COMMUNICABLE DISEASES INTELLIGENCE

ISSN 0725 - 3141 VOLUME 20 NUMBER 10 13 May 1996

CONTENTS

ARTICLES Page

An outbreak of Escherichina coli O157 infection on the Gold Coast 236
Brad McCall et al

Should STD surveillance include ethnicity data? 240
Mredith Temple-Smith and Sandra Gifford

OVERSEAS BRIEFS 243

COMMUNICABLE DISEASES SURVEILLANCE 244

Editor : Helen Longbottom
Deputy Editor : Graham Andrews

COMMONWEALTH
DEPARTMENT OF
HEALTH AND FAMILY SERVICES

Contributions covering any aspect of communicable diseases are invited. Instructions to authors can be found in CDI 1995; 20: 13.

CDI is produced fortnightly by the AIDS/Communicable Diseases Branch, Department of Health and Family Services, GPO Box 9848 Canberra ACT 2601, Fax: (06) 289 7791 Telephone : (06) 289 1555

Opinions expressed in CDI are those of the authors and not necessarily those of the Department of Human Services and Health or other Communicable Diseases Network - Australia affiliates. Figures given may be subject to revision.

CDI is available on the CDI Bulletin Board System on (06) 281 6695, and via Internet on 'ftp://ftp.health.gov.au' in directory /pub/CDI and on 'http://www.health.gov.au' in '/hsh/pubs/cdi/cdihtml.htm.'

AN OUTBREAK OF *ESCHERICHIA COLI* O157 INFECTION ON THE GOLD COAST

Brad McCall[1], *David Strain*[2], *Susan Hills*[3], *Malcolm Heymer*[2], *John Bates*[4], *Denise Murphy*[4], *Robyn Kelly*[5] *and Darrell Price*[6]

Abstract

An outbreak of bloody diarrhoea associated with *Escherichia coli* O157 infection in young children on the Gold Coast in Queensland was investigated. This outbreak was the first involving the O157 serotype in Australia. Fifty-seven people were screened and *E. coli* O157 was isolated from six people, all of whom had consumed different food items from a delicatessen. No single food item was identified as the source of the infection. One of the food handlers who was positive for *E. coli* O157 had minor symptoms of gastroenteritis preceding the onset of disease in the identified cases. This person had prior contact with an animal that showed clinical signs of infection, suggesting a possible method of entry for the organism into the delicatessen. However, it is also possible that a contaminated food product entered the delicatessen and contaminated other food products during handling. Therefore, cross contamination within the delicatessen was a likely associated factor in the transmission of this disease.

Introduction

Escherichia coli O157:H7 is one of a group of enterohaemorrhagic *E. Coli* (EHEC) capable of causing severe disease in humans. Since 1982, this pathogen has been increasingly recognised overseas as a major cause of morbidity and mortality, with an estimated 21,000 infections and as many as 250 deaths annually in the United States of America alone[1]. Overseas, the number of outbreaks associated with *E. coli* O157:H7 have been increasing in recent years, with the largest outbreak involving approximately 500 cases in the United States of America in 1993[2,3,4].

Infection with enterohaemorrhagic *E.coli* may result in a broad spectrum of clinical manifestations. These range from asymptomatic infection, diarrhoea or haemorrhagic colitis to haemolytic uraemic syndrome (HUS) in children or thrombotic thrombocytopenic purpura (TTP) in adults. The pathogenesis of these conditions in cases of EHEC infection is mediated by the bacteria producing verocytotoxin or shiga-like toxin (SLT)[5]. HUS is characterised by haemolytic anaemia, thrombocytopenia and acute renal failure (oliguria or anuria with elevated serum urea and creatinine). This infection is more frequently encountered in children under five years of age. Usually, HUS occurs in two to seven per cent of cases although in outbreaks, up to 30% of cases develop this complication[6, 7]. Mortality from HUS ranges from 3% to 17%, while long-term renal impairment may also occur[8].

Sporadic cases of EHEC infection have been recognised in Australia since 1987, although the proportion of *E. coli* O157:H7 isolates has been low[9]. A 1991 survey of children with diarrhoea at a Sydney hospital found that *E. coli* O157:H7 was an uncommon cause of acute gastroenteritis in that particular Australian context[10]. The isolates of EHEC from Australian cases of haemorrhagic colitis or HUS have been other serotypes such as O111:NM, which was implicated in the South Australian outbreak of HUS in 1995, and O157:NM[9, 11].

Most outbreaks of EHEC disease have been associated with the consumption of beef, primarily under-cooked ground beef. Other implicated means of infection include inadequately washed vegetables[12], yoghurt[7], unpasteurised milk[13] and apple cider[14]. Modes of transmission other than foodborne spread have also been identified, including person-to-person transmission[15], contact with infected farm animals[16], and from swimming in a faecally contaminated lake[17]. Cattle are considered to be the main reservoir of the infection, with EHEC organisms occurring as part of the normal intestinal flora of a small percentage of cattle[6]. Meat for human consumption may be contaminated during the slaughtering process. The infectious dose of EHEC organisms for humans appears to be extremely low[18].

This report describes an outbreak of *E.coli* O157 disease which occurred on the Gold Coast in south-east Queensland during March 1996. This is the first reported outbreak of *E.coli* O157-associated disease in Australia.

On 12 March 1996, the first two of three notifications of *E. coli* O157 infection were received by the Southern Zone Public Health Unit, Brisbane.

1. Southern Zone Public Health Unit, Queensland Health, PO Box 8509, Upper Mt Gravatt, Qld, 4122.
2. Environmental Health Unit, South Coast Regional Health Authority, Qld.
3. Communicable Diseases Branch, Queensland Health, Qld.
4. Public Health Microbiology, Queensland Health Scientific Services, Qld.
5. Pathology Department, Gold Coast Hospital, Qld.
6. Gold Coast Hospital, Qld.

Methods

Upon notification of the first two cases, parents of the affected children were interviewed. A standard foodborne illness questionnaire was used. Enquiries determined that a number of other close family members and school contacts had reported diarrhoea in the preceding or similar period. All close family contacts (symptomatic and asymptomatic) of the cases and school contacts who had diarrhoea provided faecal specimens and completed a modified foodborne illness questionnaire which specifically focussed on risk factors for EHEC infection. Initial responses from the parents of the two cases implicated a common food outlet (a delicatessen), but not a common food item. The delicatessen was inspected, food samples and environmental swabs were taken and two separate faecal specimens were requested from all staff (food handlers) working in the premises.

Upon notification of a third case, and the detection of *E. coli* O157 isolates in a family contact and two food handlers, a case control study was commenced. The case definition used was 'isolation of *E. coli* O157 from a faecal specimen, with or without the presence of an acute enteric illness characterised by fever, abdominal pain, diarrhoea or bloody diarrhoea'. Controls included *E. coli* O157 culture-negative family members, school contacts, food handlers and other children seen at the Gold Coast Hospital with bloody diarrhoea of different aetiology.

Faecal and serological specimens

Initial *E. coli* O157 isolates were detected by the Gold Coast Hospital Pathology Laboratory. This was made possible by the laboratory's protocol of routinely setting up sorbitol MacConkey agar plates when testing faecal specimens of patients with bloody diarrhoea. Non-sorbitol fermenting colonies on these plates were forwarded to Public Health Microbiology Laboratory (PHML) Queensland Health Scientific Services, where they were confirmed using a commercial antisera for *E. coli* O157. All faecal specimens collected in this investigation were examined at the PHML.

