Sexually transmissible diseases surveillance in Australia: towards a coordinated national system

Gregory J Dore[1] *and John M Kaldor*

National Centre in HIV Epidemiology and Clinical Research

Abstract

Communicable diseases surveillance is essential for directing policy development. In most regards, sexually transmissible diseases (STD) surveillance is no different to surveillance for other communicable diseases. There are nevertheless several aspects of STDs that have to be taken into consideration in designing and managing surveillance activities. These include particular confidentiality concerns associated with STDs, the disproportionate morbidity STDs confer on marginalised or stigmatised populations, and clinical limitations due to the requirement of often uncomfortable genital examinations for the diagnosis of many STDs. Furthermore, interpretation of STD surveillance data requires information on sexual behaviour which is not routinely collected for other types of surveillance. In addressing new STD surveillance strategies the key public health questions that can be answered by surveillance need to be defined. These include the prevalence of individual STDs among the total population and specific population subgroups, the rates of symptomatic versus asymptomatic disease, treatment seeking levels, and antibiotic sensitivity patterns for some agents. This article describes possible STD surveillance methodologies to meet these demands. *Comm Dis Intell* 1998;22:49-52

Introduction

Notifications of gonorrhoea and syphilis to the National Notifiable Diseases Surveillance System (NNDSS) over the period 1991-1996 demonstrated an increase of 51% and a decline of 29% respectively (Figure 1). These contrasting trends may indicate a true divergence in risk factors for these two infectious diseases, but it is likely that changes in surveillance methodology have played a role. Gonorrhoea incidence may have increased due to:

• an increase in screening, assisted by more widespread use of new diagnostic technology such as the polymerase chain reaction (PCR);

• greater access to sexual health care services;

1. Corresponding author: Dr Gregory J Dore, Level 2, 376 Victoria Street, Darlinghurst, New South Wales 2010

ISSN 0725-3141
Volume 22
Number 4
16 April 1998

Contents

• improved surveillance;

• a real increase in the rate of infection.

In contrast, syphilis incidence may have declined due to:

• a decrease in screening activities;

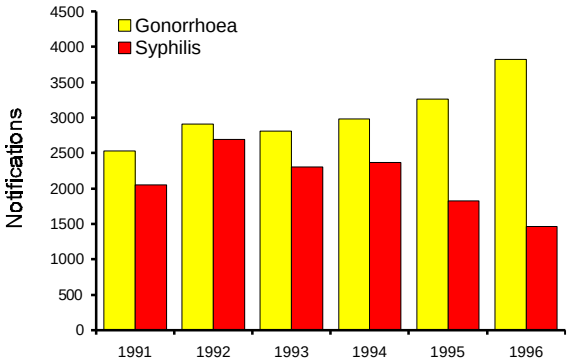
• changes to the surveillance case definition towards notification of only early cases;

• decreased surveillance;

• a real decline in the rate of infection.

The difficulty in interpretation of these trends in STD incidence highlights some of the deficiencies in the existing national surveillance system.

Figure 1. Notifications of gonorrhoea and syphillis in Australia 1991 to 1996, by year and disease



Current initiatives

The definition of disease surveillance as 'the collection, analysis and interpretation of information on the health status of population groups', indicates areas where attention can be focussed at improving STD surveillance. Some initiatives to support this goal are already underway. Firstly, there have been considerable recent efforts towards improving overall surveillance of communicable diseases in Australia, including the standardisation of case definitions, which is now taking place within the framework of the National Communicable Diseases Surveillance Strategy.[1] Surveillance of STDs should benefit from these wider initiatives in the area of communicable diseases.

Secondly, the subtext to the third National HIV/AIDS Strategy,[2] 'a strategy framed in the context of sexual health and related communicable diseases', has obvious implications for STD management and control, including surveillance activities. The monitoring and evaluation section of the strategy document specifically states that 'there will be a particular emphasis on improving surveillance and social and behavioural data on HIV/AIDS, hepatitis C, sexually transmissible diseases and sexual health among Aboriginal and Torres Strait Islander people.' The convening of the Indigenous Australians' Sexual Health Working Party as a subcommittee of the Australian National Council on AIDS and Related Diseases (ANCARD), and the subsequent release of *The National Indigenous Australians' Sexual Health Strategy 1996-97 to 1998-99*[3] has provided a policy framework for addressing STD control among indigenous people.

Thirdly, some States and Territories have made considerable advances towards the development of more comprehensive STD surveillance systems. For example, in Victoria there has been a marked improvement in STD surveillance during the 1990s. An enhanced surveillance system has been developed through the use of active and 'stimulated passive' surveillance programs for HIV/AIDS, syphilis and hepatitis B.[4] Specific measures which facilitated development of more comprehensive STD surveillance have been the introduction in 1990 of mandatory laboratory-based notification of some STDs in addition to the established clinician-based reporting mechanisms, the establishment of STD case definitions, and systematic consultation with laboratories and other stakeholders. Laboratory-based surveillance programs for gonorrhoea and syphilis have been augmented to provide data on sexual orientation and ethnicity (gonorrhoea and syphilis), site of infection and probable place of acquisition of infection (gonorrhoea), and reason for testing and stage of disease (syphilis). Call-back mechanisms have also been introduced to support these secondary surveillance mechanisms.[4] Several other States have also moved to a greater reliance on laboratory-based STD surveillance methods.

Outside the routine health department surveillance system, the reporting of data on gonorrhoea incidence and antibiotic sensitivity through the laboratory-based Australian Gonococcal Surveillance Programme (AGSP) continues to make a substantial contribution to Australian STD surveillance and has been a stimulus for enhanced gonococcal surveillance in the Asia-Pacific Region.[5]

Despite these current initiatives, further steps towards an integrated national STD surveillance program in Australia are required. An STD surveillance system should provide ongoing information on the incidence of infection, the occurrence of symptomatic versus asymptomatic disease, the extent of treatment seeking, associated patterns of sexual behaviour, and the demographic variation of these indices.

Surveillance system options

In continuing to improve national STD surveillance, it must be kept in mind that no simple system can satisfy all these requirements. Options for the future of STD surveillance in Australia include the current routine centralised case reporting system, monitoring routine STD testing in selected population groups or clinical sites, or emphasis on special surveys in selected population groups or clinical sites. The current centralised passive surveillance system provides some information on the pattern of STDs in Australia, but quantitative interpretation is impeded by both under-reporting, and duplicate reporting particularly for diseases with persistent markers such as syphilis and hepatitis B. Under-reporting for gonorrhoea has been well documented.[6] For donovanosis, a study in the Northern Territory demonstrated a reporting rate of only 30% of treated cases during 1993.[7] Incorporation of personal identifiers into the national case reporting system, as is the case for HIV and AIDS surveillance, would reduce the possibility of duplicate reporting for STDs such as syphilis, and would enable cross-linkage with other communicable

disease databases including the national HIV and AIDS databases. Even with the current extent of under-reporting however, the routine case notification rate for other STDs far outweighs that for HIV/AIDS. For this reason therefore, an identifier encoded database for other STDs would be a substantial and probably unrealistic undertaking.

Monitoring of routine STD testing in selected population groups or clinical sites (sentinel surveillance) is an alternative core surveillance mechanism. This form of surveillance would make use of existing health care infrastructure and STD screening and diagnostic services, such as sexual health clinics, blood transfusion services, and antenatal clinics. Sentinel surveillance has been employed widely in Australia for the monitoring of HIV infection. The national network of major sexual health clinics that has been reporting on HIV diagnoses since 1991, also provides limited data on a core group of STDs.[8] This system could be improved by the inclusion of information such as denominator testing data, symptomatology, and reason for testing, and could be expanded to incorporate other sexual health clinical sites. This would enable an estimation of the level of testing for diagnostic and screening purposes. Routine screening for syphilis which is already conducted through antenatal clinics and blood transfusion services, could also provide a form of sentinel surveillance. However, a potential limitation of sentinel surveillance is the representativeness of subjects. For example, blood donors are likely to be a lower risk population than the general population.

Monitoring of trends in transmission of some STDs may be most effectively performed through special surveys, such as serial cross-sectional serological or PCR-based urinary surveys. For example, genital herpes is difficult to monitor because of the lack of a standardised case definition, difficulties with distinguishing new from recurrent infection and variation in diagnostic methodology. The level of transmission of genital herpes, which is not currently notifiable at a national level, could be monitored through regular serological surveys of HSV-2 among young adults or pregnant women. In regions with low levels of classical STDs, such as Tasmania, where reports of syphilis and gonorrhoea are extremely uncommon,[9] special surveys to monitor transmission of herpes and chlamydia could play a vital role in monitoring trends in STD transmission. Surveys of chlamydia and gonorrhoea could utilise the PCR-based methodologies for urinary detection, which have recently been trialed in remote aboriginal communities in central Australia.[10] PCR detection of gonorrhoea, chlamydia, trichomonas and human papilloma virus utilising self-administered tampon specimens, also recently reported for women in the Northern Territory[11] could also be used.

Monitoring outcomes of infection is an area in which surveillance mechanisms utilised to examine patterns of, and trends in advanced HIV disease, could be adopted for other STDs and incorporated within a national surveillance program. As with the Australian AIDS Cohort,[12] a three hospital HIV/AIDS unit-based surveillance mechanism, surveillance of pelvic inflammatory disease and other complications of STDs could be established in sentinel hospital sites. The analysis of pelvic inflammatory disease from Royal Darwin Hospital over the period 1991-1994[13] is an example of the type of surveillance which could be undertaken on an ongoing basis.

The importance of sexual behaviour surveillance in defining the level of risk, and in following trends in risk activity, has been well demonstrated in the area of HIV infection. It has also complemented and enhanced HIV epidemiological surveillance activities in many areas. Due to the focus of the Australian HIV epidemic among homosexual men, and the continuing relatively low levels of risk among the heterosexual population, sexual behaviour surveillance has been concentrated among gay men. Other sub-populations in which a better understanding of sexual behaviour is required include the young adult heterosexual population, travellers to areas where considerable numbers of imported STDs are acquired, such as Thailand and the Philippines, and among the indigenous population. The support and involvement of community groups in behavioural surveillance, a feature of the monitoring of the HIV epidemic among homosexual men, is crucial for the success of these initiatives in other STDs and with other target groups.

