/a	n/a
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella[8]	0	22	0	11	2	0	18	2	55	140	45	276	0.2
Tetanus	0	0	0	0	0	1	0	0	1	3	2	2	0.5
Vectorborne													
Arbovirus infection NEC	1	0	2	1	0	0	5	0	9	2	27	27	0.3
Barmah Forest virus infection	1	67	21	198	5	0	9	23	324	172	184	252	1.3
Dengue	1	6	17	7	0	0	1	1	33	10	153	103	0.3
Japanese encephalitis	0	-	-	0	-	0	0	0	0	-	-	n/a	n/a
Kunjin virus infection	0	-	-	0	-	0	0	0	0	-	-	n/a	n/a
Malaria	9	41	14	115	6	1	30	14	230	182	273	260	0.9
Murray Valley encephalitis	0	-	-	1	-	0	0	1	2	-	-	n/a	n/a
Ross River virus infection	5	244	181	707	100	9	262	69	1,577	515	2,044	2,874	0.5
Zoonoses													
Anthrax	0	0	0	0	NDR	0	0	0	0	NDR	NDR	n/a	n/a
Australian bat lyssavirus	0	0	0	0	NDR	0	0	0	0	NDR	NDR	n/a	n/a
Brucellosis	0	0	0	4	1	0	1	0	6	8	5	9	0.8
Leptospirosis	0	15	0	44	0	1	10	0	70	63	66	61	1.4
Other lyssavirus (NEC)	0	0	0	0	NDR	0	0	0	0	NDR	NDR	n/a	n/a
Ornithosis	0	NDR	0	NDR	3	0	24	2	29	37	17	15	1.9
Q fever	0	26	0	133	1	0	6	3	169	123	136	131	1.6

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2001, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2001[1]	Total 4th quarter 2000[1]	Total 1st quarter 2000[1]	Last 5 years mean 1st quarter	Ratio*
Other													
Legionellosis	0	9	1	11	2	0	38	4	65	88	85	65	1.0
Leprosy	0	1	0	0	0	0	0	0	1	1	0	3	0.3
Meningococcal infection	2	61	2	15	4	1	31	12	128	169	93	71	1.8
Non-TB mycobacteria	1	8	0	32	0	2	0	0	43	75	151		
Tuberculosis	0	55	2	14	0	0	84	8	163	263	280	272	0.6
Total	307	5,714	1,342	5,963	2,024	467	5,196	1,862	22,741	22,945	24,886	23,858	1.0

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.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

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

NDR No data received. Only notifiable from January 2001. Data are not yet available.

* Ratio = ratio to current month total to mean of last 5 years calculated as described above.

[‡] Sexually transmitted infections data is currently under review in Victoria, therefore notifications of chlamydia and syphilis for the 1ST quarter of 2001 should be interpreted with caution.

Table 3. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:*
Bloodborne	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions except NT
Hepatitis C (incident)	All jurisdictions
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions except NSW & Victoria
Haemolytic Uraemic Syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions except NSW
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions
Vaccine preventable	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions except NSW, SA & Vic
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease	All jurisdictions except NSW, SA & Vic
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne	
Arbovirus infection NEC	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions except NSW, NT, SA
Kunjin	All jurisdictions except NSW, NT, SA
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions except NSW, NT, SA
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions except SA
Australian Bat lyssavirus	All jurisdictions except SA
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions except NSW and Qld
Other lyssaviruses (NEC)	All jurisdictions except SA
Q Fever	All jurisdictions
Other	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Non-TB mycobacteria	All jurisdictions
Tuberculosis	All jurisdictions

* Jurisdictions not yet reporting on diseases either because legislation has not yet made some diseases notifiable in that jurisdiction or data are not yet being reported to the Commonwealth

Table 4. Crude incidence of diseases by State or Territory, 1 January to 31 March 2001. (Rate per 100,000 population)

Disease[1]	State or Territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne									
Hepatitis B (incident)	0.0	1.5	4.1	0.7	6.4	3.4	2.4	1.3	2.0
Hepatitis B (unspecified)[2]	6.4	53.8	NDR	4.5	13.9	11.1	37.0	14.0	31.0
Hepatitis C (incident)	5.1	1.8	0.0	0.0	7.7	0.9	1.2	1.1	1.7
Hepatitis C (unspecified)[2]	57.3	99.6	116.6	96.2	51.0	89.3	111.0	25.5	90.0
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Gastrointestinal									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Campylobacterosis[3]	119.7	-	137.1	97.1	128.5	117.4	103.1	110.4	107.0
Cryptosporidiosis	6.4	NDR	229.2	11.3	5.1	0.0	NDR	3.8	13.2
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Hepatitis A	2.5	1.8	12.3	2.7	0.8	2.6	1.8	1.5	2.0
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Listeriosis	0.0	0.4	0.0	0.8	0.3	0.9	0.5	0.0	0.4
Salmonellosis	36.9	31.8	278.3	79.1	47.3	60.4	28.8	43.5	45.5
Shigellosis[3]	1.3	-	73.7	4.1	2.7	1.7	2.0	1.3	3.7
SLTEC,VTEC[4]	0.0	0.0	0.0	0.1	3.2	0.0	0.3	0.0	0.4
Typhoid	0.0	0.7	0.0	0.3	0.0	0.9	0.6	2.1	0.7
Quarantinable									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	n/a
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	n/a
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	n/a
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	n/a
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	n/a
Sexually transmissible									
Chlamydial infection[5]	90.4	59.5	622.1	156.8	167.7	85.9	66.7	93.0	98.1
Donovanosis	0.0	0.0	2.0	0.1	NDR	0.0	0.0	0.0	0.0
Gonococcal infection[6]	7.6	16.2	636.4	31.5	27.8	3.4	19.1	55.8	30.4
Syphilis[7]	2.5	11.4	79.8	4.0	0.0	1.7	0.0	3.0	5.8
Vaccine preventable									
Diphtheria	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	2.0	0.2	0.0	0.0	0.2	0.0	0.1
Influenza	2.5	NDR	14.3	0.0	NDR	0.0	NDR	0.6	0.6
Measles	0.0	0.6	0.0	0.4	0.0	0.9	4.5	0.2	1.5
Mumps	1.3	0.2	0.0	0.0	1.1	0.0	1.4	1.1	0.6
Pertussis	25.5	39.5	12.3	24.6	43.8	4.3	11.6	4.7	25.3
Invasive pneumococcal disease	0.0	NDR	32.7	6.4	NDR	0.0	NDR	2.8	4.4
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[8]	0.0	1.4	0.0	1.2	0.5	0.0	1.5	0.4	1.1
Tetanus	0.0	0.0	0.0	0.0	0.0	0.9	0.0	0.0	0.0
Vectorborne									
Arbovirus infection NEC	1.3	0.0	4.1	0.1	0.0	0.0	0.4	0.0	0.2
Barmah Forest virus infection	1.3	4.1	43.0	22.2	1.3	0.0	0.8	4.9	6.8
Dengue	1.3	0.4	34.8	0.8	0.0	0.0	0.1	0.2	0.7
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	11.5	2.5	28.6	12.9	1.6	0.9	2.5	3.0	4.8
Murray Valley encephalitis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0
Ross River virus infection	6.4	15.1	370.4	79.3	26.7	7.7	22.0	14.7	32.9