Isolated colonies of *E. coli* O157, and the faecal specimens from which they were derived, were tested for the presence of shiga-like toxin (SLT) using an ELISA (enzyme-linked immunosorbent assay) method. Separate tests were performed to determine the flagella antigen. Pulsed field gel electrophoresis using restriction enzymes was also carried out by PHML to examine the clonal characteristics of these isolates. Phage typing was performed at the Microbiological Diagnostic Unit (MDU) using a specific set of *E. coli* O157 phages.

Serological testing for *E. coli* O157 is a developmental, non-standardised ancillary test. Recognising these limitations, the investigators obtained blood samples for serology from *E. coli* O157 culture-negative family contacts with previous symptoms of diarrhoea and one symptomatic child who was culture-negative for *E. coli* O157.

Serological and faecal specimens were also obtained from an animal that showed clinical signs of infection, from one of the positive food handlers, and other animals associated with the person.

Food and environmental specimens

Quantities of food samples were obtained from patients' homes and the delicatessen suspected of being responsible for the infection. They were cultured specifically for the presence of *E. coli* O157 using methods supplied by the Institute of Medical and Veterinary Science (IMVS) in Adelaide. This involved a qualitative method, sampling 25 grams of food and plating directly onto sorbitol MacConkey agar plates after 24 hours enrichment in buffered peptone water. The quantitative method involved a standard five-tube Most Probable Number (MPN) method with all cultures being incubated at 37^0C, followed by plating onto Eosin Methylene Blue (EMB) agar and sorbitol MacConkey agar. Those suspect colonies which were confirmed as *E. coli* were tested for agglutination with specific *E. coli* O157 antisera. Environmental swabs taken from the delicatessen were examined qualitatively in a similar manner.

Results

In all, 57 individuals provided 84 faecal specimens for this investigation. This included 18 specimens from the six infected people, 17 family contacts, two school contacts, 24 delicatessen staff and eight others (two patients with symptoms and six of their family contacts). Thirty of these people completed a questionnaire and were eligible as controls

Six people were identified during this outbreak as having evidence of infection with *E.coli* O157. Three people were identified through screening procedures.

Case 1

Case 1 was a ten year old male who developed a sore throat and slight fever on 1 March 1996. This progressed to an illness with fever, abdominal cramps and associated bloody diarrhoea. He presented to the Gold Coast Hospital on 3 March 1996. Pathology tests showed a normal full blood count and biochemistry. Subsequent faecal samples showed *E. coli* O157. The illness lasted for six days and the patient made a full recovery without hospital treatment.

Case 2

Case 2 was a five year old male who developed abdominal cramps and malaise on 5 March 1996 with subsequent bloody diarrhoea. This patient also presented to the Gold Coast Hospital where his blood count and biochemistry were normal. A faecal specimen grew *E. coli* O157. This child had a brief illness and made a complete recovery.

Case 3

Case 3 was a 22 month old female who developed fever, nausea and vomiting on 7 March 1996, followed by watery diarrhoea ten days later. Examination of a stool

specimen obtained on 19 March 1996 detected a growth of *E.coli* O157. Other pathology results were normal. Despite the duration of her illness (17 days), this child also made a complete recovery.

Case 4

The fourth person was the asymptomatic 58 year old grandmother of case 1.

The last to be identified were two food handlers working in a delicatessen.

Case 5

Food handler one, a 20 year old female, reported mild abdominal cramps on 26 February 1996, suggesting that she might have been the index case of this outbreak. This person had contact with a pet dog that had had bloody diarrhoea in the week before the onset of her own symptoms. Food handler one and the pet had frequently visited a rural property prior to the onset of symptoms. However, there were no cattle resident on that property or in the vicinity. The faecal specimens from all sampled animals, including the pet dog of the food handler, were negative for *E. coli* O157.

Case 6

Food handler two, a 17 year old female, reported no gastroenteric symptoms.

The case-control study did not reveal any statistically significant associations between disease and particular food items. A salient finding was that all six infected people had consumed different food items from the same delicatessen. Inspection of the food handling procedures in the delicatessen revealed a generally satisfactory standard of food hygiene in the workplace. Protocols, including periodic training, were in place to emphasise the importance of food hygiene to employees. However, the attention of management was directed to several areas of importance, including the adequate maintenance of hand washing facilities and the appropriate storage of products in display cabinets. Upon the identification of the infected food handlers and their potential as asymptomatic carriers to contaminate food items, the management cooperated in the disposal of any items which the food handlers may have contaminated and in the thorough sanitisation of the workplace. The infected staff members were excluded from food handling duties until they had demonstrated microbiological clearance of the organism (defined as two negative faecal specimens taken at intervals of not less than 48 hours)[19]. All manufacturers of smallgoods retailed at the outlet were subjected to review of quality assurance procedures.

Faecal and serological specimens

All isolates of *E. coli* O157, and some of the faecal specimens from which they were derived, tested positive for SLT. Pulsed field gel electrophoresis of all six isolates demonstrated greater than 95% homology, suggesting that the isolates were clonally identical. All isolates were phage typed as phage type 14.

Tests for the flagella antigen have demonstrated motility, but have not yet identified the H7 antigen. However, these tests are being repeated at the Fairfield Hospital, Melbourne. The final results of serological testing are currently unavailable.

Food and environmental specimens

Although a large number of food samples were provided from the cases' residences and the delicatessen thought responsible for the spread of the infection, *E. coli* O157 was not detected. Similarly, environmental swabs of surfaces, cutting machinery, utensils and other areas in the delicatessen did not detect *E. coli* O157.

Discussion

No particular food item could be identified bacteriologically or epidemiologically as the source of this outbreak of *E. coli* O157 infection. However, a common epidemiological link was established with the consumption of different food items from a particular delicatessen. It is likely that cross contamination of multiple food items within the delicatessen was a factor in the spread of this infection. The dates of onset of illness suggest that transmission of this pathogen commenced during late February. The onset of symptoms in food handler one, prior to onset in the documented cases, suggests that she (and hence the animal (dog) with the bloody diarrhoea) may have been the source of the infection. This investigation cannot exclude the possibility that a contaminated food product entered the delicatessen and contaminated other food products during its handling. The possibility exists that lapses in personal hygiene amongst the staff may also have contributed to the transmission of this organism.

An important factor in the identification of this outbreak was the use of sorbitol MacConkey agar plates by the Gold Coast Hospital Pathology Laboratory in the routine investigation of patients with bloody diarrhoea. The Australian experience with EHEC has suggested that *E. coli* O157 was not a prevalent pathogen. However, this experience suggests that the use of sorbitol MacConkey agar plates in the investigation of bloody diarrhoea should be reconsidered as an aid to detecting further outbreaks. It is important to remember that not all EHEC serotypes fail to ferment sorbitol. Laboratories also need to be aware of the necessity to serotype and test for SLT production in any heavy growth of *E. Coli* associated with bloody diarrhoea in the absence of other pathogens[9].

Another important feature of this investigation was the role of enhanced surveillance. All pathology laboratories and medical practitioners on the Gold Coast were notified about the occurrence of the first two cases of *E. coli* O157 infection. Surveillance was also carried out in the schools that the first two cases attended. This enhanced surveillance facilitated the detection of the third case and assisted in further defining the nature of the outbreak.

The occurrence of this outbreak emphasises the importance of correct food handling procedures and strict hygiene and sanitation precautions in the food industry, at both the manufacturing and retail levels. Inspection, and reinforcement of education about food hygiene should be a standard practice in food outlets, particularly for those dealing with smallgoods.

This outbreak is the first documented outbreak of *E. coli* 0157 infection and only the second recognised outbreak of EHEC infection in Australia. It may be that the apparent increased number of outbreaks overseas is about to be experienced in Australia. The lessons from this outbreak include the value of routine microbiological surveillance for EHEC organisms (including the use of sorbitol MacConkey agar plates), the role of extensive screening and enhanced surveillance in detecting further cases and the importance of obtaining industry cooperation in the prevention and management of outbreaks of this disease.