Appropriate feedback mechanisms for STD surveillance data also need to be developed. These mechanisms need to take into consideration the potentially sensitive nature of outputs from an STD surveillance system. For example, reporting of STD data by indigenous status would require the involvement and support of indigenous people through mechanisms such as the Indigenous Australians' Sexual Health Working Party. The inclusion of STD surveillance data collected through the NNDSS and the AGSP in the *HIV/AIDS and Related Diseases in Australia Annual Surveillance Report*[8] should improve the integration of STD and HIV data in policy development and evaluation, particularly as further improvements in STD data collection, analysis and interpretation take place.

National coordination

Many of these STD surveillance considerations could be facilitated by the designation of specific responsibility for coordination of STD surveillance to a body with maintenance of linkages with the National Centre for Disease Control and Communicable Diseases Network Australia New Zealand within the framework of the National Communicable Diseases Surveillance Strategy. National coordination would be improved through the creation of a STD surveillance network and the development of a National STD Surveillance Strategy. The STD surveillance network should involve State and Territory representation along with representation from appropriate bodies such as the National Venereology Council of Australia (NVCA) and the Indigenous Sexual Health Working Party and the AGSP.

References

1. National Centre for Disease Control. *National Communicable Diseases Surveillance Strategy.* Canberra: Commonwealth Department of Health and Family Services, 1996.
2. Commonwealth Department of Health and Family Services. *Partnerships in Practice. National HIV/AIDS Strategy 1996-97 to 1998-99.* Canberra: Looking Glass Press, 1996.
3. ANCARD Working Party on Indigenous Australians' Sexual Health. *The National Indigenous Australians' Sexual Health Strategy 1996-7 to 1998-99.* Canberra, Looking Glass Press, 1997.

4. Crofts N, Gertig DM, Stevenson E, Thompson S, Lester R, Forsyth J. Surveillance for sexually transmissible diseases in Victoria, 1983-1992. *Aust J of Public Health* 1994; 18:433-439.
5. Tapsall J. The Australian Gonococcal Surveillance Programme. *Venereology* 1991; 4:99-102.
6. Condon R, Roberts M, Rouse I, Sesnan K. A comparison of doctor and laboratory notifications of gonorrhoea in Western Australia during 1989. *Venereology* 1992; 5:84-86.
7. Mein J, Patel A, Bowden FJ. Surveillance of donovanosis in the Northern Territory. *Venereology* 1995; 8:16-19.
8. National Centre in HIV Epidemiology and Clinical Research (editors). *HIV/AIDS and Related Diseases Annual Surveillance Report* 1997.
9. Evans D, Ho D. Tasmania: sexually transmissible diseases. *Venereology* 1994; 7:191-194.
10. Skov SJ, Miller P, Hateley W, Bastian IB, Davis J, Tait PW. Urinary diagnosis of gonorrhoea and chlamydia in men in remote Aboriginal communities. *Med J Aust* 1997; 166:468-71.
11. Fairley CK, Bowden FJ, Gay NJ, Paterson BA, Garland SM. Sexually transmitted diseases in disadvantaged Australian communities. *JAMA* 1997; 278:117-18.
12. Dore GJ, Hoy JF, Mallal SA, Li Y, Mijch AM, French MA, Cooper DA, Kaldor JM. Trends in incidence of AIDS illnesses in Australia 1983-1994: The Australian AIDS Cohort. *J Acquir Immune Defic Syndrom Human Retrovirol* 1997; 16:39-43.
13. Mein J, Bowden FJ. A profile of inpatient STD-related pelvic inflammatory disease in the Top End of the Northern Territory of Australia. *Med J Aust* 1997; 166:464-67.

Acknowlegdment

The National Centre in HIV Epidemiology and Clinical Research is supported by the Australian National Council on AIDS and Related Diseases through its Research Advisory Committee.

An outbreak of non-sexually transmitted gonococcal conjunctivitis in Central Australia and the Kimberley region

Rex Matters,[1] Ignatius Wong, [2] and Donna Mak[3]

Abstract

From 13 February to 27 June 1997, 447 cases of gonococcal conjunctivitis were identified by Communicable Disease and Public Health Centres and Community Clinics in the Northern Territory, Western Australia and South Australia. The outbreak involved Aboriginal communities predominantly in Central Australia and the Kimberley region in Western Australia. This was the first outbreak recorded in the Kimberley region. It is not yet known whether the Kimberley cases were part of the larger Central Australian outbreak or whether they represented a separate and unrelated outbreak. Environmental factors associated with this outbreak were similar to those seen in previous outbreaks. Control measures were based on early recognition and treatment of index cases and identifying and treating contacts. Until sexually transmitted *Neisseria gonorrhoeae* is controlled in communities gonococcal conjunctivitis is likely to appear again. The role of oropharyngeal carriage of *N. gonorrhoeae* needs to be evaluated further. *Comm Dis Intell* 1998;22:52-58

Introduction

Gonococcal conjunctivitis is an acute painful conjunctivitis characterised by rapid transmission between individuals through non-sexual person to person contact. It has caused considerable morbidity in Aboriginal communities in Central Australia during five previously documented outbreaks (Table 1).[1,2,3,4,5]

Environmental factors associated with these outbreaks included: above average summer rainfall preceding the outbreak; summer temperatures at the onset changing to winter temperatures towards the end; an increase in the percentage of *Haemophilus* species isolates from eye swabs; and increased fly numbers at the start of the outbreak. None of the outbreaks were characterised by an increase in notifications of sexually transmitted gonorrhoea in the time period preceding the outbreak.

This article reports on an outbreak of gonococcal conjunctivitis in Aboriginal communities predominantly in Central Australia during the period 13 February to 27 June 1997, and examines some of the environmental factors associated with the outbreak. The Central Australian area involved included the western Alice Springs region in the Northern Territory (NT), the Ngaanyatjarra central desert area and the Pitjantjatjara Lands of South Australia (SA). Cases also occurred in the Kimberley region in Western Australia (WA).

Methods

Case definition

A clinical illness was defined as intense inflammation of the conjunctivae, copious purulent discharge with or without periorbital oedema. A clinical case was confirmed

1 Corresponding author: Territory Health Services, Alice Springs Hospital, PO Box 2234, Alice Springs, Northern Territory 0871
2 Western Diagnostic Pathology, Alice Springs, Northern Territory
3 Kimberley Public Health Unit, Western Australia

when *Neisseria gonorrhoeae (N. gonorrhoeae)* was isolated on culture, detected by polymerise chain reaction (PCR) or Gram negative diplococci were seen by microscopy. Unconfirmed clinical cases were included in this analysis if there was a laboratory confirmed case notified from the same community. Date of onset was defined as the date on which the eye swab confirming the diagnosis was taken.

Collection of data

Patient information including age, gender, date of onset of illness and address was obtained from Disease Control Centres in Alice Springs, NT and Adelaide, SA; Goldfields Public Health Services, Boulder, WA; Kimberley Public Health Unit, Derby, WA; Western Diagnostic Pathology, Alice Springs, NT and Pathology, Alice Springs Hospital (ASH), NT. Information on the number of unconfirmed clinical cases, the spread of disease and fly density was obtained from Community Health Clinics in Central Australia. Population demographic statistics were obtained from Nganampa Health Council, Ngaanyatjarra Health Service and Rural Health - Alice Springs, Territory Health Services (THS). Rainfall and temperature data were obtained from the Bureau of Meteorology, Darwin and Alice Springs Regional Offices NT and WA. The number of unconfirmed clinical cases was not obtained from the Kimberley and only laboratory confirmed cases from this area were included in the analysis.

Laboratory investigation

N. gonorrhoeae strains from Central Australia were sent to the Prince of Wales Hospital, Sydney, for serotyping, auxotyping and minimum inhibitory concentration (MIC) testing. Records at the Pathology Laboratory, ASH, were examined to determine the percentage of *Haemophilus* spp. identified from eye swabs between June 1996 and June 1997 and the number of cases of disseminated gonococcal infection (DGI) between 1 August 1995 and 18 July 1997. DGI was defined as the isolation of *N. gonorrhoeae* from a sterile site, for example joint fluid.

Results

Number of cases

A total of 447 cases of gonococcal conjunctivitis were reported, 242 confirmed cases (including 5 reinfections) and 205 unconfirmed cases. Of the confirmed cases, 120 were culture positive, 53 PCR positive and 69 microscopy positive. The NT had 121, WA 105 and SA 16 confirmed cases. There appear to have been two epidemics during the outbreak (Figure 1). The smaller epidemic peaked in the Kimberley during March before the larger epidemic, involving many more communities in Central Australia, which peaked in May. The sharp peak in the number of Kimberley cases, however, is artefactual. Cases had been observed during late January and early February, but no swabs had been taken as gonococcal conjunctivitis had not been suspected at that time.

Gender and age distribution and of cases

There were 113 males and 129 females with confirmed infection. Over three-quarters of the confirmed cases were children under 10 years old, with the greatest number recorded in the 5 to 9 years age group (Table 2). The youngest child was four months old.

Figure 1. Gonococcal conjunctivitis, laboratory confirmed cases by week and geographic region

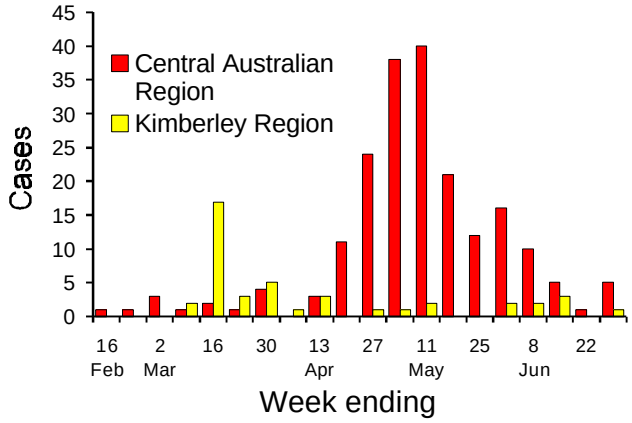


Table 1. Gonococcal conjunctivitis outbreaks in Central Australia, by date and number of cases

Year of outbreak	Cases[1]	Period of outbreak
1934	91	-
1981	35	May to June
1986-87	>140	November to May
1991	432	January to July
1992	>62	April to July

1. Includes both clinical and laboratory confirmed cases

Table 2. Age distribution of confirmed cases

Age group (years)	Number of cases	Per cent of cases
0 to 4	73	30
5 to 9	114	47
10 to 14	35	15
15 and over	12	5
Unknown	8	3
Total	242	100.0

Rainfall

The Giles Meteorological Station is in WA, approximately 150 kms north of the junction of the WA, NT and SA borders. This station was one of the closest to the communities which were involved in the start of the outbreak. The rainfall at Giles in December was above average and there was substantial rainfall in January and February preceding the outbreak (Figures 2a and 2b). The first cases appeared in the middle of February. Alice Springs received 80 mm of rain in January and 242 mm in February. The Kimberley region received 136 mm of rain in January and 274 mm in February. This is well above the average rainfall for these months.