Table 4. Crude incidence of diseases by State or Territory, 1 January to 31 March 2001. (Rate per 100,000 population)

Disease[1]	State or Territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	NDR	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	NDR	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.4	0.3	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.9	0.0	4.9	0.0	0.9	0.8	0.0	1.5
Other lyssavirus (NEC)*	0.0	0.0	0.0	0.0	NDR	0.0	0.0	0.0	0.0
Ornithosis	0.0	NDR	0.0	NDR	0.8	0.0	2.0	0.4	1.3
Q fever	0.0	1.6	0.0	14.9	0.3	0.0	0.5	0.6	3.5
Other									
Legionellosis	0.0	0.6	2.0	1.2	0.5	0.0	3.2	0.8	1.4
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	2.5	3.8	4.1	1.7	1.1	0.9	2.6	2.5	2.7
Non-TB mycobacteria	1.3	0.5	0.0	3.6	0.0	1.7	0.0	0.0	2.2
Tuberculosis	0.0	3.4	4.1	1.6	0.0	0.0	7.1	1.7	3.4

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

NDR No data received (Table 3).

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 January to 31 March 2001, and total reports for the year[2]

	State or Territory[1]								This period 2001	This period 2000	Year to date 2001[3]	Year to date 2000
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	-	-	-	-	-	-	69	1	70	12	70	12
Mumps virus	-	-	-	-	-	-	4	-	4	23	4	23
Rubella virus	-	2	-	7	-	-	1	-	10	13	10	13
Hepatitis viruses												
Hepatitis A virus	-	-	-	5	-	-	-	-	5	53	5	53
Hepatitis D virus	-	-	-	1	-	-	-	-	1	1	1	1
Arboviruses												
Ross River virus	-	7	61	107	34	-	17	2	228	573	228	573
Barmah Forest virus	-	3	2	54	-	-	-	-	59	63	59	63
Flavivirus (unspecified)	-	-	-	1	-	-	2	-	3	31	3	31
Adenoviruses												
Adenovirus type 3	-	-	-	-	-	-	2	-	2	9	2	9
Adenovirus type 7	-	-	-	-	-	-	2	-	2	2	2	2
Adenovirus type 8	-	-	-	-	-	-	1	-	1	-	1	-
Adenovirus type 37	-	-	-	-	-	-	1	-	1	2	1	2
Adenovirus not typed/pending	-	17	-	7	42	-	27	5	98	283	98	283
Herpes viruses												
Cytomegalovirus	-	15	3	26	67	6	55	4	176	318	176	318
Varicella-zoster virus	2	33	4	90	11	1	102	5	248	424	248	424
Epstein-Barr virus	1	8	4	72	65	3	33	21	207	543	207	543
Other DNA viruses												
Parvovirus	-	1	-	19	5	-	5	-	30	94	30	94
Picornavirus family												
Coxsackievirus A9	-	1	-	-	-	-	-	-	1	2	1	2
Coxsackievirus B3	-	-	-	-	-	-	1	-	1	-	1	-
Coxsackievirus B4	-	-	-	-	-	-	4	-	4	-	4	-
Echovirus type 9	-	2	-	-	-	-	-	-	2	3	2	3
Echovirus type 33	-	1	-	-	-	-	-	-	1	1	1	1
Poliovirus type 1 (uncharacterised)	-	3	-	-	-	-	-	-	3	3	3	3
Poliovirus type 2 (uncharacterised)	-	3	-	-	-	-	-	-	3	2	3	2
Rhinovirus (all types)	2	22	-	2	1	-	1	-	28	96	28	96
Enterovirus type 71 (BCR)	-	7	-	-	-	-	-	-	7	-	7	-
Enterovirus not typed/pending	-	13	-	4	-	6	6	-	29	300	29	300
Ortho/paramyxoviruses												
Influenza A virus	2	5	-	1	48	-	1	1	58	196	58	196
Influenza B virus	1	1	-	1	9	-	2	2	16	27	16	27
Parainfluenza virus type 1	-	1	-	-	3	-	-	-	4	50	4	50
Parainfluenza virus type 2	-	-	-	-	2	-	-	1	3	6	3	6
Parainfluenza virus type 3	1	10	-	2	16	-	2	16	47	71	47	71
Respiratory syncytial virus	-	16	-	8	7	4	4	4	43	253	43	253
Other RNA viruses												
Rotavirus	1	23	-	-	28	1	19	10	82	131	82	131
Norwalk agent	-	-	-	-	-	-	46	-	46	1	46	1