Acknowledgments

The authors would like to gratefully acknowledge the assistance of the following colleagues: Dr J Sheridan, Dr AS Cameron, Ms C Walker, Dr C Dalton, Dr M Bell, Dr L Selvey, Dr K Bettelheim (Fairfield Hospital), Ms D Lightfoot (MDU), Dr D Spain, Dr P Elliott, Dr M Higgins, and the Microbiology Department, Queensland Medical Laboratories (Southport).

References

1. Boyce TG, Swerdlow DL, Griffin MD. *Escherichia coli* O157:H7 and the Hemolytic-Uremic Syndrome. *N Engl J Med* 1995; 333:364-368.
2. Bell BP, Goldoft M, Griffin PM, *et al.* A multistate outbreak of *Escherichia coli* O157:H7 - Associated bloody diarrhoea and hemolytic uremic syndrome from hamburgers, *JAMA* 1994; 272: 1349-1353.
3. Jay JM. Foods with low numbers of microorganisms may not be the safest foods OR Why did human listeriosis and hemorrhagic colitis become foodborne diseases? *Dairy, Food Environ Sanitation* 1995; 15:674-677.
4. Neill MA. Pathogenesis of *Escherichia coli O157:H7 infection. Current Opinion Infect Dis* 1994; 7:295-303.
5. Moake L. Haemolytic-uraemic syndrome: basic science. *Lancet* 1994; 343:393-397.
6. Griffin PM, Tauxe RV. The epidemiology of infections caused by *Escherichia coli* O157:H7, other enterohemorrhagic *E. coli,* and the associated hemolytic uremic syndrome. *Epidemiol Rev* 1991; 13:60-98.
7. Morgan D, Newman CP, Hutchinson DN, *et al.* Verotoxin producing *Escherichia coli* O157 infections associated with the consumption of yoghurt. *Epidemiol Infect* 1993; 111:181-187.
8. Subcommittee of the PHLS Working Group on Vero cytotoxin producing *Escherichia coli* (VTEC). Interim guidelines for the control of infections with Vero cytotoxin producing *Escherichia coli* (VTEC). *CDR Rev* 1995; 5:R77-R81.
9. Goldwater PN, Bettelheim KA. The role of enterohaemorrhagic *Escherichia coli* serotypes other than O157:H7 as causes of disease in Australia. *Comm Dis Intell* 1995; 19:2-4.
10. Ong J, Zhe L, Robins-Browne R, *et al.* Prevalence of verocytoxigenic *Escherichia coli* serotype O157:H7 in children with diarrhoea attending a Sydney hospital, *J Paediatr Child Health* 1993;29:185-187.
11. Cameron AS, Beers MY, Walker CC, *et al.* Community outbreak of hemolytic uremic syndrome attributable to *Escherichia coli* O111:NM - South Australia, 1995, *MMWR Morb Mort Wkly Rep* 1995;44:550-558.
12. Cieslak PR, Barrett TJ, Griffen PM, *et al. Escherichia coli* O157:H7 infection from a manured garden [letter]. *Lancet* 1993; 342:367.
13. Chapman PA, Wright DJ, Higgins R. Untreated milk as a source of verotoxigenic *Escherichia coli* O157. *Vet Rec* 1993; 133:171-172.
14. Besser RE, Lett SM, Weber JT, *et al.* An outbreak of diarrhoea and hemolytic uremic syndrome from *Escherichia coli* O157:H7 in fresh-pressed apple cider. *JAMA* 1993; 269:2217-2220.
15. Spika JS, Parsons JE, Nordenberg D, *et al*. Hemolytic uremic syndrome and diarrhoea associated with *Escherichia coli* O157:H7 in a day care centre. *J. Pediatr* 1986; 109:287-291.
16. Sharp JCM, Ritchie LD, Curnow J, Reid TMS. High incidence of haemorrhagic colitis due to *Escherichia coli* O157 in one Scottish town: clinical and epidemiological features. *J Infect Dis* 1994; 29:343-350.
17. Keene WE, McAnulty JM, Hoesly FC, *et al*. A swimming-associated outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7 and *Shigella sonnei. N Engl J Med* 1994; 331: 579-584.
18. Benenson A (ed.). *Control of Communicable Diseases Manua*l. American Public Health Association. Washington, 1995: 140-144.
19. PHLS Salmonella Committee. The prevention of human transmission of gastrointestinal infections, infestations, and bacterial intoxications. *CDR Rev* 1995; 11:R164.

SHOULD SEXUALLY TRANSMISSIBLE DISEASE SURVEILLANCE INCLUDE ETHNICITY DATA?

Meredith Temple-Smith and Sandra Gifford, Centre for the Study of Sexually Transmissible Diseases, La Trobe University, Locked Bag 12, Carlton South, Victoria 3053

Abstract

Australia is recognised as having one of the world's most ethnically diverse populations, yet indicators of ethnicity are not consistently collected with data relating to HIV and sexually transmissible diseases. Such information is essential if we are to determine if people of non-English speaking backgrounds and other Australians are at equal risk of STDs. This has implications for effective planning in prevention and treatment. Current ethnicity data for STDs in Australia are examined. Suggestions are made for a staged framework for collecting such information and for uses of these data.

Introduction

Australia's diverse ethnic population is well recognised[1]. The importance of tailoring treatment and preventive services to meet the specific needs of our ethnic communities has been well documented[2,3]. This requires the collection of ethno-specific data, yet few epidemiological studies or national databases in Australia collect indicators of ethnicity[4]. While there are many difficulties in gathering accurate information on ethnicity and health status, collecting these data for sexually transmissible diseases (STDs) including HIV and AIDS is even more difficult, given their often stigmatised nature. However, systematic collection of ethnicity data for STDs is essential if we are to determine whether STD risk for people of non-English speaking backgrounds is higher than, lower than, or is equal to that of the English-speaking Australian-born population. Many migrants arrive as refugees from countries with a level of STDs higher than Australia, and this makes it all the more important to collect uniform and accurate information about ethnicity if we are to assess the need for treatment and prevention among these groups.

There has been much debate around the operational definition of ethnicity and the best way to measure it on a population basis[3]. In the United States of America, much of the STD and HIV/AIDS data is reported according to race, such as 'Black or African American' and 'Hispanic'. Race is an indicator of biological factors, and is important when, for example, genetic characteristics are responsible for a population's vulnerability to infection. It is important to distinguish biological factors from socio-cultural factors which may expose groups to infection in a different manner[5,6]. Socio-cultural factors are more important than biological ones in the case of STDs, as sexual beliefs and practices play a key part in the risk of exposure. It is essential that any indicator of ethnicity capture information on social and cultural factors.

Reporting of ethnicity for STDs in Australia

Although the establishment of the National Notifiable Diseases Surveillance System (NNDSS) in 1990 represents a significant advance in public health in Australia, surveillance for STDs is poorer than for many other infectious diseases. Although uniform diagnostic criteria for individual STDs have been defined, the way in which these are used may vary at the State and Territory level[7]. This, along with the variation between requirements in States and Territories about which STDs are notifiable, affects the overall accuracy of national surveillance data[8]. Although there are guidelines recommending a core set of variables for the collection of ethnicity data (Aborigines and Torres Strait Islanders, birthplace, language spoken, English proficiency)[9], there is wide variation between the States and Territories in the way ethnicity is reported on STD notifications (Table). The most common information on ethnicity collected for STD notifications is country of birth, but some States and Territories do not collect any data on ethnicity.