Temperature

The mean daily minimum temperature at Giles in February was 24.7°C (Figures 2a and 2c) at the start of the outbreak and was below 10°C in June at the end of the epidemic. At the start of the epidemic the mean daily maximum temperature in February was 36°C, which is a typical summer temperature.

Sexually transmitted gonorrhoea in Central Australia

There was no increase in the notifications of sexually transmitted *N. gonorrhoeae* preceding the outbreak. The notification information from the NT Centre for Disease Control indicated that sexually transmitted disease was endemic at nearly the same level all year round.

Proportion of *Haemophilus* spp. isolates from eye swabs received at Alice Springs Hospital

The total number of *Haemophilus* spp. identified each month from eye swabs, expressed as a proportion of the monthly total number of eye swabs received, can be used as an indicator of the amount of general bacterial/viral eye disease in communities. During the period June 1996 to June 1997, the highest proportion of *Haemophilus* spp. (47%) occurred in March 1997. The mean monthly proportion was 33%. There was a gradual increase in the proportion of eye disease in which *Haemophilus* spp. was isolated at the start of the outbreak.

Fly density

Thirteen Central Australian communities involved in the epidemic were contacted and asked when they thought flies were at their most dense and when the flies decreased in number. Five communities where the first gonococcal conjunctivitis index cases appeared reported the flies were 'terrible' in February. Ten communities reported that the fly density was greatest in March and April. Fly density in four communities was greatest in May. Fly density reduced dramatically in June.

Laboratory investigation

Delays in transport frequently resulted in the death of *N. gonorrhoeae* in swabs and in many cases confirmation of infection could only be made from smears and PCR. All the Kimberley cases were tested by PCR.

Auxotyping, serotyping and MIC testing from Central Australia

The *N. gonorrhoeae* strains submitted for serotyping, auxotyping and MIC testing included 89 eye, 34 genital and 2 joint isolates. Of the 53 fully typed eye isolates, all were auxotype/serovar class Wt/IB3**.** Fourteen background isolates from standard STD surveillance obtained during the outbreak were typed as six Wt/IB3 strains, three Pro/IB3 strains, and one each of strains Pro/IB1, Pro/IB5, Wt/IA6, Pro/IA4 and Pro/IA6. Two eye isolates not involved in the outbreak, consisted of a Pro/IB3 strain and a Pro/IB1 strain. Two joint isolates consisted of a Wt/IA4 strain and a IB3 serovar (the Wt/IA4 strain was not involved in the outbreak but was included as a background strain). STD background isolates from April 1996 consisted of one Wt/IB3, thirteen Pro/IB3, four Wt/IA6 and three Wt/IA4 strains. The majority of the eye isolates tested so far have had a penicillin MIC of between 0.125 and 0.25 mg/L, with one isolate having a penicillin MIC of 0.5 mg/L. According to the criteria established by the Australian Gonococcal Surveillance Programme, all the eye isolates are classified as sensitive to penicillin, but in the less sensitive range.

Figure 2a. Gonococcal conjunctivitis, number of cases at Giles, June 1996 to June 1997, by month

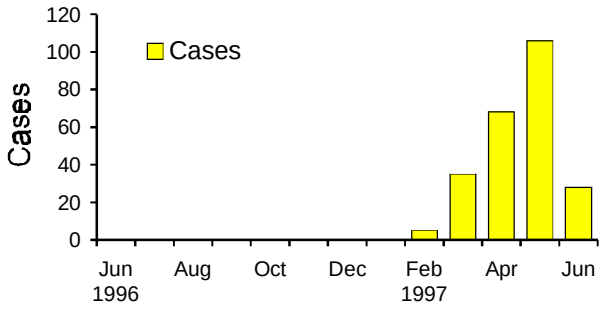


Figure 2b. Rainfall at Giles, June 1996 to June 1997, by month

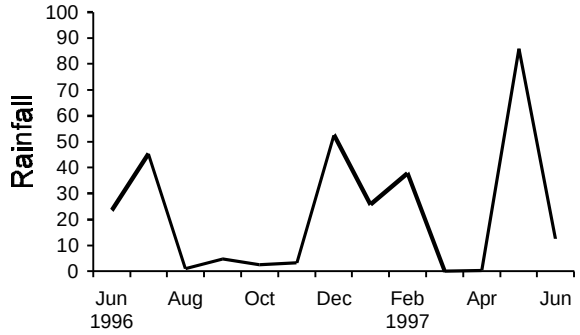
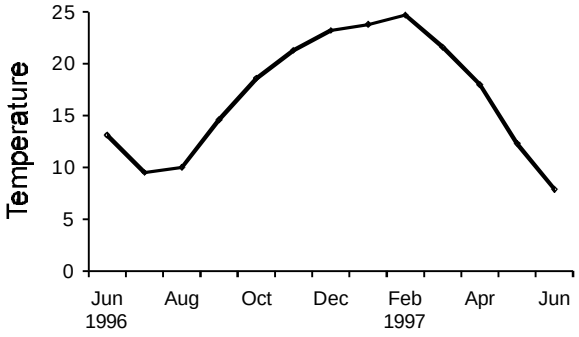


Figure 2c. Mean daily minimum temperatures at Giles, June 1996 to June 1997, by month



Public health response

An alert was sent by Alice Springs Disease Control to Central Australian communities (NT, WA, SA) in March after several cases had been reported. This alerted the communities to the presence of current cases, urged that swabs and cultures be taken and that procaine penicillin or amoxycillin with probenecid be given. A second alert was

sent out in April. In May, with over 40 cases reported, a further alert was sent out which included advice to treat household contacts, a direction not explicitly stated in the Central Australian Rural Practitioners Association (CARPA) standard treatment manual. An alert also went to all remote schools telling of the outbreak and the need for treatment. The outbreak, protocol, and need to treat all household contacts were discussed at both the Central Australia Disease Control Coordinating Committee (CADCCC) meeting/teleconference and the CARPA Conference, during May 1997.

Public Health Unit staff in the Kimberley region arrived at the affected community within 24 hours of the first notification (though not necessarily the index case) of gonococcal conjunctivitis. They then assisted local staff with screening and treatment.

Discussion

The number of cases reported is undoubtedly an underestimate of the true number of gonococcal eye infections, as not all patients presenting with conjunctivitis had an eye swab and smear taken for laboratory confirmation by either culture, smear or PCR. Direct input from Central Australian communities was invaluable in obtaining a more accurate estimate of the amount of clinical disease that was treated.

Fly counts were not performed in any community and the subjective assessment of fly numbers by staff may be inaccurate, but the survey covered many communities and a reasonable picture was obtained.

There is some indication that conjunctivitis caused by *Haemophilus* spp. is endemic throughout the year and Haemophilus conjunctivitis rises prior to and during gonococcal conjunctivitis outbreaks. This has been observed in every previous outbreak and is possibly due to the same environmental and other stimuli which promote gonococcal transmission.

The environmental factors present in this outbreak included:

1. heavy summer rainfall at least one month before the first cases appeared;

2. mean daily minimum temperature above 20^{o}C for the first two months of the outbreak; and

3. an extremely high fly population in the first three months of the outbreak.

In addition, the percentage of eye infections caused by *Haemophilus* spp. increased to its highest monthly level for the year during the second month of the outbreak (March). These findings were similar to those seen in the previously reported outbreaks.

Factors which may have contributed to the occurrence and spread of the epidemic include: a background of high levels of sexually transmitted gonorrhoea; environmental conditions favouring increased survival of the organism; bushflies acting as a mechanical vector of *N. gonorrhoeae*; and, a high level of mobility of the population.

There is a large reservoir of sexually transmitted *N. gonorrhoeae* in remote Aboriginal communities in Central Australia,[6] however, in common with previously reported outbreaks, the incidence of sexually transmitted *N. gonorrhoeae* did not increase before the outbreak.

N. gonorrhoeae can only survive in warm moist conditions and will die rapidly in a dry cold atmosphere. Survival of the organism on fomites would have been optimal at the start of the outbreak, rapidly decreasing as the temperature dropped below 10^{o}C.

The breeding conditions, including humidity and temperature, for bushflies were ideal[7] just after the rainfall in December and deteriorated markedly in June. A study conducted by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) in Central Australia in 1980-1982, where fly counts were performed, recognised that the only major increase in bush fly abundance occurred after the first summer rainfall.[8] Bushfly pupae cannot survive at all when the temperature fluctuates between 6^{o}C and 18^{o}C. During the outbreak small adult flies were present (indicating a reduced larval stage) and there was extreme pressure for the available moisture/food necessary for the breeding cycle and for survival. It is likely that flies were attracted to moist, purulent eyes and became ‘sticky’ flies.

There is still insufficient evidence to determine whether flies definitely contribute to the spread of gonococcal conjunctivitis. Recent studies in WA have shown that *Chlamydia trachomatis* can survive in the bushfly population, however, further work is still in progress to ascertain whether the number of organisms carried is sufficient to cause trachoma (personal communication, Dr. Ian Dadour, entomologist).

Aboriginal people have a high level of mobility between communities. This contributes to the spread of disease. Examination of ‘dates of onset of disease’ figures and discussions with staff at community clinics, have indicated that the outbreak seemed to have started at the WA/NT border and moved eastwards into the Alice Springs region as well as down into the top of SA.

Mini-outbreaks may occur in different communities. Auxotyping and serotyping of gonococcal isolates from the 1986-87 and 1991 outbreaks identified four different strains of gonococci in one outbreak and four in the other. Only one strain has been identified to date from the Central Australian eye isolates obtained during this outbreak. As no organisms were available for typing from the Kimberley, it is not possible to determine whether this was part of the Central Australian outbreak or was a separate outbreak. Two non-outbreak isolates were tested and found to be different strains. As has happened in previous outbreaks there were numerous sexually transmitted *N. gonorrhoeae* strains, and eight different strains were detected in the sample tested preceding and during this outbreak. It appears that different strains predominate during different times of the year and that any strain is capable of causing an outbreak.