Table 5 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 January to 31 March 2001, and total reports for the year[2]

	State or Territory[1]								This period 2001	This period 2000	Year to date 2001[3]	Year to date 2000
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	11	121	30	171	69	5	30	5	442	878	442	878
Chlamydia psittaci	-	-	-	-	-	-	12	-	12	25	12	25
Chlamydia species	-	1	-	-	-	-	-	-	1	3	1	3
Mycoplasma pneumoniae	-	8	1	60	12	-	19	3	103	149	103	149
Mycoplasma hominis	-	1	-	-	-	-	-	-	1	-	1	-
Coxiella burnetii (Q fever)	-	-	-	15	1	-	2	-	18	24	18	24
Streptococcus group A	-	3	13	41	-	-	22	-	79	109	79	109
Yersinia enterocolitica	-	1	-	-	-	-	1	-	2	5	2	5
Bordetella pertussis	-	18	-	33	24	2	63	-	140	165	140	165
Legionella pneumophila	-	-	-	-	-	-	3	-	3	3	3	3
Cryptococcus species	-	-	-	2	1	-	-	-	3	1	3	1
Leptospira species	-	-	1	2	1	-	-	-	4	9	4	9
Treponema pallidum	-	3	53	70	37	-	-	-	163	130	163	130
Entamoeba histolytica	-	-	-	-	-	-	1	-	1	8	1	8
Toxoplasma gondii	-	-	-	-	1	-	3	-	4	4	4	4
Total	21	350	172	801	484	28	563	80	2,499	5,099	2,499	5,099

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. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 January to 31 March 2001[1]

State or Territory	Laboratory	January 2001	February 2001	March 2001	Total this period[2]
Australian Capital Territory	The Canberra Hospital	-	-	-	-
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	121	99	22	242
	New Children's Hospital, Westmead	26	-	-	26
New South Wales	Repatriation General Hospital, Concord	-	-	-	-
	Royal Prince Alfred Hospital, Camperdown	24	28	5	57
	South West Area Pathology Service, Liverpool	-	-	-	-
Queensland	Queensland Medical Laboratory, West End	525	280	199	1,004
	Townsville General Hospital	5	-	-	5
South Australia	Institute of Medical and Veterinary Science, Adelaide	484			484
Tasmania	Northern Tasmanian Pathology Service, Launceston	11	9	1	21
	Royal Hobart Hospital, Hobart	-	-	-	-
Victoria	Monash Medical Centre, Melbourne	-	-	-	-
	Royal Children's Hospital, Melbourne	105	84	28	217
	Victorian Infectious Diseases Reference Laboratory, Fairfield	156	158	35	349
Western Australia	PathCentre Virology, Perth	-	-	-	-
	Princess Margaret Hospital, Perth	32	14	8	54
	Western Diagnostic Pathology	-	40	-	40
Total		1,489	712	298	2,499

1. The complete list of laboratories reporting for the 12 months, January to December 2001, will appear in every report 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 7. Australian Sentinel Practice Research Network reports, weeks 1-4 to 9-12, 2001

Week number	1-4		5-8		9-12	
Week ending on	28 January 2001		25 February 2001		25 March 2001	
Doctors reporting	220		327		199	
Total encounters	23,901		36,712		23,622	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	22	0.9	32	0.9	47	2.0
Influenza with culture	0	0.0	1	0.0	3	0.1
Chickenpox	41	1.7	47	1.3	27	1.1
Shingles	35	1.5	45	1.2	32	1.4

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. The system coordinates the national surveillance of more than 60 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia 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 *quarterly. 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 2001. Communicable Diseases Intelligence *reports the consultation rates for 4 of these. For further information, see* Commun Dis Intell *2000;24:7-8.*

Additional Reports

Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme.

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented.

Reporting period 1 July to 30 September 2000

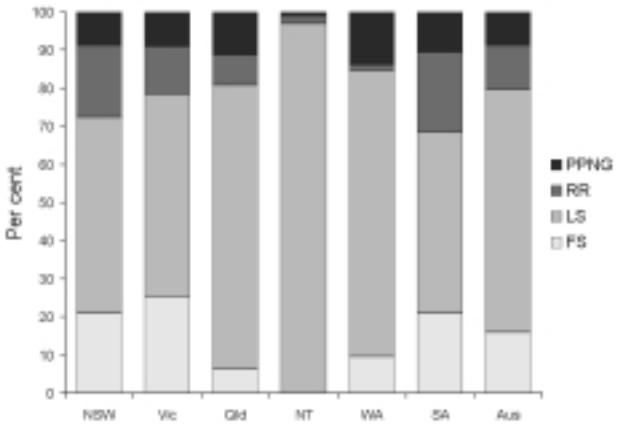
The Australian Gonococcal Surveillance Programme (AGSP) laboratories examined a total of 794 isolates in this quarter, slightly less than the 859 available in this period in 1999. About 33 per cent of this total was from New South Wales, 25 per cent from Victoria, 17 per cent from Queensland, 13 per cent from the Northern Territory, 9 per cent from Western Australia and 2.5 per cent from South Australia. There were few isolates from other centres.