Although indicators of Aboriginality are often included in ethnicity data, we have purposely not included issues of Aboriginality in this paper for two reasons. First, the difficulties in gathering and interpreting data on Aboriginality are complex and warrant discussion in their own right. Secondly, we acknowledge the principles of health research set out by Aboriginal groups and adopted by the National Health and Medical Research Council, which stress the need for a consultative and cooperative relationship between researchers and Aboriginal communities that are the subjects of the research. Particularly because of the sensitive nature of STDs, we believe that the question of STD surveillance among Aboriginal communities should be addressed with the full involvement of Aboriginal people themselves.

Reporting of ethnicity for HIV in Australia

In contrast to other STDs, the notification system for HIV is good. Surveillance for HIV is maintained by the National Centre in HIV Epidemiology and Clinical Research (NCHECR) in collaboration with State and Territory health authorities and the Commonwealth of Australia. The National HIV Database is notified on the first occasion of an HIV diagnosis within Australia by either the diagnosing laboratory (ACT, NSW, Tasmania and Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia and Western Australia). States and Territories differ in the type of data collected on ethnicity and no information on ethnicity is provided to NCHECR (Table).

Table. Measures of ethnicity collected in HIV/AIDS and STD surveillance data in Australia[1]

State / Territory	HIV	AIDS	STDs	Comments
Australian Capital Territory	Not collected	COB	COB	all notifiable STDs (mid-1993)
New South Wales	LSH	COB, LSH	COB	Syphilis only
Northern Territory	A / nA	*	A / nA	all notifiable STDs
Queensland	COB, A / nA	COB	COB, EO	optional on all notifiable STDs
South Australia	R	COB	R	all notifiable STDs
Tasmania	Not collected	*	Not collected	all notifiable STDs
Victoria	Not collected	COB	COB	Hepatitis
Western Australia	A / nA	COB	COB, A / nA	all notifiable STDs

1. Source: personal communication with State or Territory health authorities.

KEY:

COB	**Country of birth**
LSH	**Language spoken at home**
EO	**Ethnic origin**
R	**Race: Aborigine, Asian, Caucasian, other**
A/nA	**Aborigine/non-Aborigine**
*****	**Not collected on State form, but this information is sought for notification to NCHECR**

Reporting of ethnicity for AIDS in Australia

AIDS has been a notifiable condition in all States and Territories since 1984, although the mechanism of reporting differs. In 1989 NCHECR developed a new AIDS notification form. Some States and Territories now use this, while others have retained their old notification forms, resulting in some variation between data collected at a State and at national level. All States and Territories notify their AIDS cases to the National AIDS Registry and make efforts to provide the data required by the registry, which includes two questions relating to ethnicity - 'Country of birth - Australia or Other (specify)', and 'If Other- state year of arrival in Australia'.

Even though collected, this information was not included in the database at its commencement but more recently has been included for current, and updated for retrospective, notifications. Some ethnicity data remain missing despite having been actively sought. This has resulted in the absence of information on country of birth for 22% of the total of 3,160 AIDS cases diagnosed in Australia between 1981 and 1992. The remaining data show, however, that until 1992 the proportion of people of non-English speaking backgrounds with AIDS was lower than for the proportion of people of non-English speaking backgrounds in the Australian population[10]. Surveillance figures for the population of Victoria, with less than one per cent of missing data, show the proportion of people of non-English speaking backgrounds with AIDS to be the same as the proportion of people of non-English speaking backgrounds in the Victorian population[11].

What kind of information on ethnicity is worth collecting?

Ethnicity has commonly been measured through a variety of variables including country of birth, first language, languages spoken at home, proficiency in languages including English, and length of residence in Australia[1,12]. Which of these measures would be most useful to collect for STDs?

First, data on country of birth should be collected because it provides opportunities to compare rates of STDs with rates of other diseases and with rates in home countries[9]. However, within any one country there may be many different ethnic groups and a diversity of religious, cultural and social patterns. Country of birth does not capture these other indicators of ethnicity. For example, country of birth for people who migrated from Vietnam does not distinguish between Chinese Vietnamese or ethnic Vietnamese, nor those who migrated in the 1970s and those who arrived more recently. All these factors may play an important role in patterns of health and disease including STDs, thus country of birth alone as a measure is insufficient.

A second indicator of ethnicity is the language spoken at home. This identifies the diversity of ethnic populations migrating from a country or a series of countries and is important in determining the need for interpreter and translation services. It is also important for targeting prevention campaigns to specific subgroups within non-English speaking background populations.

Thirdly, length of residence in Australia can be an indicator of the migration patterns which may impact on prevention or risk. For example, patterns of STDs may differ with recent migrants who have arrived in Australia as refugees, compared with those from the same country who migrated for socio-economic reasons.

Finally, religion can provide additional information about intra-ethnic diversity and about specific beliefs and practices which may impact on risk and prevention.

How should ethnicity data be collected?

Given the difficulties of gathering a comprehensive set of data on ethnicity, we suggest a three-tier approach. First, we recommend that data on country of birth and language spoken at home be routinely collected as part of all HIV and other STD notifications. Although the current levels of compliance of notification for diseases other than AIDS is a problem, we believe that these two variables should be included while efforts are being made to improve surveillance, especially as this information is already being collected in some States and Territories[13,14]. This is in line with current recommendations on the collection of ethnicity for health data Australia wide[12].

At the second level, we propose length of residence in Australia be added to the data about country of birth and language spoken at home in any periodic epidemiological or sentinel survey of STDs. These studies generally require greater efforts than routine surveillance, so the addition of this variable should not be too burdensome. At the third level, we suggest that data on religion be collected for public health research studies on STDs.

This three-tier system for collection of ethnicity data from the most basic to the more specific in both surveillance and research projects will greatly improve our ability to describe prevalence and risk for STDs within Australia's multicultural population. It will also enable us to direct education programs to specific risk groups for specific STDs.

A critical process in changing any standard data collection is consultation with the providers of the information about easy and reliable methods of eliciting, recording and reporting these data. Providers of data frequently do not perceive the benefits of their labours, so feedback to them is important[15]. One of the difficulties of surveillance generally is the lack of denominator data for calculation of rates. In the area of ethnic health, this is compounded by the fact that the number of members of any single ethnic group may be quite small. For the purposes of STDs and ethnicity, data collected by the Australian Bureau of Statistics on indicators of ethnicity are available for both the non-English speaking background populations as a whole and for specific groups. These data are adequate to provide denominator information on the socio-demographic characteristics of specific ethnic groups.

How should ethnicity data be used?

Data need to be easily accessible to those providing services, conducting research and developing policy to improve STD prevention and treatment for people of non-English speaking backgrounds. Despite the poor history of STD surveillance, collation of data nationally has provided some useful recommendations, for example about screening for gonorrhoea[16].

Access to ethnicity data would be improved through a national register of ethnicity-specific information on STDs. Some States already collect country of birth on their notification forms, yet this information is not readily available to researchers because it is either not added to the database or not collated. Many researchers are unaware of its existence.

Despite the benefits of collecting these data, consideration must be given to the potential unanticipated harmful consequences of more thorough collection of ethnicity data. While more accurate data can assist in targeting prevention programs to the specific needs of non-English speaking background communities, this can also serve to stigmatise already marginalised groups. Migrants are vulnerable to being singled out for importing diseases, especially infectious diseases, into Australia. It is also possible that sensitive and detailed epidemiological data could be misused, for example by carrying out selective screening of specific communities, or by supporting discriminatory treatment in relation to access to employment or health care[17,18,19].