Injectable penicillin or amoxycillin with probenecid worked well during this outbreak to eradicate gonococcal conjunctivitis. The highest penicillin MIC recorded against the isolates of *N. gonorrhoeae* causing this outbreak was 0.5 mg/L, which lies in the less sensitive range.

Another possible site of *N. gonorrhoeae*, though not necessarily involved in transmission to the eye, is oropharyngeal carriage of the organism. One investigation[9] found three asymptomatic oropharyngeal carriers of *N. gonorrhoeae* in the 1991 gonococcal conjunctivitis outbreak. Two of these carriers were children under 10 years of age. During the same outbreak three individuals had confirmed gonococcal conjunctivitis as well as oropharyngeal carriage

of *N. gonorrhoeae*. It was thought there was autoinoculation of the oropharynx via the nasolacrimal duct. In one report gonococci were detected in the saliva of pharyngeal carriers.[10] Autoinoculation may also occur from purulent eyes to hands to mouth and throat. There may be autoinoculation to the eyes from the throat via saliva. The prevalence of pharyngeal carriage in outbreaks of non-sexually transmitted gonococcal conjunctivitis in Central Australia is unknown.

Oropharyngeal carriage of *N. gonorrhoeae* is more difficult to eradicate than uncomplicated infections at other mucosal sites.[11] The cure rate for a single dose treatment with less effective antibiotic regimens is approximately 70%. The most highly effective single dose treatments are likely to cure at least 80% of pharyngeal infections, although the same treatments will cure greater than 95% of uncomplicated anogenital gonorrhoea infections. The failure to eliminate pharyngeal carriage may have three consequences:

1. the probability of a continuing reservoir of infection to further infect the community;

2. the possibility of recurrent infection in the individual; and

3. a possible source of disseminated gonococcal infection (DGI) in an individual.

DGI in a 3 year old boy was documented as a complication in the 1991 epidemic.[4] Two cases of DGI were diagnosed at the ASH in May 1997. *N. gonorrhoeae* was isolated from the joint fluid of a 14 year old boy and a 26 year old woman. The isolate from the boy was serovar IB3. Unfortunately the isolate from the woman was not kept for testing. Both of these patients had no history of sexually transmitted *N. gonorrhoeae* at the time and both lived in communities which were affected by the outbreak.

Control measures

Central Australian outbreaks have been occurring approximately every five years since 1981. We have enough information to help predict when an outbreak is likely to occur and communities should monitor their populations in these circumstances to detect index cases. If a case is detected, the relevant Public Health and Disease Control Centres should be notified immediately. Interstate Disease Control Centres also need to be notified so all adjacent communities can be informed of a possible epidemic.

Following the collection of the specimen, including a direct smear, treatment should be given to cases and their close contacts prior to laboratory confirmation as some specimens, due to transportation availability, can take up to one week to reach the laboratory. Direct smears are essential for a rapid diagnosis. PCR is more sensitive than either culture or smear but culture is essential for antibiotic susceptibility monitoring.

Once the disease is established, contact tracing can become extremely difficult due to limited local resources. The epidemic is self limiting once the weather becomes colder and the majority of clinical cases have been treated, but sporadic cases can still occur, as can be seen in the Kimberley outbreak (Figure 1). With favourable conditions, a further epidemic could eventuate.

Treatment failures must be detected rapidly. Community members must be educated on the transmission of the disease from eye to eye between children. Poor hygienic practices have been observed in previous outbreaks, for example, wiping an infected child's eyes and then using the same material to wipe another child's face.[4]

Flies may act as a mechanical vector to establish index cases from reservoirs of *N. gonorrhoeae* and then, along with person to person transmission, contribute to the spread of the disease. However, fly control is virtually impossible during the start of the outbreak because of the extremely high population of flies.

Early recognition and treatment of index cases and identifying and treating contacts is currently the only way of preventing an epidemic. Strategies are in place to detect and treat sexually transmitted *N. gonorrhoeae* in communities. Until this reservoir of disease is controlled gonococcal conjunctivitis is likely to appear again. The role of oropharyngeal carriage of *N. gonorrhoeae* needs to be evaluated further.

Acknowledgments

We would like to thank Nganampa Health Council, Ngaanyatjarra Health Service and community clinic staff; Western Diagnostic Pathology staff and microbiology staff - Pathology Dept, ASH; Virginia Sitzler - Disease Control Centre, Alice Springs and John Tapsall, Department of Microbiology, Prince of Wales Hospital, Sydney.

References

1. Kirkland W. Endemic and epidemic diseases in the NT. Report of the National Health and Medical Research Council Sixth Session, Adelaide SA, 24-25 May 1939. Adelaide: Government Printer 1939.
2. Matters R. Non-sexually transmitted gonococcal conjunctivitis in Central Australia. *Comm Dis Intell* 1981; 13:3.
3. Brennan R, Patel M, Hope A. Gonococcal conjunctivitis in Central Australia. *Med J Aust* 1989; 150:48-49.
4. Merianos A, Condon R, Tapsall J, Jayathissa S, Mulvey G, Lane J, Patel M, Rouse I. Epidemic gonococcal conjunctivitis in Central Australia. *Med J Aust* 1995; 162:178-181.
5. Monger K, Brennan R. Gonococcal conjunctivitis outbreak in a Northern Territory Aboriginal community. *Comm Dis Intell* 1992; 16(25):534.
6. Skov S, Miller P, Hateley W, Bastian I, Davis J, Tait P. Urinary diagnosis of gonorrhoea and chlamydia in men in remote Aboriginal communities. *Med J Aust* 1997; 166:468-471.
7. Hughes R, Greenham P, Tyndale-Biscoe M, Walker J. A synopsis of observations on the biology of the Australian bushfly (*Musca vetustissima* Walker). *J Aust Ent Soc* 1972; 11:311-331.
8. Matthiessen J, Hall G, Chewings V. Seasonal abundance of *Musca vetustissima* Walker and other cattle dung fauna in Central Australia. *J Aust Ent Soc* 1986; 25:141-147.
9. Condon R. Gonococcal conjunctivitis in the Ngaanyatjarra homelands of Western Australia. April-June 1991. Health Department of Western Australia Occasional Paper 1991/44. Perth: Health Department of Western Australia, 1991.
10. Hutt D, Judson F. Epidemiology and treatment of oropharyngeal gonorrhoeae. *Ann Intern Med* 1986; 104:655-658.
11. Moran J. Treating uncomplicated *Neisseria gonorrhoeae* infections: is the anatomic site of infection important? *Sex Transm Dis* 1995; 22(1):39-47.

Editorial comment

Vicki Krause

Centre for Disease Control, Territory Health Services, Northern Territory

The above is a comprehensive review of the most recent large outbreak of non-sexually transmitted gonococcal conjunctivitis. It raises several interesting points regarding the circumstances which 'set the scene' for such an outbreak, the possible methods of disease transmission and treatment considerations. Most importantly, however, it highlights to health care providers that childhood conjunctivitis may be caused by *N. gonorrhoeae* which is highly contagious and requires systemic antibiotics. This contrasts with the more common causes of conjunctivitis: allergy, viral infection and infection with the non gonococcal bacteria *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Streptococcus pyogenes* and *Staphylococcus* species; which are usually treated topically.[1]

The possibility of *N. gonorrhoeae* needs to be considered in childhood conjunctivitis, especially in areas with endemic high prevalence of venereal gonorrhoea disease and environmental factors which may promote its spread to the eyes. Swabs and cultures should be obtained to establish the diagnosis. The use of air-dried smears to detect Gram negative diplococci is useful for presumptive diagnosis in remote communities where transporting specimens to regional laboratories may be delayed and cultures unsuccessful.[2]

This outbreak as well as past experience[2] suggests that one case of non-sexually transmitted gonococcal conjunctivitis should be treated as a potential outbreak. Effective control measures include a standardised approach to case and contact management. Single dose treatment with an appropriate penicillin is recommended[2,3,4] as it reduces the problem of multidose treatment, especially in a mobile population. Standard and alternative treatment recommendations are shown below. Alternative treatment regimens are indicated for: patients allergic to penicillin; when standard treatment has failed; when infection is known to be due to penicillinase producing *N. gonorrhoeae* (PPNG); or, where a pharyngeal swab has been taken and is positive.

In this outbreak the greatest number of cases were in the 5 to 9 years age group, whereas the highest attack rate in the large 1991 epidemic was in the 0 to 4 years age group. The carers of cases under 10 years need to be advised to monitor the treated child for persisting eye infection, reinfection, fevers or other symptoms such as arthritis. Cases should be excluded from school or child care for 24 hours after treatment has been given. Where neonatal infection is found a full STD screen is indicated for the mother.

Gonorrhoea is a notifiable disease and, in view of the potential for the very rapid spread of this clinical form, gonococcal conjunctivitis should be immediately reported by phone or facsimile to the local Disease Control or Public Health Unit. Early reporting will assist in contact tracing and enable adjacent health services and communities to be informed.

Treatment for gonococcal conjunctivitis

Standard

Neonates < 1 month (Ophthalmia neonatorum)	Admit to hospital urgently for intravenous antibiotics		
Older infants, children and adults	Procaine penicillin intramuscularly (IM) as a single dose 50,000 units/kg (50 mg/kg) to a maximum of 1,500,000 units or 1.5g		
	OR		
	Amoxycillin plus probenecid as a single dose		
	Weight	Amoxycillin	Probenecid
	3kg to <6kg	500mg	nil
	6kg to <10kg	1g	nil
	10kg to 15kg	1.5g	250mg
	15kg to <20kg	2g	500mg

Alternative

Infants <6 weeks of age	Should not be given ceftriaxone. Refer to hospital	
Older infants, children and adults	Weight ≤25 kg	Ceftriaxone - 125 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose
	Weight >25 kg	Ceftriaxone - 250 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose

Household contacts of cases should be treated with a single dose of a standard treatment or alternative treatment if allergic to penicillin. If a case attends a childcare centre or school the childcare or class mates should be treated. For cases and contacts, and their families, emphasis should be given to thorough hand and face washing, the use of individual clean towels and to ensuring that these are available in affected households or schools.