Penicillins

Figure 10 shows the proportions of gonococci fully sensitive (MIC 0.03 mg/L), less sensitive (MIC 0.06 – 1 mg/L), relatively resistant (MIC 1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by State and Territory. A high proportion of PPNG and relatively resistant strains fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

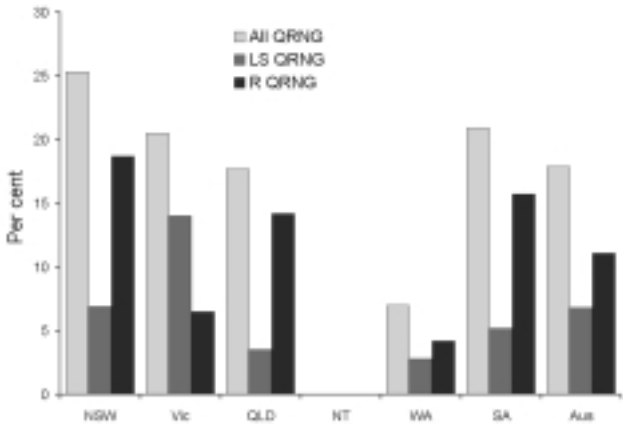
About 20 per cent of all isolates were penicillin resistant by one or more mechanisms – 8.8 per cent PPNG and 11.6 per cent by chromosomal mechanisms (CMRNG). The penicillin-resistant isolates comprised about 31 per cent of all isolates in South Australia, about 27 per cent in New South Wales, 21 per cent in Victoria, 19 per cent in Queensland and 15 per cent in Western Australia. In the

Figure 10. Gonococci isolated, Australia, 1 July to 30 September 2000, by penicillin susceptibility and region



FS fully sensitive to penicillin, MIC 0.03 mg/L
LS less sensitive to penicillin, MIC 0.06 – 0.5 mg/L
RR relatively resistant to penicillin, MIC 1 mg/L
PPNGpenicillinase producing *Neisseria gonorrhoeae*

Figure 11. Quinolone-resistant *N. gonorrhoeae*, Australia, 1 July to 30 September 2000, by region



LS QRNG = Ciprofloxacin MICs 0.06 – 0.5 mg/L
R QRNG = Ciprofloxacin MICs 1 mg/L

Northern Territory 3 isolates out of 100 examined were penicillin resistant – 1 PPNG and 2 CMRNG.

The number of PPNG isolated in Australia (70) was higher in this quarter than in the corresponding period in 1999 (56). Numerically most PPNG were found in New South Wales (23), Victoria (18) and Queensland (16) whereas the highest proportion of PPNG was found in isolates from Western Australia (14%). In Queensland, Victoria, New South Wales and South Australia the proportion of PPNG ranged between 9 and 11 per cent of isolates examined. A single PPNG was present in the Northern Territory. Acquisition data on PPNG indicated local infection with these strains was occurring throughout Australia. South East Asian countries remained the main source of external acquisition.

Once again more isolates (92) were resistant to the penicillins by separate and distinct chromosomal mechanisms. These CMRNG were especially prominent in New South Wales where 49 such isolates were detected. Victoria (25) and Queensland (11) were also prominent sources of CMRNG. Two strains of this type were present in the Northern Territory.

Ceftriaxone and spectinomycin

Most isolates in Australia were again susceptible to these injectable agents. A small number of strains exhibited decreased ceftriaxone susceptibility.

Quinolone antibiotics

Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06 – 0.5 mg/L) or resistant (MIC 1 mg/L) groups. The distribution of QRNG in Australia in this quarter is shown in Figure 11.

The total number (142) and proportion (19.8%) of all QRNG was again high and very similar to numbers and proportions seen in the corresponding quarter of 1999 (152 isolates, 18%). In the current quarter, the QRNG were widely dispersed and were present in all centres except the Northern Territory. High rates were maintained in New South Wales (26%), Victoria (21%) South Australia (21%) and Queensland (17%). A lower proportion of Western Australian isolates (7%) was QRNG.

Forty-nine of the New South Wales, 13 of the Victorian and 20 Queensland QRNG exhibited high level resistance (MIC ciprofloxacin 1 mg/L) and 3 higher level QRNG were also seen in South Australia and Western Australia. MICs ranged up to 16mg/L.

A significant shift occurred in New South Wales in this quarter insofar as the proportion of less sensitive strains decreased and fully resistant isolates acquired through local contact predominated. In Victoria about 60 per cent of the QRNG were still in the 'less sensitive' MIC range 0.06 – 0.5 mg/L and were found almost exclusively in males. QRNG were increasingly acquired through local contact but also from overseas from such diverse sources as Bahrain, Cambodia, China, Madagascar, Malaysia, the Philippines, Thailand, the United Kingdom and Vietnam.

High level tetracycline resistance (TRNG)

The number (69) and proportion (8.7%) of TRNG detected was less than in the corresponding period of 1999 (85, 10%). TRNG represented 21 per cent of isolates from Queensland, 10 per cent from Western Australia, 8 per cent from Victoria and 5 per cent from New South Wales and South Australia. A single TRNG was found in the Northern Territory. Most TRNG were imported into Australia from Cambodia, Indonesia, Thailand and Vietnam.

Reference

1. Anonymous. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Rotavirus Surveillance

The National Rotavirus Reference Centre (NRRC) undertakes surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children throughout Australia.

There are currently fourteen laboratories contributing data and rotavirus specimens for the characterisation of representative rotavirus serotypes.

The NRRC is happy to give and receive notifications of rotavirus outbreaks Australia-wide. The NRRC can be contacted at the Department of Gastroenterology and Clinical Nutrition, Royal Children's Hospital, Flemington Road, Parkville, Victoria 3052. Telephone: (03) 9345 5069, Facsimile: (03) 9345 6240,
Email: masendyp@cryptic.rch.unimelb.edu.au. For more information see Commun Dis Intell *2000;24:10.*

June 2000 to April 2001

The 2000 rotavirus season of most Australian collaborating centres appears to have come and gone and the task of completing the rotavirus serotyping is well under away. Molecular and serological serotyping techniques have been employed to serotype rotavirus positive specimens received Australia-wide. Serotype G3 rotaviruses appeared sporadically in Australia during a 4-year Australia-wide epidemiological survey between 1993-1996,[1] however none have been identified in any of the centres for the sampling period, June 2000 to December 2000. The Serotype G1 rotaviruses have been detected in all centres studied.