Conclusion

At present, few data are available on STD rates in people of non-English speaking backgrounds compared with the rest of the Australian community. This has lead to difficulties in developing effective strategic planning for prevention and treatment. If non-English speaking background communities suffer less from the burden of STDs than the rest of the Australian community, then we have nothing to worry about. However, if they suffer more, then they may be further disadvantaged by the stigma of these diseases, which can also hinder access to prevention and treatment. We suggest a staged framework for improving what is known about the burden of STDs in Australia's multicultural population.

Acknowledgments

We wish to acknowledge the assistance and advice given to us by Dr Sandy Thompson of the Macfarlane Burnet Centre for Medical Research.

References

1. Young C. Australia's heterogeneous immigrant population: a review of demographic and socioeconomic characteristics. In: Donovan J, d'Espaignet E, Merton C, van Ommeron M, eds. Immigrants in Australia: a health profile. Canberra: Australian Government Publishing Service, 1992.

2. Health Department Victoria. Working with People from Non-English Speaking Backgrounds: Guidelines for Health Agencies. Melbourne: Health Department Victoria, 1992.

3. Donovan J, d'Espaignet E, Merton C, van Ommeren M. Rationale of the contents. In: Donovan J, d'Espaignet E, Merton C, van Ommeren M, eds. Immigrants in Australia: A Health Profile. Canberra: Australian Government Publishing Service, 1992.

4. Schofield T. Health of Australians of non-English speaking background. *Aust J Public Health* 1995; 19:117-118.

5. Hahn R. The state of federal health statistics on racial and ethnic groups. *JAMA* 1992; 267:268-271.

6. Wyatt, GE. Examining ethnicity versus race in AIDS related sex research. *Soc Sci Med* 1991; 33:37-45.

7. Longbottom H, Evans D, Myint H, Hargreaves J. Annual Report of the National Notifiable Diseases Surveillance System, 1993. *Comm Dis Intell* 1994; 18:518-549.

8. Moran J, Anderson B. Australian guidelines for the maintenance of sexual health in sex industry workers. *Venereology* 1994; 7:82-85.

9. Powles J, Gifford S. How healthy are Australia's immigrants? In: Reid J, Trompf P, eds. The health of immigrant Australia: a social perspective. Sydney: Harcourt Brace Jovanovich, 1990.

10. Kaldor J, McDonald A, Blumer CE, *et al*. The Acquired Immune Deficiency Syndrome in Australia: incidence 1982-1991. *MJA* 1993; 158:10-17.

11. AIDS / STD Unit. Department of Health and Community Services. Surveillance of Sexually Transmissible Diseases in Victoria 1993. Department of Health and Community Services: Victoria, 1994.

12. Office of Multicultural Affairs. Diversity Counts: A handbook on ethnicity data. Canberra: Australian Government Publishing Service, 1994.

13. Crofts N, Gertig D, Stevenson E *et al*. Surveillance for sexually transmissible diseases in Victoria, 1983 to 1992. *Aust J Public Health* 1995; 18:433-439.

14. Condon R, Roberts M, Rouse I, Sesnan. A comparison of doctor and laboratory notifications of gonorrhoea in Western Australia during 1989. *Venereology* 1992; 5:84-86.

15. Hart G. Information systems for STD control programmes. *Venereology* 1990; 3:32-37.

16. Hart G. STD epidemiology in Australia: syphilis and gonorrhoea. *Venereology* 1992; 5:115-120.

17. Haour-Knipe M, Dubois-Arber F. Minorities, immigrants and HIV / AIDS epidemiology: concerns about the use and quality of data. *Eur J Public Health* 1993; 3:259-263.

18. Gilmore N, Somerville M. Stigmatisation, scapegoating and discrimination in sexually transmitted diseases: overcoming 'them' and 'us'. *Soc Sci Med* 1994; 39:1339-1358.

19. Goldin CS. Stigmatisation and AIDS: critical issues in public health. *Soc Sci Med* 1994; 39:1359-1366.

OVERSEAS BRIEFS

In the last two weeks, the following information has been provided by the World Health Organization (WHO) and the Centers for Disease Control (CDC).

Ebola haemorrhagic fever: Gabon outbreak over

No new cases of Ebola haemorrhagic fever have been reported in Gabon since the death of the last case on 12 March 1996. The outbreak was therefore officially declared over on 23 April 1996, after a lapse of 42 days corresponding to twice the maximum incubation period. The outbreak occurred in the village of Mayibout II, Makokou health district, Ogooue-Ivindo Province. It was linked to the butchering, transport and preparation for consumption of a chimpanzee found dead in the forest on 24 January 1996. The total number of cases was 37 (20 males, 17 females) and the mean age was 27 years (range 7 months to 70 years). Rapid identification of the disease and appropriate control measures quickly brought the outbreak under control.

Meningococcal meningitis, Mali

An increased number of cases of meningococcal meningitis have been reported in Mali since the beginning of February 1996, first in the District of Dioila, followed by the District of Bamako. By the end of March, 2,347 cases had been notified of which 319 (13.6%) died. Over one-third of the cases were from the District of Bamako. *Neisseria meningitidis* group A has been identified in the laboratory. By 13 March the health authorities declared the situation in the District of Bamako as epidemic and initiated vaccination of all age groups between 1 and 25 years on 15 March. The vaccination campaign has been extended to other affected regions. So far, 708,183 persons in the District of Bamako, 74,411 in the region of Mopti and 83,309 in the region of Koulikoro have been vaccinated. Analysis of the epidemiological situation indicates that the outbreaks have spread to other regions, in particular Ségou, Mopti and Sikasso.

Mali is situated within the meningitis belt in Africa and has experienced outbreaks with regular intervals in the past, such as in 1969, 1981 and 1989. However, the epidemic intervals appear to have shortened as indicated by the epidemic in 1994 preceding this current epidemic. Following reports of outbreaks in neighbouring countries, the health authorities implemented a preparedness strategy of strengthened surveillance, public health information campaigns and distribution of 20,000 doses of vaccines and drugs to each of the eight regions in December 1995.

Since 15 March 1996 the national health authorities have allocated 200 million CFA for vaccine and field operations and have ordered 1 million doses of vaccine and other material needed for the campaign. In addition, international agencies such as WHO, UNICEF,

MSF and *Fondation pour l'Enfance* have contributed about 700,000 doses. The health authorities plan to extend vaccination to the population at risk in areas close to outbreaks in neighbouring countries for which an additional 1.6 million doses would be needed.

Hantavirus pulmonary syndrome update, United States of America

Since hantavirus pulmonary syndrome was first recognised in May 1993, CDC has recorded 133 cases on the national registry. Of the 133 total cases, 37 cases have been identified retrospectively, with an onset of illness prior to May 1993. The earliest case to be confirmed serologically occurred in 1959. Twenty-three confirmed cases occurred in 1995. By April 1996, two cases with onset of illness in 1996 had been added. Fifty-nine per cent of the 133 cases were male and 27% were native American. The mean age of cases was 35 years, with a range from 11 to 69 years. Sixty-six cases died, giving a case fatality rate of 50%.