It is reassuring to know that the penicillin MICs for the Central Australian isolates continue to fall in the fully sensitive to less sensitive range, 0.0125 to 0.5mg/L, and that PPNG has not emerged as a problem in Central Australia and in the NT[5]. Standard treatment, therefore, is still adequate to treat conjunctivitis caused by the current strains of *N. gonorrhoeae*. While recognising the diagnostic role for air-dried smears in remote settings, and for PCR testing as used in the Kimberly, it is prudent to culture and susceptibility test at least sentinel samples during an outbreak in order to monitor the antibiotic MICs.

The place, if any, of pharyngeal carriage as a reservoir for transmission does need to be further considered. However, the ability to control outbreaks with penicillins, and without ceftriaxone or spectinomycin, suggests that pharyngeal carriage was not an important contributor to this outbreak. Pharyngeal swabs are not routinely recommended in outbreaks unless patients are symptomatic. A preparedness to investigate this issue in the event of another outbreak should be considered.

References

1. Victorian Drug Usage Advisory Committee. Antibiotic Guidelines 1996-1997, 9th edition. North Melbourne: Victorian Medical Postgraduate Foundation Therapeutics Committee, 1998.
2. Merianos A, Condon R, Tapsell J. Epidemic gonococcal conjunctivitis in Central Australia. *Med J Aust* 1995;162:178-181.
3. Center for Disease Control. Guidelines for the control of gonococcal conjunctivitis. Darwin: Territory Health Services, 1997.
4. Central Australian Rural Practitioners Association. Standard Treatment Manual, 3rd edition. Alice Springs: Central Australian Rural Practitioners Association, 1997.
5. Australian Gonococcal Surveillance Program. Annual report of the Australian Gonococcal Surveillance Programme 1996. *Comm Dis Intell* 1997;21:189-192.

Current issues in immunisation

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS), Royal Alexandra Hospital for Children, Westmead, New South Wales.

The NCIRS was established by the National Centre for Disease Control, Commonwealth Department of Health and Family Services. The Centre analyses, interprets, and evaluates national surveillance data on immunisation coverage and vaccine preventable diseases. NCIRS also identifies research priorities, and initiates and coordinates research on immunisation issues and the epidemiology of vaccine preventable diseases in Australia.

This occassional report series in Communicable Diseases Intelligence provides commentary on topical immunisation issues.

Measles vaccine, inflammatory bowel disease and pervasive developmental disorder: is there cause for concern?

Janaki Amin, Peter B. McIntyre, Timothy C. Heath

On 28 February 1998, the *Lancet* published a report of a case series from the Royal Free Hospital, London suggesting a temporal association between measles-mumps-rubella (MMR) vaccine and an apparently new syndrome, consisting of an unusual type of inflammatory bowel disease (IBD) with pervasive developmental disorder (PDD).[1] This report was published with an editorial by Dr Robert Chen,[2] head of the Vaccine Safety and Development Activity National Immunization Program, US Centers for Disease Control and Prevention, which refuted its conclusion. Despite this, the subsequent intense media attention and public concern has challenged the integrity of the MMR immunisation program in the United Kingdom.

The hypothesis generated by the Royal Free Hospital group is that MMR is associated with IBD, and that IBD is associated with PDD. To examine this further, both microbiological and epidemiological evidence need to be considered.

The microbiological evidence

An association between wild and vaccine strains of the measles virus and IBD has been postulated since 1993.[3] Previous studies by the Royal Free group have reported detection of measles vaccine viruses in biopsies from patients with IBD, but other investigators have not been able to reproduce their findings.[4,5] Using nested polymerase chain reaction (PCR), a much more specific test than those used by the Royal Free Hospital group, Azafal et al.,[6] could not detect measles virus in the gut mucosal biopsies of patients with Crohn's disease or ulcerative colitis. The recent Royal Free Hospital study provided no evidence of vaccine virus in the bowel, brain or any other tissue of the reported subjects.

The epidemiological evidence

As highlighted in the *Lancet* editorial,[2] the Royal Free Hospital report is essentially one of hypothesis generation. The study design is a case series and does not enable conclusions to be drawn about causation. In addition, both selection and recall biases are likely to have affected the findings.

In the Royal Free Hospital case series, any association between MMR and IBD is likely to be inflated by selection bias arising from the referral of subjects to a group known to

be specially interested in an association between measles and IBD.

In 1997, a Canadian review of studies supporting and disputing the association between IBD and measles concluded that 'current scientific data do not permit a causal link to be drawn between the measles virus and chronic inflammatory bowel disease'.[7] A subsequent World Health Organization (WHO) report[8] updated the Canadian review and included a British case control study.[9] The WHO report found no additional support for an association between measles (disease or immunisation) and Crohn's disease.

The Royal Free Hospital group acknowledge that the reported intestinal and behavioural pathologies may have occurred together by chance, reflecting a selection bias in a self-referred group. Alternatively, they suggest that intestinal disease may play a part in the behavioural changes and cite three papers to support this hypothesis.[1] However, a recent French population-based study of conditions associated with autism found no link to IBD.[10] British data indicate a rise in the incidence of autism, but show that this started over a decade before the introduction of MMR in 1988, with no change since that time.[11] A recent review of British autistic spectrum disorders and medical disorders databases found that the incidence of IBD was nil among autistic children born since the introduction of MMR.[12] It is of note that, in the current Royal Free Hospital study, most subjects exhibited behavioural changes prior to bowel symptoms, arguing against the researchers' proposal that IBD is the antecedent for PDD.[1]

The association between vaccination and PDD was primarily based on parental recall. Estimating the time of onset of autistic behaviour is difficult. In addition, parents are likely to have linked this behaviour to other memorable events which occur at a similar age, such as immunisation. Onset of autism and MMR vaccination may appear associated in time because the mean age of children at which parents first report concern about child development is 18 to 19 months,[13] and over 90% of children receive MMR vaccine before their second birthday.[14]

Public health impact

Parental response to previous media coverage of the measles-IBD link is thought to have resulted in a 1% fall in MMR vaccine coverage during August to December 1996 in the United Kingdom.[15] While this was a small reduction, there are fears that further adverse press publicity about the current Royal Free Hospital study could result in a sustained fall in MMR immunisation, as occurred with pertussis in the 1970s following anecdotal reports that linked infant pertussis vaccination with brain damage.

The press release issued by the authors of the Royal Free Hospital paper stated 'The majority of opinion among the researchers involved in this study support the continuation of MMR vaccination'.[16] However Dr Wakefield, one of the study authors, is reported as saying 'that until this issue is resolved by further research there is a case for separating the three vaccines into separate measles, mumps and rubella components and giving them individually spaced by at least 1 year'.[16] This comment by Dr Wakefield was publicised by the media in the United Kingdom and led to greatly increased requests for separate doses of measles, mumps and rubella vaccines.

Implications for Australia

In view of Australia's proposed national Measles Control Program, information regarding the safety and side effects of MMR vaccination needs to be clearly communicated. On 23 March 1998, Sir Kenneth Calman, British Chief Medical Officer, convened a meeting of the Medical Research Council (MRC) and a group of national and international experts, including the World Health Organization, to review the work of the Royal Free Hospital Inflammatory Bowel Disease Study Group. Sir Kenneth concluded that based on current evidence 'there is no link between measles, measles vaccine, and either Crohn's Disease or autism'; 'there is no evidence that giving the component vaccines separately has any benefit'; and that 'giving the vaccines separately may even be harmful because it would expose children and their contacts to these serious diseases over a much longer period'.[17] We endorse this interpretation and consider that it needs to be confidently conveyed to both health professionals and the public.

1. Wakefield AJ, Murch SH, Anthony A, Linnell J, Casson DM, Malik M, et al. Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *Lancet* 1998; 351:637-641.
2. Chen RT, DeStefano F. Vaccine adverse events: causal or coincidental? (Comment). *Lancet* 1998; 351:611-612.
3. Wakefield AJ, Pittilo RM, Sim R, Cosby SL, Stephenson JR, Dhillon AP, et al. Evidence of persistent measles virus infection in Crohn's disease. *J Med Virol* 1993; 39:345-353.
4. Iizuka M, Nakagomi O, Chiba M, Ueda S, Masamune O. Absence of measles virus in Crohn's disease (letter). *Lancet* 1995; 345:199.
5. Haga Y, Funakoshi O, Kuroe K, Kanazawa K, Nakajima H, Saito H, et al. Absence of measles viral genomic sequence in intestinal tissues from Crohn's disease by nested polymerase chain reaction. *Gut* 1996; 38:211-215.
6. Afzal MA, Minor PD, Begley J, Bentley ML, Armitage E, Ghosh S, et al. Absence of measles-virus genome in inflammatory bowel disease (letter). *Lancet* 1998; 351:646-647.
7. Ward B, DeWals P. Association between measles infection and the occurrence of chronic inflammatory bowel disease. *Can Commun Dis Rep* 1997; 23:1-5.
8. Anonymous. Expanded Programme on Immunization (EPI). Association between measles infection and the occurrence of chronic inflammatory bowel disease. *Wkly Epidemiol Rec* 1998; 73:33-39.
9. Feeney M, Ciegg A, Winwood P, Snook J. A case-control study of measles vaccination and inflammatory bowel disease. The East Dorset Gastroenterology Group. *Lancet* 1997; 350:764-766.
10. Fombonne E, Du Mazauabrun C, Cans C, Grandjean H. Autism and associated medical disorders in a French epidemiological survey. *Am Acad Child Adolesc Psychiatry* 1997; 36:1561-1569.
11. Nicoll A, Elliman D, Ross E. MMR vaccination and autism 1998 (Editorial). *BMJ* 1998; 7133:715.
12. Fombonne E. Inflammatory bowel disease and autism. *Lancet* 1998; 351:955.
13. Siegel B, Pliner C, Eschler J, Elliott GR. How children with autism are diagnosed: difficulties in identification of children with multiple developmental delays. *J Dev Behav Pediatr* 1988; 9:199-204.
14. Miller E. Reported association between measles, mumps and rubella (MMR) vaccine, autism and bowel syndrome. *Eurosurveillance* 1998; 2:2-3.
15. Anonymous. MMR vaccine coverage falls after adverse publicity. *Comm Dis Rev* 1998;8(5):41.
16. Handysides S. Public health, science and news media. *Eurosurveillance* 1998; 2:1-2.
17. Department of Health. MMR vaccine is not linked to Crohn's disease or autism, press release (98/109).1998. http://www.open.gov. uk

Japanese encephalitis on the Australian mainland

Japanese encephalitis has been diagnosed in an adult male in Queensland. The man who has recovered and been discharged from hospital is believed to have acquired the virus while working on a boat on the west coast of Cape York Peninsula. Queensland Health, the Australian Quarantine and Inspection Service and the Department of Primary Industries are working to determine the source of virus and to contain its spread. This will include testing residents of two nearby Cape York communities for evidence of infection with the virus. Local pig populations will also be tested.