Serotype G4 viruses were found in the following 9 centres: Brisbane, Melbourne, Adelaide, Perth, Gove, Narrabri, Horsham, Darwin and Sydney. The G4 viruses were the most prevalent serotype (57%) in specimens received from The Prince of Wales Hospital in south-east Sydney. G4 viruses were also identified from The Children's Hospital at Westmead in Sydney's west (21%), but serotype G2 specimens were found to be the most prevalent serotype (45.6%) in this location. Serotype G2 viruses from Sydney's south-east were low in comparison (7.7%).

This unusual serotype distribution of the G2 and G4 viruses in Sydney suggests different serotypes can exhibit different distribution within a relatively small geographical area. G2 rotaviruses have also been detected in Melbourne, Hobart, Adelaide, Perth, Gove, Narrabri, Horsham and Darwin. Serotype G9 viruses which were identified for the first time in Australia in 1997, appeared in Sydney, Perth, Melbourne and Horsham, with G9 the most prevalent serotype in Horsham (72.7%) for the sampling period June 2000 to January 2001.

Rotavirus collection continues, and the National Rotavirus Reference Centre welcomes any notifications of rotavirus outbreaks.

References

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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 3 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 between 1 October and 31 December 2000, as reported to 31 March 2001, are included in this issue of Communicable Diseases Intelligence *(Tables 8 and 9).*

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2000, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2000	This period 1999	Year to date 2000	Year to date 1999
HIV diagnoses	Female	0	2	1	2	0	0	5	0	10	20	73	75
	Male	0	52	0	25	3	0	31	0	111	161	620	656
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total[1]	0	54	1	27	3	0	36	0	121	183	695	733
AIDS diagnoses	Female	0	2	0	0	0	0	0	0	2	4	20	19
	Male	0	22	1	7	2	0	12	0	44	38	187	156
	Total[1]	0	24	1	7	2	0	12	0	46	42	207	176
AIDS deaths	Female	0	0	0	0	1	0	0	0	1	0	7	4
	Male	0	15	0	1	2	0	7	0	25	29	115	115
	Total[1]	0	15	0	1	3	0	7	0	26	29	122	120

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

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2001, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	28	634	10	163	63	5	228	119	1,250
	Male	228	11,236	108	2,070	694	78	4,022	925	19,361
	Sex not reported	0	243	0	0	0	0	24	0	267
	Total[1]	256	12,134	118	2,240	757	83	4,288	1,049	20,925
AIDS diagnoses	Female	9	201	0	50	25	3	72	26	386
	Male	87	4,742	36	853	350	45	1,679	356	8,148
	Total[1]	96	4,955	36	905	375	48	1,759	384	8,558
AIDS deaths	Female	4	114	0	33	16	2	49	17	235
	Male	68	3,249	24	577	234	29	1,294	252	5,727
	Total[1]	72	3,371	24	612	250	31	1,350	270	5,980

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

Childhood Vaccination Coverage

Tables 11 and 12 provide the latest quarterly report on childhood vaccination coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully vaccinated at age 12 months for the cohort born between 1 October and 31 December 1999 and at 24 months of age for the cohort born between 1 October and 31 December 1998 according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in Commun Dis Intell *1998;22:36-37.*

Table 11. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 1998; assessment date 31 March 2001[1]

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,085	21,388	781	11,504	4,501	1,609	15,618	6,279	62,765
Diphtheria, Tetanus, Pertussis (%)	89.6	87.3	82.0	90.4	89.7	91.4	88.5	86.5	88.3
Poliomyelitis (%)	93.9	92.2	93.0	93.5	94.5	95.3	94.0	91.9	93.1
Haemophilus influenzae type b (%)	94.9	94.4	93.6	94.7	95.5	95.5	95.5	93.5	94.7
Measles, Mumps, Rubella (%)	93.0	91.4	91.1	93.3	94.1	94.3	93.0	91.2	92.4
Fully immunised (%)[2]	87.6	82.7	78.9	87.8	87.5	89.7	85.1	82.6	84.8
Change in fully immunised since last quarter (%)	-0.2	+0.8	-1.1	-0.5	-1.1	+1.1	-1.3	-0.5	-0.3

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2000;24:109.
2. These data relating to 2 year old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Aged Care. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone 02 6124 6607.

Table 8. percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 1999; assessment date 31 March 2001

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	990	20,721	778	11,641	4,375	1,453	15,119	6,074	61,151
Diphtheria, Tetanus, Pertussis (%)	92.9	91.0	89.0	91.8	91.5	93.1	92.2	90.1	91.5
Poliomyelitis (%)	92.8	90.9	89.6	91.8	91.5	93.2	92.3	90.0	91.4
Haemophilus influenzae type b (%)	95.4	94.3	94.0	94.4	95.0	95.6	95.3	93.6	94.6
Fully immunised (%)	92.7	90.7	88.6	91.4	91.4	92.6	92.1	89.6	91.2
Change in fully immunised since last quarter (%)	+0.0	+0.2	-0.6	-0.6	-0.8	+1.0	0.0	-0.7	-0.1

Overseas briefs

World Health Organization

This material has been summarised from information on the World Health Organization Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Cholera

South Africa

As of 16 April 2001, 86,107 cases and 181 deaths due to cholera have been reported since the start of the outbreak in mid- August 2000.