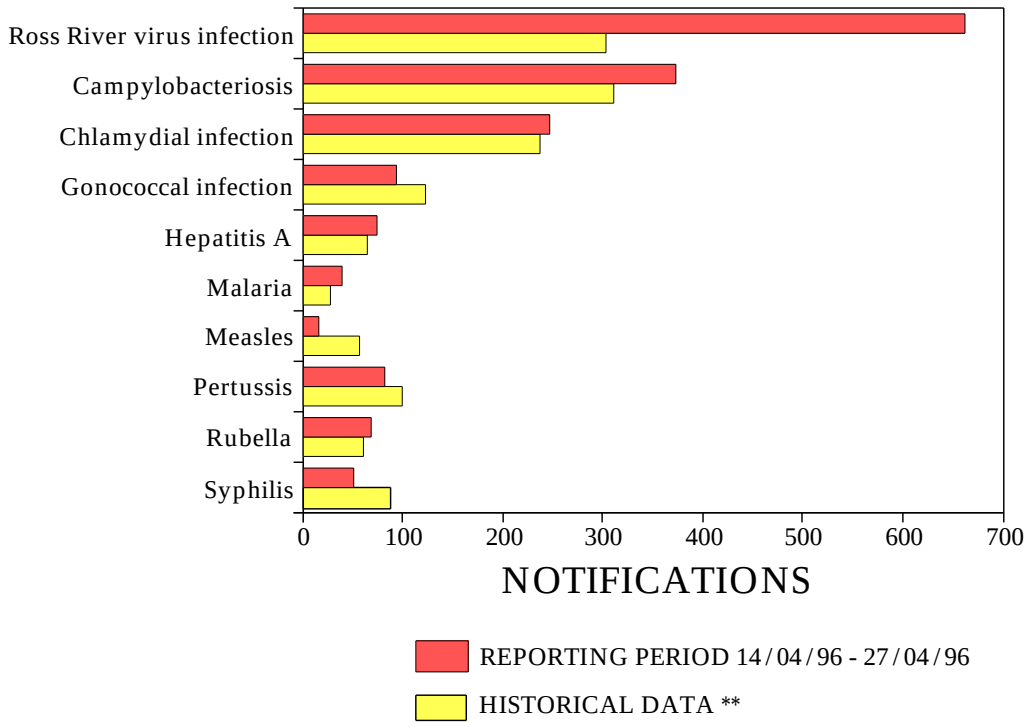
COMMUNICABLE DISEASES SURVEILLANCE

National Notifiable Diseases Surveillance System, 14 to 27 April 1996

There were 3,086 notifications received for this two week period (Tables 1, 2 and 3, and Figure 1).

Ross River virus infection: The epidemic which began in late 1995 and became apparent in the first reporting week of 1996 (Figure 2) has become the most severe ever recorded in Australia, with nearly 6,000 cases being reported between 3 December 1995 and 27 April 1996. The epidemic has been most severe in Queensland (especially the Southern and Central Coastal Statistical Divisions, and Darling Downs). In Western Australia, the greatest numbers and rates have occurred in the South West Statistical Division. In New South Wales, the epidemic has been largely confined to the Northern and Richmond-Tweed Statistical Divisions. Latest available figures suggest that the epidemic is declining in Western Australia and New South Wales, but not yet in Queensland.

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



1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods: the corresponding periods of the last 3 years and the periods immediately preceding and following those.

Hepatitis A has continued to be reported at higher rates than in 1995. Since early December 1995, 1,020 cases have been reported, 91% of them being notified from New South Wales (456 cases), Victoria (295) and Queensland (173). The groups predominantly affected were males between 20 and 44 years of age (Figure 3).

Figure 2. Ross River virus notifications from December 1995 to April 1996, by State

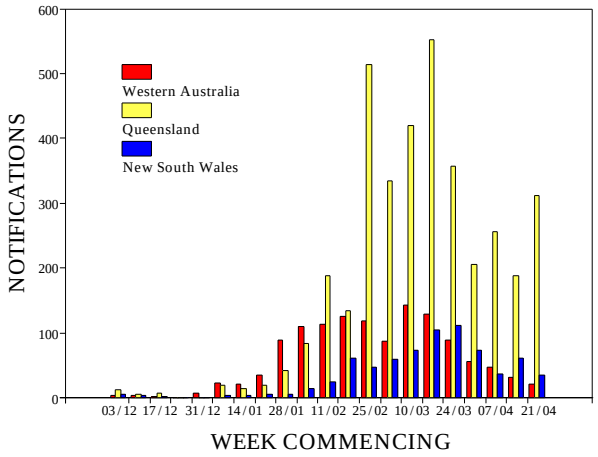


Figure 3. Hepatitis A, cases reported from 3 December 1995 to 27 April 1996, by sex and age group

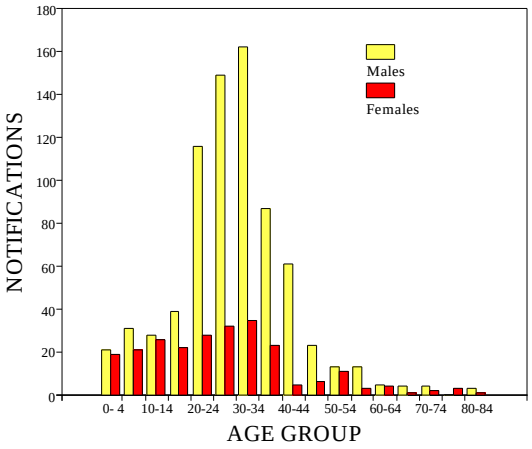


Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 14 to 27 April 1996

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[1] This period 1996	This period 1995	Year to date 1996	Year to date 1995
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae b infection	0	0	0	0	1	0	0	0	1	4	20	33
Measles	0	8	0	3	0	2	4	0	17	32	162	663
Mumps	0	0	1	NN	0	0	3	1	5	4	46	40
Pertussis	0	22	1	18	28	0	8	5	82	70	973	1525
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0
Rubella	10	12	1	29	1	0	23	2	78	54	1045	901
Tetanus	0	0	0	0	0	0	0	0	0	0	1	2

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.

NN Not Notifiable.

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 14 to 27 April 1996

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This period 1996	This period 1995	Year to date 1996	Year to date 1995
Arbovirus infection (NEC)[3,4]	0	20	0	0	0	0	3	0	23	60	192	226
Barmah Forest virus infection	0	0	-	41	1	0	-	-	42	11	312	153
Ross River virus infection	0	95	2	499	3	-	11	52	662	207	5769	1055
Dengue	0	0	0	0	0	-	0	0	0	0	16	7
Campylobacteriosis[5]	9	-	13	110	78	21	97	45	373	279	3821	3441
Chlamydial infection (NEC)[6]	3	NN	30	96	1	11	55	51	247	233	2238	2084
Donovanosis	0	NN	1	0	NN	0	0	0	1	1	18	28
Gonococcal infection[7]	0	18	6	19	0	0	18	34	95	68	1134	981
Hepatitis A	5	23	4	13	0	0	25	4	74	44	852	589
Hepatitis B incident	1	0	1	0	0	3	2	1	8	15	85	124
Hepatitis B unspecified	1	0	0	18	0	1	0	9	29	41	499	592
Hepatitis C incident	0	1	0	0	0	0	0	0	1	3	6	29

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 14 to 27 April 1996, continued

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This period 1996	This period 1995	Year to date 1996	Year to date 1995
Hepatitis C unspecified	11	0	26	60	0	11	144	26	278	224	2995	2651
Hepatitis (NEC)	0	0	0	1	0	0	1	NN	2	2	9	10
Legionellosis	0	3	0	1	0	0	3	0	7	4	62	77
Leptospirosis	0	0	0	12	0	0	2	0	14	3	85	43
Listeriosis	0	0	0	0	0	0	0	0	0	2	19	34
Malaria	0	2	1	31	0	0	5	0	39	16	267	202
Meningococcal infection	1	3	2	0	0	0	3	1	10	12	82	98
Ornithosis	0	NN	0	0	0	0	0	0	0	9	33	57
Q fever	0	7	0	3	0	0	1	1	12	10	150	146
Salmonellosis (NEC)	1	40	13	67	16	10	29	23	199	183	2349	2785
Shigellosis[5]	0	-	1	8	0	0	2	2	13	16	220	309
Syphilis	2	23	8	11	0	1	0	6	51	62	491	616
Tuberculosis	0	10	2	2	0	1	9	3	27	32	369	381
Typhoid[8]	0	0	0	0	0	0	1	0	1	2	34	29
Yersiniosis (NEC)[5]	0	-	0	7	1	0	0	0	8	8	94	142

1. For HIV and AIDS, see Tables 5 and 6. For rarely notified diseases, see Table 3 .
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. Tas: includes Ross River virus and dengue.
4. WA, NT and Vic: includes Barmah Forest virus.
5. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
6. WA: genital only.
7. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
8. NSW, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 14 to 27 April 1996

DISEASES	Total this period	Reporting States or Territories	Year to date 1996
Botulism			
Brucellosis			
Chancroid			
Cholera			
Hydatid infection			
Leprosy			
Lymphogranuloma venereum			
Plague			
Rabies			
Yellow fever			
Other viral haemorrhagic fevers			

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1994.