This is the first case of Japanese encephalitis to be diagnosed on the Australian mainland. In 1995 three cases, including two deaths, were reported in the outer Torres Strait islands. A further case was reported in the Torres Strait in 1998.

Murray Valley encephalitis in Western Australia

A case of Murray Valley encephalitis has been diagnosed in a young male from Western Australia who is now recovering in hospital. The evidence of infection has also been detected in sentinel chickens in Wyndham in the north east of the State. Only one or two out of every 1,000 persons infected with the virus develop serious disease. Symptoms include fever, headache, nausea and vomiting and convulsions. No specific treatment is available. Local residents and visitors have been reminded to avoid mosquito bites by wearing loose fitting clothing, using insect repellant, and ensuring that fly screens on houses and caravans are in working order.

Notice to readers

Control of Communicable Diseases in Australia Conference

under the auspices of the Communicable Diseases Network Australia New Zealand (CDNANZ)

10 November 1998, Canberra

Call for abstracts

Control of communicable diseases continues to be one of the highest public health priorities both nationally and internationally. Emerging and re-emerging microbial threats and drug resistance pose an ever increasing challenge for public health practitioners. Added to this challenge are high public expectations of protection from public health hazards, increasing scrutiny from the media, lawyers, and politicians.

This conference will study public health communicable disease control issues and examine investigations of recent disease outbreaks in Australia.

This conference is for anyone working in the field of public health or communicable diseases: officers of local, State/Territory and Commonwealth health departments; health practitioners involved in communicable diseases and infection control; epidemiologists; microbiologists; infectious disease physicians; environmental health officers and public health officers.

For abstract submission, registration forms and further information please contact:

Miss Alison Milton
National Centre for Disease Control, MDP 6
Department of Health and Family Services
GPO Box 9848
Canberra ACT 2601

Phone (02) 62898245
Fax (02) 62897791
email ccd.conf@health.gov.au

Communicable Diseases Surveillance

Communicable Diseases Surveillance consists of data from several sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. The Australian Sentinel Practice Research Network (ASPREN) is a general practitioner-based 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'. Data from the LabVISE scheme are referred to as 'laboratory reports'.

Vaccine preventable diseases

Although the epidemic of pertussis in Australia continues, the number of notifications is declining, which is consistent with the seasonal drop expected in the first few months of the year. The majority of reports of pertussis with onset in 1998 have been for children aged 0 to 4 years (15%), 5 to 9 years (20%) and 10 to 14 years (15%). The male:female ratio was 1:1.1.

Measles notifications have remained relatively low for the past 3 years, with a small increase in activity in the last quarter of 1997, as would be expected for the time of year (Figure 1). The majority of cases with onset in 1998 were reported from Victoria (34%), New South Wales (25%) and Queensland (18%). In 1997 and 1998 most cases were in children under 5 years of age (75%), with an overall male:female ratio of 1.0:1 (Figure 2).

Arboviruses

The number of notifications of Ross River virus infection remains low for the time of year (Figure 3). Similarly few cases of Barmah Forest virus infection have been notified compared to recent years (Figure 4).

One hundred and seven cases of dengue were notified this period, bringing the total for the year so far to 183. All but 10 of the current notifications had a recorded date of onset before March (Figure 5). Of the 186 cases with onset since

Figure 1. Notifications of measles, 1991 to 1998, by month of onset

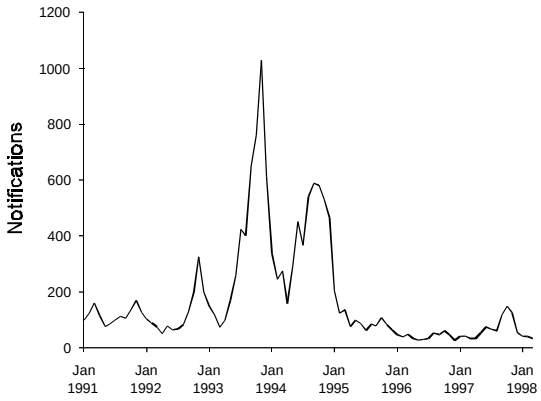


Figure 2. Notifications of measles, 1997 and 1998, by age group and sex

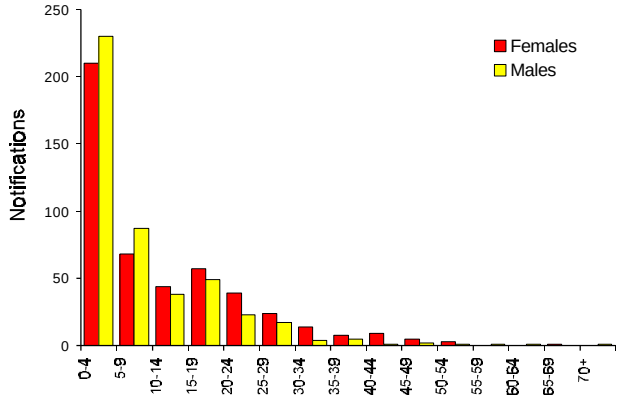


Figure 3. Notifications of Ross River virus infection, 1991 to 1998, by month of onset

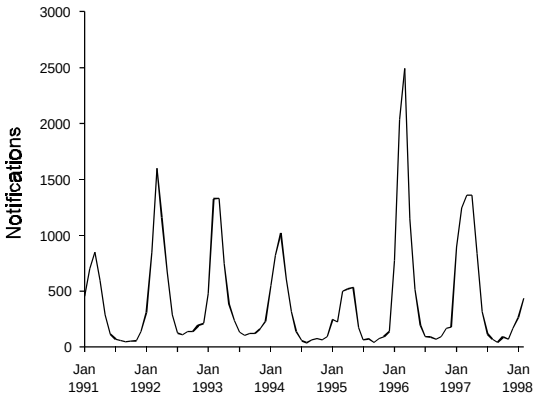


Figure 4. Notifications of Barmah Forest virus infection, 1995 to 1998, by month of onset

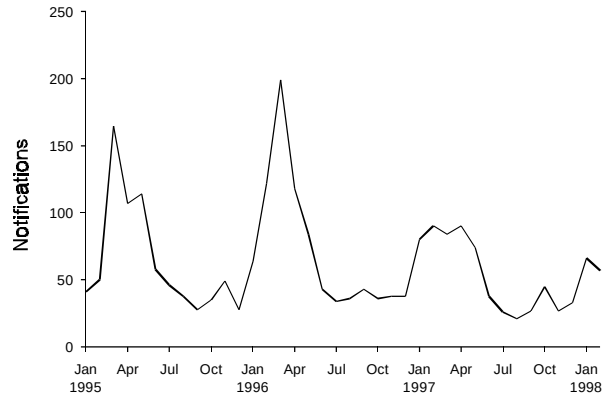


Figure 5. Notifications of dengue, 1991 to 1998, by month of onset

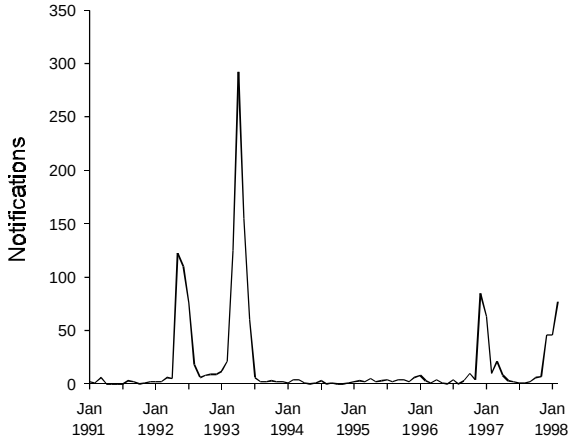
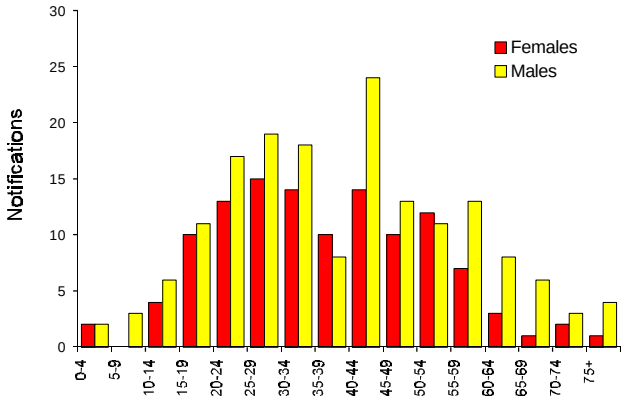


Figure 6. Notifications of dengue, 1997 and 1998, by age group and sex



November 1997, 140 (75%) were residents of the Queensland Statistical Division of Far North. In the 1996-97 outbreak, a similar pattern was observed, a larger proportion (88%) being residents of the same Statistical Division of Far North. In the recent outbreak the male:female ratio was 1.3:1 and 66% of cases were aged between 25 and 59 years (Figure 6).

Enteric infections

Hepatitis A was notified for 240 persons this period, of which 113 (47%) were from Queensland. The number of cases rose in January and February but remain below the peak seen in early 1997 (Figure 7). Of the 740 cases reported with onset in 1998 so far, 31% were for the 20 to 29 years age group, the male:female ratio for this group being 2.3:1 (Figure 8).

Fifty-six laboratory reports of hepatitis A were received by the sentinel laboratory scheme LabVISE this period, 68% of which were from Queensland. Included were 37 males and 19 females, a male:female ratio of 2.0:1. Forty-one per cent of reports were for those in the 25-44 years age group.

The number of notifications of salmonellosis rose in late 1997 and early 1998 but remain below the level seen for the same period last year (Figure 9). Overall for 1997, 6,830 notifications of this disease were reported with onset in that year, of which 36% were for children under the age of 5 years.

The number of cases of campylobacteriosis reported to the NNDSS remains low compared to the same period last year. One thousand eight-hundred and forty-one notifications with onset in 1998 have been received so far. Of these 18% of cases were under the age of 5 years and 22% were in the 20-29 years age group.