Meningococcal disease in the African meningitis belt

An outbreak of meningitis that has spread across Africa has infected about 38,000 individuals and killed at least 3,500. A statement by the International Federation of Red Cross and Red Crescent Societies claimed that it was the worst outbreak of the disease in the last decade and has resulted in a worldwide shortage of the vaccine. Burkino Faso and Benin are particularly affected, reporting 10,987 cases and 7,532 cases, respectively. The federation and other international health organisations are supplying vaccines to the most affected areas. The emergency health coordinator of the federation stated that the infection is likely to spread without widespread vaccination until the seasonal rains arrive.

Burkina Faso

WHO has reported a total of 10,897 cases, including 1,525 deaths between 1 January and 15 April 2001. A further 900,000 doses of vaccine have been released to WHO to continue the mass vaccination campaigns in the most affected areas.

Benin

WHO has reported a total of 7,532 and 300 deaths between 1 January and 25 March 2001 in 4 départements in the Central African Republic. A total of 1,816 cases including 343 deaths have been reported for the period 18 February to 30 March 2001 for the entire country.

Chad

WHO has reported a total of 5,780 cases including 607 deaths from 25 December 2000 to 25 March 2001.

Ethiopia

WHO has reported a total of 4,138 cases including 242 deaths up 31 March 2001 from a widespread area across almost the whole country.

Niger

WHO has reported a total of 4,014 cases including 321 deaths between 2 January and 8 April 2001. Six hundred thousand doses of vaccine have been released to assist in the implementation of the vaccination campaigns in the affected areas.

Meningococcal disease, serogroup W135

During 2001, the following countries have reported to WHO, cases of laboratory confirmed *Neisseria meningitidis* serogroup W135 meningococcal disease, associated with international travel or contact with travellers to Saudi Arabia: Burkina Faso, Central African Republic, France, Norway, Singapore, Germany, Spain and the United Kingdom.

In Saudi Arabia 109 cases (predominantly Haj pilgrims from outside Saudi Arabia) including 35 deaths were reported between 9 February and 22 March 2001. *N. meningitidis* serogroup W135 has been laboratory confirmed in more than half of the cases. As with all types of meningococcal disease, early diagnosis and treatment are essential.

The symptoms of group W135 meningococcal disease are the same as for other groups of the disease: sudden onset of intense headache; high fever; nausea and vomiting; photophobia; and stiff neck. The most severe clinical form of the disease, meningococcal septicaemia, can be presented by abrupt onset, high fever, petechial rash or purpura. WHO recommends that chemoprophylaxis is given to close contacts of the cases, such as persons sleeping in the same dwelling. In most countries rifampicin is recommended. Vaccination of pilgrims to Saudi Arabia against meningococcal meningitis with the quadrivalent vaccine (serogroups A, C, Y and W135) has been made a health requirement.

Rapid reporting system for meningitis W135: 2a: P1.2, 5: update

Reported by Sarah Handford, Brian Henderson and Mary Ramsay, Public Health Laboratory Service Communicable Disease Surveillance Centre, London, England.

The enhanced surveillance system for meningococcal meningitis W135: 2a: P1.2, 5 set up among several European countries after an epidemic of this strain among travellers to the Haj in 2000,[1,2,3,4,5] received reports of 20 cases of infection with this or compatible strains between 31 March and 27 April 2001. Cases were reported by France, Germany, the Netherlands, Spain, and the United Kingdom. Independently, the World Health Organization reported 4 laboratory confirmed cases in Norway (no deaths), 2 of whom had a connection with Haj pilgrims.[6]

In France, meningococci strain W135 were isolated from the blood of a 36-year-old woman admitted to hospital with respiratory symptoms and from the blood and cerebrospinal fluid (CSF) of a 3-week-old baby girl. Both patients survived. Multilocus DNA fingerprinting (MLDF) analysis showed markers of electrophoretic type ET-37 complex in both isolates. The woman had no known link to the Haj, but the baby's grandfather had visited Mecca for the 2001 Haj between 4 and 8 March). *Neisseria meningitidis* strain W135: 2a: P1.2, 5 was isolated from the CSF of a 2-year-old girl who was admitted to hospital with meningitis but had no known link to the Haj. MLDF typing showed markers of ET-37 complex.

In Germany, meningococci strain W135: 2a: P1.2 were isolated from a woman aged 39 years with a haemato-

logical-oncological disease, who presented with meningitis. The woman had no known link to the Haj.

The reference laboratory in the Netherlands received 1 isolate of *N. meningitidis* strain W135: 2a: P1.2, 5. No further epidemiological details are currently available and any link of the patient to the Haj has yet to be confirmed. The laboratory also received an isolate of *N. meningitidis* W135: NT: P1.6 from a woman aged 19 years with no known association with the Haj.

In Spain, meningococci of strain W135: 2a: P1.2,5 were isolated from the blood of a 36-year-old woman with meningitis and sepsis. The clonal line of the strain is to be determined by using multilocus sequence typing. A link to the 2001 Haj has yet to be established.

In the United Kingdom, 8 cases of *N. meningitidis* strain W135: 2a: P1.2,5 were reported in association with the Haj. Two patients died. None of the patients was a pilgrim; 4 were household contacts and 2 were non-household contacts. Two further cases of the same strain were associated with travel to the Middle East other than at the time of the Haj. Three further cases of *N. meningitidis* strain W135: 2a: P1.2,5 were reported, but the patients had no apparent link to the Haj.

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6. World Health Organization. Meningococcal disease, serogroup W135. *Wkly Epidemiol Rec* 2001;76:141-2. <http://www.who.int/wer/pdf/2001/wer7619.pdf>

Plague in Zambia

WHO has received preliminary reports of 23 hospitalised cases, including 3 deaths in Petauke district, Eastern Province. The last case reported was on 15 March 2001. Measures have been taken to intensify surveillance, strengthen control and management of the disease, and provide health education messages on its prevention.