HIV and AIDS Surveillance

Methodological note

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) 332 4648 Facsimile: (02) 332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for November 1995, as reported to 29 February 1996, are included in this issue of *CDI* (Tables 4 and 5).

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 30 November 1995, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
										This period 1995	This period 1994	Year to date 1995	Year to date 1994
HIV diagnoses	Female	0	3	0	0	0	0	1	0	4	6	68	71
	Male	0	30	0	7	2	0	9	2	50	69	698	793
	Sex not reported	0	1	0	0	0	0	0	0	1	1	9	10
	Total[1]	0	34	0	7	2	0	10	2	55	77	777	875
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	6	26	39
	Male	0	25	0	2	0	0	7	1	35	59	592	797
	Total[1]	0	25	0	2	0	0	7	2	36	65	620	840
AIDS deaths	Female	0	0	0	1	0	0	0	0	1	1	35	34
	Male	0	13	0	13	1	0	18	1	46	54	521	617
	Total[1]	0	13	0	14	1	0	18	1	47	56	557	656

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

Table 5. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 1995, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	15	546	3	94	44	4	164	69	939
	Male	165	9,878	79	1,548	556	70	3,309	745	16,350
	Sex not reported	0	2,048	0	0	0	0	42	0	2,090
	Total[1]	180	12,479	82	1,647	600	74	3,523	816	19,401
AIDS diagnoses	Female	5	130	0	27	18	2	47	17	246
	Male	71	3,687	25	625	260	32	1,299	269	6,268
	Total[1]	76	3,827	25	654	278	34	1,353	288	6,535
AIDS deaths	Female	2	96	0	21	13	2	31	9	174
	Male	49	2,598	19	427	172	21	1,013	198	4,497
	Total[1]	51	2,700	19	450	185	23	1,050	208	4,686

National influenza surveillance 1996

Australian Sentinel Practice Research Network; Communicable Diseases Intelligence Virology and Serology Reporting Scheme Contributing Laboratories, New South Wales Department of Health; Victorian Department of Health; World Health Organization Collaborating Centre for Influenza Reference and Research.

Three types of data will be included in *National Influenza Surveillanc*e, 1996. These include Sentinel General Practitioner Surveillance, Laboratory Surveillance and Absenteeism Surveillance

Sentinel General Practitioner Surveillance

Data will be included from three sources this season, ASPREN (the Australian Sentinel Practice Research Network), the New South Wales Department of Health and the Victorian Department of Health.

The ASPREN consultation rate for influenza-like illness has risen in recent weeks , as has that for the New South Wales scheme (Figure 4). The Victorian Department of Health will be commencing influenza surveillance next fortnight.

Laboratory Surveillance

Laboratory surveillance data from the *Communicable Diseases Intelligence* Virology and Serology Reporting Scheme will be included in *National Influenza Surveillanc*e, 1996. The World Health Organization Collaborating Centre for Influenza Reference and Research will also contribute information on strains isolated.

This fortnight there was one report of influenza A received. A total of 34 reports has been received for the year to date which is as expected for the time of year. A single report of sub-type H_3N_2 and no reports of H_1N_1 have been received so far this year.

Three reports of influenza type B have been received so far for the year to date.

Absenteeism Surveillance

National absenteeism data will be supplied by Australia Post and included in *National Influenza Surveillanc*e, 1996.

The national absenteesim rate has remained at approximately 2.5% since March (Figure 5).

Figure 4. Sentinel general practitioner influenza reports per 1,000 encounters, 1996, by week

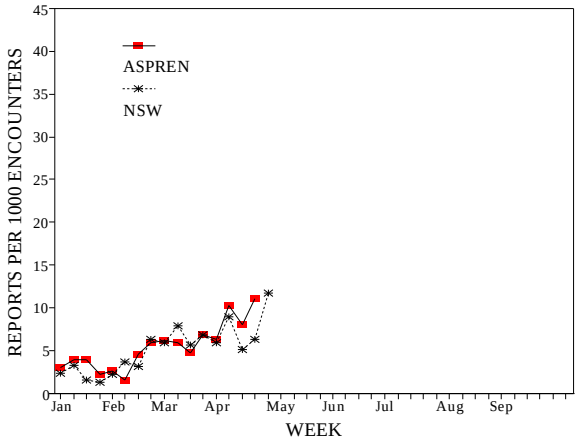
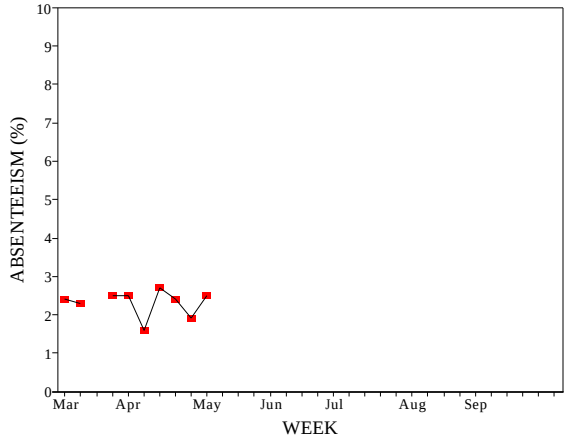


Figure 5. Australia Post absenteeism, 1996, by week



Australian Sentinel Practice Research Network

Data for weeks 15 and 16 ending 14 April and 21 April 1996 respectively are included in this issue of *CDI* (Table 6). The rate of reporting of influenza-like illness rose to 11.2 per 1,000 consultations for week 16, the highest rate recorded by the scheme this year. The rate of reporting of gastroenteritis fell in the second week of the reporting period to the lowest level recorded this year.

Table 6. Australian Sentinel Practice Research Network, weeks 15 and 16, 1996

Condition	Week 15, to 14 April 1996		Week 16, to 21 April 1996	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	64	8.1	81	11.2
Rubella	4	0.5	1	0.1
Measles	0	0	0	0
Chickenpox	23	2.9	14	1.9
Pertussis	1	0.1	1	0.1
Gastroenteritis	106	13.4	84	11.6

Virology and Serology Reporting Scheme

There were 1,182 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 7, 8 and 9).

Ross River virus was reported for 24 patients this fortnight. Diagnosis was by IgM detection (22), single high titre (one) and fourfold change in titre (one). Since the peak in February the number of cases has markedly decreased (figure 6).

Forty two reports of **parainfluenza virus type 1** were received this period. Diagnosis was by antigen detection (3) and virus isolation (39). Seventy four per cent of reports (31) came from Queensland, NSW (7) and Victoria (4). The number of reports received has continued to increase in recent weeks.

Repiratory syncytial virus was reported for 150 patients this period. Diagnosis was by antigen detection (49), virus isolation (100) and nucleic acid detection (one). Ninety eight percent (147) of the patients were between the ages of one month and 4 years. Reports have continued to increase in recent months (figure 7).