The LabVISE scheme recorded 5 cases of echovirus type 11 this reporting period, all from New South Wales. Of these 4 had specimen collection dates in January, which is usually the peak month for enterovirus activity in Australia. Included were 3 males aged 0 to 5 years and 2 females both of whom were in the 15-44 years age group.

Rhinovirus

The number of rhinovirus laboratory reports has fallen in recent months after peaking last September. For 1997 a total of 550 laboratory reports was received of which 85% were for children under the age of 5 years.

Correction: The laboratory report of Stratford virus from Western Australia in last issue of *CDI* was incorrect. This was a case of Japanese encephalitis which was acquired in Vietnam. The patient presented with late neuropsychiatric sequelae.

Figure 7. Notifications of hepatitis A, 1995 to 1998, by month of onset

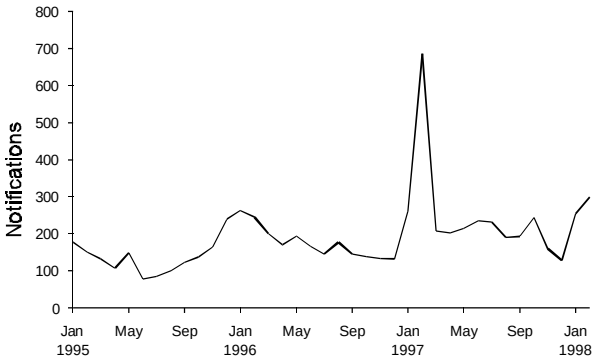


Figure 8. Notifications of hepatitis A, 1998, by age group and sex

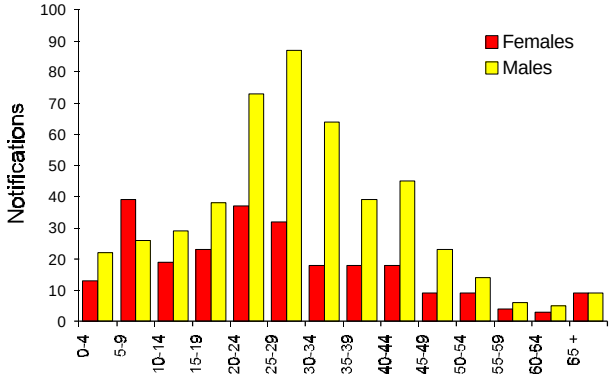
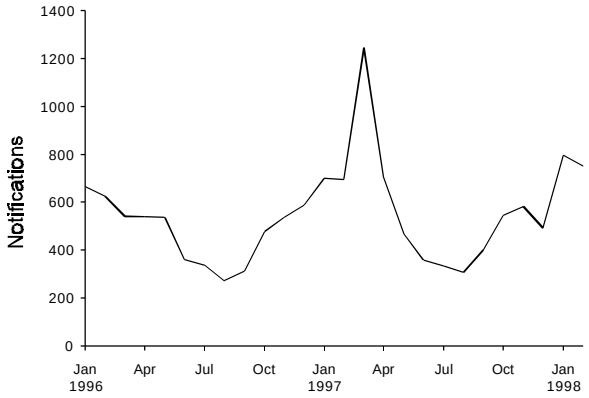


Figure 9. Notifications of salmonellosis, 1996 to 1998, by month of onset



There were 5,078 notifications received for this four-week period, 4 to 31 March 1998 (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 10).

NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of more than 40 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1998;22:4-5.

There were 1,047 reports received in the CDI Virology and Serology Laboratory Reporting Scheme (LabVISE) this four week period, 26 February to 25 March (Tables 4 and 5). Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence every four weeks. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1998;22:8.

Table 1. Notifications of rare[1] diseases received by State and Territory health authorities in the period 4 to 31 March 1998

Disease[2]	Total this period	Reporting States or Territories	Total notifications 1998
Brucellosis			13
Cholera	1	Qld	2
Hydatid infection	1	Vic	6
Leprosy			1

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1998.
2. No notifications have been received during 1998 for the following rare diseases: botulism, lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 9 to 12 ending 29 March 1998 are included in this issue of CDI (Table 6). ASPREN currently comprises about 100 general practitioners from throughout the country. Up to 9,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance. CDI reports the consultation rates for all of these. For further information, including case definitions, see CDI 1998;22:5-6.

HIV and AIDS Surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648 Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for October 1998, as reported to 31 January 1998, are included in this issue of CDI (Tables 7 and 8).

Table 2. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 4 to 31 March 1998

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
H. influenzae type b infection	0	1	0	0	0	0	0	2	3	2	6	15
Measles	3	11	0	6	0	0	13	3	36	41	129	119
Mumps	1	7	0	0	1	0	6	3	18	22	45	46
Pertussis	0	190	3	100	54	2	10	29	388	629	2,472	2,261
Rubella[3]	0	5	2	15	2	1	11	7	43	74	154	453
Tetanus	0	0	0	0	0	0	0	0	0	1	1	2

NN. Not Notifiable

1. No notifications of poliomyelitis have been reported since 1986.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. Includes congenital rubella

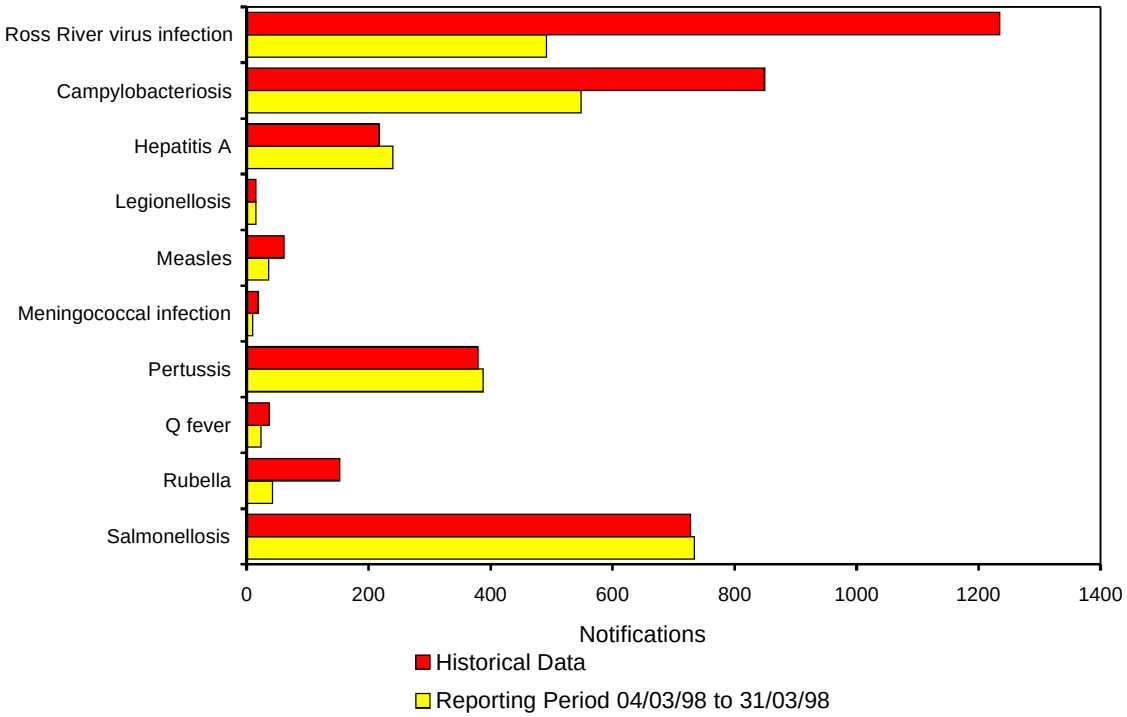
Table 3. Notifications of other diseases received by State and Territory health authorities in the period 4 to 31 March 1998

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
Arbovirus infection (NEC)[3]	0	6	2	3	0	0	0	1	12	15	25	56
Barmah Forest virus infection	0	10	-	38	0	0	0	-	50	85	172	237
Campylobacteriosis[4]	40	-	18	274	78	11	20	107	548	778	2,175	2,983
Chlamydial infection (NEC)[5]	10	NN	68	254	0	18	142	141	633	665	2,155	2,006
Dengue	0	5	0	100	0	0	0	2	107	11	183	101
Donovanosis	0	NN	3	0	NN	0	0	0	3	7	13	8
Gonococcal infection[6]	1	53	99	96	0	1	46	101	397	334	1,244	849
Hepatitis A	4	88	4	113	10	0	11	10	240	224	822	1,159
Hepatitis B incident	0	2	2	3	0	0	0	0	7	19	40	58
Hepatitis C incident[7]	0	3	0	-	0	0	-	-	3	1	19	2
Hepatitis C unspecified	29	NN	30	207	NN	13	3	91	373	660	1,297	2,103
Hepatitis (NEC)	0	4	0	0	0	0	0	NN	4	1	5	7
Legionellosis	0	2	1	2	1	0	9	0	15	12	55	38
Leptospirosis	0	3	1	6	0	0	2	0	12	4	39	30
Listeriosis	0	6	0	0	1	0	0	0	7	7	20	24
Malaria	3	12	3	0	1	0	11	8	38	40	163	164
Meningococcal infection	0	0	0	5	2	0	2	1	10	15	48	63
Ornithosis	0	NN	0	0	0	1	0	0	1	5	5	22
Q Fever	1	5	0	15	0	0	1	2	24	36	95	138
Ross River virus infection	0	34	17	400	5	2	8	26	492	1,193	1,079	2,849
Salmonellosis (NEC)	12	128	66	331	56	6	74	61	734	774	2,396	2,171
Shigellosis[4]	3	-	9	16	7	0	4	15	54	75	188	256
Syphilis[8]	2	36	23	28	0	2	0	4	95	123	304	333
Tuberculosis	0	16	3	10	4	1	31	2	67	86	241	264
Typhoid[9]	0	5	0	0	0	0	1	0	6	9	34	27
Yersiniosis (NEC)[4]	0	-	0	9	0	0	1	0	10	29	82	99

1. For HIV and AIDS, see Tables 7 and 8. For rarely notified diseases, see Table 1.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. NT: includes Barmah Forest virus.
4. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
5. WA: genital only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. Qld, Vic and WA incident cases of Hepatitis C are not separetly reported.
8. Includes congenital syphilis
9. NSW, Vic: includes paratyphoid.