Yellow fever in Brazil

As of 13 March 2001, the Ministry of Health of Brazil and the WHO Regional Office for the Americas have reported a total of 48 suspected cases of yellow fever. Minas Gerais State which has the largest number of cases, is now reported to have 20 laboratory confirmed cases including 9 deaths.

ProMED-mail

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

Polio in Bulgaria

Source: Associated Press Online, 18 May 2001, (edited)

SOFIA: A vaccination campaign announced after 2 cases of poliomyelitis were discovered in Bulgaria was postponed on 18 May 2001 for lack of vaccine. The Bulgarian Health Ministry said that some 800,000 doses of polio vaccine would be imported within days with the assistance of the United Nations Children's Fund.

About 5 per cent of Bulgarian children have never been vaccinated, according to a national epidemiology counsellor who called the statistic 'alarmingly high'. Bulgarian health authorities identified the virus in a 13-month-old Roma child in the Black Sea city of Burgas on 17 April 2001. Other Roma, or Gypsy, children were immediately vaccinated, and the World Health Organization was notified 6 days later.

(Despite this, the virus was detected subsequently on 15 May 2001 in a 2-year-old Roma girl in Yambol, about 60 miles west of Burgas. Doctors said the girl's legs and left arm were paralysed. Laboratories in Paris, Rome, and Atlanta used genetic sequencing to identify the infectious agent as poliovirus type 1 and traced it to northern India, according to a statement from the WHO's European Region Headquarters in Copenhagen, Denmark.

According to official statistics, the 2 infections are the first cases of poliomyelitis in Bulgaria since 1991. During a poliomyelitis epidemic in this year, the Bulgarian health authorities identified the virus in 43 children under the age of 18 months. One died and 42 were disabled. The recent cases are the first cases of polio recorded in Europe since November 1998, when the virus was found in a Turkish province on the Iranian border.

PROMED Editor: This article identifies the poliovirus involved as type 1, presumably wild virus. It is to be hoped that sufficient vaccine will be available to mount the vaccination campaign indicated to interrupt transmission.

Avian influenza – China (Hong Kong)

Source: South China Morning Post 12 April 2001 (edited)

A strain of the deadly bird flu virus has appeared at a market in Hong Kong although officials assured the public there was no health risk to humans. The virus was identified as being in the same family as the virus that caused a fatal outbreak in humans in 1997 and led to the slaughter of 1.6 million chickens, but authorities said it was a different version that had never been known to cross to humans. The virus found, Goose 96-type H5N1, generally occurred in ducks and geese but could cross to chickens and infect them, potentially fatally. The 1997 bird flu outbreak, which killed 6 people, involved Chicken 97-type H5N1 virus, which is known to cause disease and death in humans.

Over the past year, authorities have conducted 28,000 tests and found 17 samples of the goose version of the virus in geese and ducks in wholesale markets, but no examples of the dangerous chicken version. The latest goose version-infected faecal sample, taken on 22 February 2001, was the first discovery of a H5N1 family virus in a retail market since the fatal outbreak in 1997.

Avian influenza – China (Hong Kong)

Contributed by: Marianne Hopp. Source: WHO Disease Outbreaks Report, 18 May 2001 (edited)

There has been an increase in the number of deaths of poultry from influenza A(H5N1) virus in additional retail live-bird poultry markets [see previous post: Avian influenza, H5N1 - China (Hong Kong) (02) 20010517.0962].

As a result, the Government of Hong Kong SAR has announced that all wholesale and retail markets selling chickens will be closed and the birds will be destroyed. Retail outlets for live chickens will remain closed for 4 weeks. Farms with live chickens ready for slaughter will be depopulated within the next 2 weeks. Importation of chickens from the Mainland has been stopped.

No human cases of influenza A(H5N1) virus have been detected. The strains isolated from the birds differ genetically from the H5N1 virus which caused human disease in 1997. There is no cause for public health concern.

All chickens in the Hong Kong SAR to be slaughtered

Contributed by: George A. Robertson.
Source: smh,com.au, 19 May 2001 (edited).

HONG KONG: About 2 million chickens and other poultry will be slaughtered by the territory and imports halted from mainland China to stop the spread of a bird flu outbreak.

The Secretary for the Environment and Food said all poultry in local food markets would be killed by tomorrow and at farms throughout the territory within 2 weeks. The Government had asked mainland China to halt exports of live birds to the territory. Hong Kong consumes around 100,000 fresh chickens a day, of which more than 70 per cent come from mainland China. The Secretary stressed the virus would not affect humans.

The Government had already slaughtered about 6,600 chickens on Wednesday after discovering the strain had killed a large number of birds at 3 markets, but it has since discovered more chickens dying of the viral infection. The source of the strain has not been traced. In total, 10 markets have been found to be infected with the virus.

Virologists said that the new strain derives from a virus that infected geese in China's Guangdong province in 1996, Scientists call southern China an 'epicentre' of (influenza) viruses because farmers there rarely segregate different types of poultry, creating a perfect breeding ground for new flu strains.

Unlike ducks and geese, which are centrally slaughtered in Hong Kong, chickens are sold live in markets and are killed and plucked in front of customers. The latest outbreak brings that practice into question. 'Scientifically and logically, it would of course be best to centralise the slaughtering of chickens,' the Secretary said. 'But would the public accept that?'

PROMED Editor: The central slaughtering of chickens might facilitate the monitoring and control of outbreaks, but it would not have much effect on the evolution of new strains of avian influenza virus in the poultry rearing areas of South China and the Hong Kong SAR.

Avian influenza – Japan: pet parakeets

Source: New Scientist, Vol. 169, No. 2283, p. 16; 24 March 2001 (edited)

Scientists at the Japanese National Institute of Animal Health recently diagnosed influenza virus strains in some imported parakeets that died after arriving in Japan. This is the first time the H9N2 viruses have been discovered in pet birds; prior to this incident, only chickens and mice were known to be susceptible to these viruses.

H9N2 is not a threat to humans in its current form, only causing mild symptoms. The concern is the virus's kinship to a cousin called H5N1, which normally only infects chickens. In 1997, H5N1 made an alarming leap into humans, infecting 18 people in Hong Kong and killing 6. In an effort to contain the virus outbreak, masses of chickens in the city's open markets were destroyed. The more benign H9N2 virus could become a critical threat to humans if it mutates or combines with the more aggressive H5N1 strain. There are calls in Japan for the establishment of quarantine and surveillance precautions in any country that deals in imported birds.

Unexplained deaths – India (North Bengal)

Source: British Medical Journal 2001; 322: 693. 24 March 2001 (edited).

Indian scientists have warned that India may be witnessing the emergence of a highly lethal measles-like virus, which causes encephalitis in adults and children. Scientists investigating an outbreak of encephalitis among adults told the Health Ministry that a mutant measles virus that affects the brain, lungs, or kidneys caused the disease.

This is India's third outbreak since 1998 of a highly fatal illness involving the brain or the kidneys and attributed to measles virus. The outbreak during February this year killed at least 28 people, including 2 doctors and 5 nurses in a clinic. The infection spread through droplets in air expelled by patients during the terminal phase of the illness, which is marked by pneumonia.

Epidemiologists say that adequate protection and barrier nursing helped to quell the outbreak. Investigations had ruled out vector-borne infections common in India, such as cerebral malaria or Japanese encephalitis. Tissue samples showed antibody to measles virus in 17 samples collected. Measles virus antigen was detected in brain tissues of 2 patients.

USA (California) - Mycobacteriosis

At least 110 customers of a California nail salon developed boils and skin ulcers after receiving a pedicure during which they soaked their feet and calves in a footbath for 10 to 15 minutes. The boils appeared between 10 days and

4 months following the pedicure, and in some cases, the infection was not responsive to antibiotics. The infection was caused by a fast-growing microbe, Mycobacterium fortuitum, which was found in high concentrations in the footbaths. The strains of M. fortuitum collected from the footbaths matched those of the infected customers. According to the epidemiologist who led the investigation of the outbreak, which occurred last fall, this was the first spread of the infection in a community. A subsequent spot check of other footbaths throughout California revealed that the bacteria were present in the majority of them. California officials are expected to develop regulations calling for thorough cleaning and disinfection of footbaths used in nail salons.

UNAIDS warns Asia could exceed Africa in AIDS cases

Source Unwire 24 April 2001

At a special session of the United Nations (UN) Economic and Social Commission for Asia and the Pacific in Bangkok, UN officials warned that the number of HIV infections in Asia over the next 10 years could surpass those of Africa unless immediate action is taken to stem the spread of the virus. Kathleen Cravero, deputy executive director of the Joint UN Program on HIV/AIDS, stated that there were 900,000 new infections last year in the Asia-Pacific region, and 490,000 people died of AIDS. In Africa, 3.9 million people were infected last year, and 2.4 million died. According to Cravero, South Asia is the fastest growing epidemic outside of sub-Saharan Africa, with an infection rate of 5 per cent. She also stated that epidemics in Cambodia, Myanmar, Thailand, and parts of India already have spread beyond sex workers and intravenous drug users to the general population. Cravero and other UN officials emphasised the pivotal roles of governments, businesses, and other sectors in helping to fight the spread of the disease at a national level while the epidemics are still in their early stages.

Additional suspected clusters of vCJD cases in Britain

Source: The Sunday Times, Sun 13 May 2001 (edited)

Scientists are investigating 6 new potential clusters of variant Creutzfeldt-Jakob disease (vCJD) victims scattered throughout Britain. At least 2 people have died from the human form of 'mad cow' disease in each of the 6 areas, and experts are examining links between the cases by studying local butchery practices, medical records, and eating habits. So far only one group of cases has been confirmed; namely, in the village of Queniborough, Leicestershire, where 5 young people died.

Yesterday it emerged that a third victim of the disease had died in the town of Eastleigh, Hampshire, one of the potential cluster sites. A 25-year-old man was diagnosed with vCJD 5 months ago. A preliminary investigation was started earlier this year into the deaths of 2 other people in the Eastleigh area. It found no obvious links between the cases, but further investigations are to take place during the coming months. Scientists from the government-funded CJD Surveillance Unit, based in Edinburgh, are working in conjunction with local health authorities in an effort to isolate common factors between cases.

The Department of Health refused to identify the areas being examined, but last week local health authorities covering Glasgow, Chester-le-Street (Durham), and Stockton-on-Tees, in addition to Eastleigh, confirmed that they were assisting in investigations. The unit is believed to be preparing to launch studies in 2 other unnamed areas. Studies have already been conducted in Stockport and in Armthorpe, South Yorkshire, but have failed to find any links between cases. Victims' families are being interviewed and sent questionnaires which examine where they bought meat, where children went to school, and where they had hospital or dental treatment to ascertain if there was exposure to contaminated blood.

Research by the CJD Surveillance Unit and the London School of Hygiene and Tropical Medicine found that the incidence of vCJD was twice as high in the north of Britain as in the south. Dr Simon Cousens, who led the study, confirmed that potential new clusters were being investigated. However, experts warn that trying to find links between cases was extremely difficult. In Armthorpe no direct links were found, even though the 2 victims of the disease were friends who lived in the same street. A 28-year-old woman, who died last September, was a friend of a 19-year-old man, who died in 1997. Last week Doncaster Health Authority said investigations had been hampered because many small abattoirs from the 1980s had closed. In Stockport a 29-year-old man and a 34-year-old man who lived only 2 streets apart both died of vCJD, but again no link has been found.

There have been 99 cases in total of ‘definite and probable’ vCJD in Britain.