NN Not Notifiable.
NEC Not Elsewhere Classified
- Elsewhere Classified.

Figure 10. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



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

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 26 February to 25 March 1998, and total reports for the year

	State or Territory[1]								Total this period	Total reported in *CDI* in 1998
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles, mumps, rubella										
Measles virus							9	1	10	30
Mumps virus							1		1	9
Rubella virus		1		6				4	11	35
Hepatitis viruses										
Hepatitis A virus		4	4	38			1	9	56	115
Arboviruses										
Ross River virus		1	12	85			2	22	122	348
Barmah Forest virus			2						2	10
Dengue not typed								1	1	6
Kunjin virus								1	1	1
Flavivirus (unspecified)				5					5	23
Adenoviruses										
Adenovirus not typed/pending		12		1			5	1	19	163
Herpes viruses										
Herpes virus type 6								1	1	2
Cytomegalovirus		7		26	1		19	4	57	267
Varicella-zoster virus		13		24			16	12	65	360
Epstein-Barr virus		6	9	33			8	53	109	494
Other DNA viruses										
Parvovirus				2			5		7	42

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 26 February to 25 March 1998, and total reports for the year, continued

	State or Territory[1]								Total this period	Total reported in *CDI* in 1998
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Picorna virus family										
Echovirus type 9							1		1	1
Echovirus type 11		5							5	8
Poliovirus type 2 (uncharacterised)		1							1	1
Rhinovirus (all types)		9					2	5	16	136
Enterovirus not typed/pending		3		5			1	7	16	95
Ortho/paramyxoviruses										
Influenza A virus		1						2	3	121
Influenza B virus								3	3	45
Parainfluenza virus type 1		7		1			13		21	53
Parainfluenza virus type 3		1					2	13	16	154
Respiratory syncytial virus		5		4			3	2	14	218
Other RNA viruses										
Rotavirus		5							5	90
Astrovirus							5		5	8
Norwalk agent							4		4	17
Other										
Chlamydia trachomatis not typed		22	89	59			1	108	279	1,030
Chlamydia species		7							7	9
Mycoplasma pneumoniae		8	3	45			33	5	94	470
Coxiella burnetii (Q fever)				7			1	2	10	25
Bordetella pertussis		1	1	32			32	13	79	509
TOTAL		119	120	373	1		164	270	1,047	4,896

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

Table 5. Virology and serology laboratory reports by contributing laboratories for the reporting period 26 February to 25 March 1998

State or Territory	Laboratory	Reports
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	54
	New Children's Hospital, Westmead	26
	Royal Prince Alfred Hospital, Camperdown	20
Queensland	Queensland Medical Laboratory, West End	397
Victoria	Monash Medical Centre, Melbourne	65
	Royal Children's Hospital, Melbourne	38
	Victorian Infectious Diseases Reference Laboratory, Fairfield	64
Western Australia	PathCentre Virology, Perth	137
	Western Diagnostic Pathology	246
TOTAL		1,047

Table 6. Australian Sentinel Practice Research Network reports, weeks 9 to 12, 1998

Week number	9		10		11		12	
Week ending on	8 March 1998		15 March 1998		22 March 1998		29 March 1998	
Doctors reporting	51		55		53		49	
Total consultations	6,831		7,018		7,298		6,609	
Condition	Reports	Rate per 1,000 consultations	Reports	Rate per 1,000 consultations	Reports	Rate per 1,000 consultations	Reports	Rate per 1,000 consultations
Influenza	13	1.9	14	2.0	11	1.5	10	1.5
Rubella	1	0.1	3	0.4	2	0.3	0	0.0
Measles	1	0.1	0	0.0	0	0.0	0	0.0
Chickenpox	5	0.7	3	0.4	7	1.0	5	0.8
Pertussis	3	0.4	1	0.1	1	0.1	3	0.5
HIV testing (patient initiated)	14	2.0	8	1.1	15	2.1	9	1.4
HIV testing (doctor initiated)	6	0.9	7	1.0	4	0.5	4	0.6
Td (ADT) vaccine	48	7.0	58	8.3	55	7.5	46	7.0
Pertussis vaccination	44	6.4	36	5.1	39	5.3	41	6.2
Reaction to pertussis vaccine	3	0.4	0	0.0	2	0.3	0	0.0
Ross River virus infection	0	0.0	1	0.1	0	0.0	0	0.0
Gastroenteritis	99	14.5	90	12.8	94	12.9	87	13.2

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 October 1997, by sex and State or Territory of diagnosis

										Totals for Australia		
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
HIV diagnoses	Female	0	4	1	0	0	0	1	0	6	3	64	59
	Male	0	20	0	1	3	0	10	5	39	72	539	686
	Sex not reported	0	4	0	0	0	0	0	0	4	1	25	5
	Total[1]	0	28	1	1	3	0	11	5	49	76	628	751
AIDS diagnoses	Female	0	0	0	1	0	0	0	0	1	2	21	25
	Male	0	5	0	2	1	0	4	2	14	38	244	536
	Total[1]	0	5	0	3	1	0	4	2	15	40	265	561
AIDS deaths	Female	0	0	0	0	0	0	1	0	1	0	11	15
	Male	0	3	0	0	2	0	6	1	12	34	188	413
	Total[1]	0	3	0	0	2	0	7	1	13	34	200	428

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

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 October 1997, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	21	504	6	114	49	4	191	81	970
	Male	179	10,642	92	1,773	623	77	3,637	834	17,857
	Sex not reported	0	2,066	0	1	0	0	28	1	2,096
	Total[1]	200	13,225	98	1,893	672	81	3,866	919	20,954
AIDS diagnoses	Female	7	157	0	42	19	2	59	23	309
	Male	80	4,301	30	745	318	41	1,495	333	7,343
	Total[1]	87	4,469	30	789	337	43	1,561	358	7,674
AIDS deaths	Female	2	112	0	27	14	2	43	14	214
	Male	52	3,025	23	522	214	26	1,190	240	5,292
	Total[1]	54	3,144	23	551	228	28	1,239	255	5,522

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

Overseas briefs

Source: World Health Organization (WHO) and Pacific Public Health Network

Cholera

Comoros Islands (update). As of 11 March, 945 cases of cholera with 18 deaths (case fatality rate 1.9%) had been reported since early January when the epidemic began. Over 50% (476) of cases occurred in Moroni. The other 469 cases were in the health districts of Mitsamiouli (259), Mbéni (105), Foumbouni (80), Ouzioini (18), and Mitsoudjé (7). All cases occurred on Grand Comore Island. The National Cholera Control Committee has been set up in collaboration with WHO to implement a control plan. Control measures include improved personal, domestic and environmental hygiene, particularly the supply of clean water.

Zimbabwe. A total of 335 cases with 12 deaths were reported up to 27 March 1998 in Zimbabwe. These were the first reported cases in Zimbabwe since 1993 when 5,385 cases, including 332 deaths were notified to the WHO. The government, in collaboration with the WHO and other agencies is implementing cholera prevention activities.

Uganda. Cholera activity has increased in Uganda since the beginning of the current epidemic in late 1997. A total of 16,982 cases with 849 deaths (case fatality rate 5%) was notified to the WHO up to 24 March 1998. Most cases have been in the Central and Eastern Regions. The situation in Eastern Region remains particularly serious with an average of 65 new cases per day. Cholera has also spread to Western Region where 7 districts have notified 2,755 cases with 218 deaths to date and new cases are still occurring.

Latin America. Many countries in the Americas are experiencing unexpected outbreaks of cholera associated with extreme weather conditions brought by the arrival of the El Niño phenomenon. During 1998, the following countries have already reported cholera outbreaks: Bolivia, 165 cases and 5 deaths; Honduras, 219 cases and 12 deaths; Ecuador, 11 cases and 1 death; Peru, 2,863 cases and 16 deaths; and Nicaragua, 3 suspected cases. It is expected that other countries in the region will report increased cholera incidence in the coming months. Preventive and control measures are being taken by the Ministries of Health of the affected countries. The WHO and the Pan Amercian Health Organization are working closely with countries in the region to reactivate cholera preparedness and response plans.

Influenza A(H_5N_1) in Hong Kong

The results of a case-control study on avian influenza conducted in Hong Kong have shown that visiting a poultry stall in the week before becoming ill was the strongest risk factor for infection. The study was aimed at comparing different exposure risk factors between patients and controls. It covered a number of areas including exposure to live poultry, food preparation and food eaten the week before onset, and contact with human illness the week before onset. The results support earlier findings that human to human transmission of the disease is inefficient. The case-control study was jointly carried out by the Department of Health, Hong Kong and the Centers for Disease Control and Prevention in Atlanta (CDC). In all, 18 cases of influenza A (H_5N_1) were confirmed in Hong Kong. The date of onset of illness of the last case was 28 December 1997. A 24 year old female patient is still under treatment and in a stable condition while 11 others have been discharged after recovery. Six people died of the disease.

Dengue

Fiji. Dengue fever continues to circulate in Fiji, with more than 1,300 suspected cases reported in the two weeks to 30 March. Nearly 200 people were hospitalised between 10 and 30 March, mostly in the Northern Division, and in the outer islands of Kadavu and Lakeba. No deaths were reported in March. The death toll remains at 11. Cases continue to be reported from most areas of the country. Public warnings continue and include the need to protect against mosquito bites, to destroy places where mosquitoes may breed (including standing water around human dwellings), and to seek medical treatment for fever lasting longer than three days. The public has also been advised that there have been two deaths due to leptospirosis, which can be confused with dengue fever, but unlike dengue, requires treatment with antibiotics.

Tonga. The number of clinical cases of dengue reported from the main island of Tongatapu appear to be declining. Since February 1998, 20 patients have tested positive dengue IgM. There have been no deaths. Virus typing is in progress.

Editor: Bronwen Harvey

Deputy Editor: Corrine Rann

Assistant Editor: Margaret Curran

Contributions covering any aspects of communicable diseases are invited. Instructions to authors can be found in *CDI* 1998;22:9.

CDI is produced every four weeks by the National Centre for Disease Control, Department of Health and Family Services, GPO Box 9848 Canberra ACT 2601; fax: (02) 6289 7791, phone: (02) 6289 6895.

For subscriptions or change of address please fax (02) 6269 1212 or write to PO Box 650, Fyshwick ACT 2609.

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