Figure 6. Ross River virus laboratory reports, 1995

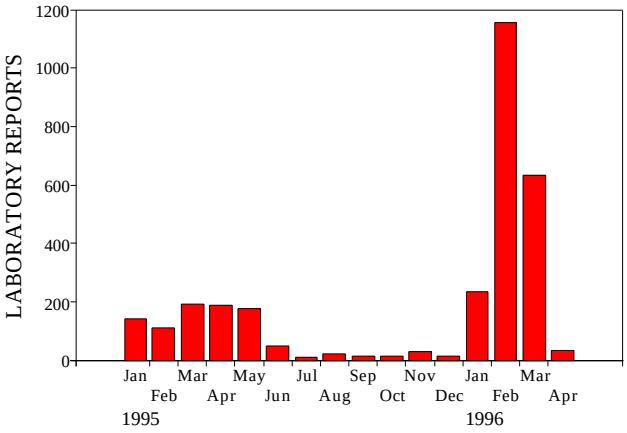


Figure 7. Respiratory syncytial virus laboratory reports, 1995 to 1996, by month of specimen collection.

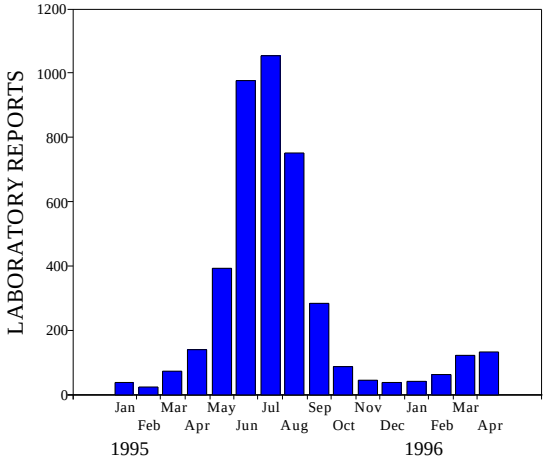


Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 18 April to 1 May 1996, historical data[2], and total reports for the year

.	State or Territory [1]							Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA										
Measles virus				1				1	37.5	25
Rubella virus	1							1	16.3	222
HEPATITIS VIRUSES										
Hepatitis A virus		4				2		6	17.9	154
Hepatitis D virus				1				1	.5	8
Hepatitis E virus							1	1	.3	1
ARBOVIRUSES										
Ross River virus		7			1		16	24	104.7	2,090
Flavivirus (unspecified)		3						3	1.0	19
ADENOVIRUSES										
Adenovirus type 40							1	1	.0	5
Adenovirus type 41						1		1	.0	3
Adenovirus not typed / pending	4	12		58		1	4	79	38.2	588
HERPES VIRUSES										
Herpes simplex virus type 1	1	36	1	141		2	29	210	158.5	2,364
Herpes simplex virus type 2		22		89		3	39	153	174.8	2,296
Herpes simplex not typed / pending	8	34				1	1	44	24.7	256
Cytomegalovirus	2	10		54			2	68	64.0	615
Varicella-zoster virus		8		9		1	9	27	41.8	489
Epstein-Barr virus		13				4	11	28	61.5	731
Herpes virus group - not typed							2	2	.5	59
OTHER DNA VIRUSES										
Parvovirus							1	1	1.7	42
PICORNA VIRUS FAMILY										
Coxsackievirus A9		1						1	.0	9
Echovirus type 9		3						3	.2	19
Echovirus type 14		1						1	.0	24
Echovirus type 22		1						1	.5	5
Poliovirus type 1 (uncharacterised)		1						1	.7	6
Rhinovirus (all types)		3		37			7	47	23.2	249
Enterovirus not typed / pending				38			8	46	42.5	361
ORTHO / PARAMYXOVIRUSES										
Influenza A virus				1				1	34.0	73
Parainfluenza virus type 1		7		31		4		42	24.8	86
Parainfluenza virus type 2				5				5	14.7	27
Parainfluenza virus type 3				23		3		26	16.0	273
Parainfluenza virus type 4				3				3	.0	3
Respiratory syncytial virus		51		85		3	11	150	62.2	523
OTHER RNA VIRUSES										
HIV-1							1	1	6.2	51
Rotavirus	6	2						8	30.0	299

Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 18 April to 1 May 1996, historical data[2], and total reports for the year, continued

.	State or Territory [1]							Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	Tas	Vic	WA			
OTHER										
Chlamydia trachomatis not typed	7	5			3		32	47	85.5	1,233
Chlamydia psittaci							1	1	5.7	49
Chlamydia species		7						7	1.0	60
Mycoplasma pneumoniae	1	11				3	3	18	23.3	204
Coxiella burnetii (Q fever)		4						4	8.8	54
Streptococcus group A							2	2	14.8	151
Bordetella pertussis						1	3	4	10.2	180
Legionella pneumophila						1		1	.0	3
Treponema pallidum		3				1	2	6	16.8	126
Schistosoma species							3	3	1.5	129
TOTAL	30	249	1	576	4	31	189	1,080	1166.4	14,164

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 8. Virology and serology laboratory reports by contributing laboratories for the reporting period 18 April to 1 May 1996,

	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	TOTAL
MEASLES, MUMPS, RUBELLA												
Measles virus				1								1
Rubella virus							1					1
HEPATITIS VIRUSES												
Hepatitis A virus						1					5	6
Hepatitis D virus						1						1
Hepatitis E virus						1						1
ARBOVIRUSES												
Ross River virus							2		5		17	24
Flavivirus (unspecified)											3	3
ADENOVIRUSES												
Adenovirus type 40											1	1
Adenovirus type 41					1							1
Adenovirus not typed / pending				37	12		1	10			19	79
HERPES VIRUSES												
Herpes simplex virus type 1				24			105	9		40	32	210
Herpes simplex virus type 2				4			65	1		50	33	153
Herpes simplex not typed / pending				4						6	34	44

Table 8. Virology and serology laboratory reports by contributing laboratories for the reporting period 18 April to 1 May 1996, continued

	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	TOTAL
Cytomegalovirus			1	33			1				33	68
Varicella-zoster virus							17				10	27
Epstein-Barr virus						2	1		1		24	28
Herpes virus group - not typed							1			1		2
OTHER DNA VIRUSES												
Parvovirus											1	1
PICORNA VIRUS FAMILY												
Coxsackievirus A9					1							1
Echovirus type 9											3	3
Echovirus type 14											1	1
Echovirus type 22											1	1
Poliovirus type 1 (uncharacterised)											1	1
Rhinovirus (all types)				43			1	1			2	47
Enterovirus not typed / pending	1	2		24	8		3				8	46
ORTHO / PARAMYXOVIRUSES												
Influenza A virus				1								1
Parainfluenza virus type 1				36							6	42
Parainfluenza virus type 2				5								5
Parainfluenza virus type 3		1		22							3	26
Parainfluenza virus type 4				3								3
Respiratory syncytial virus				125	1						24	150
OTHER RNA VIRUSES												
HIV-1											1	1
Rotavirus					8							8
OTHER												
Chlamydia trachomatis not typed										18	29	47
Chlamydia psittaci											1	1
Chlamydia species											7	7
Mycoplasma pneumoniae				3							15	18
Coxiella burnetii (Q fever)											4	4
Streptococcus group A											2	2
Bordetella pertussis				2							2	4
Legionella pneumophila				1								1
Treponema pallidum											6	6
Schistosoma species											3	3
TOTAL	1	3	1	368	31	5	198	21	6	115	331	1180

Table 9. Laboratory reports by contributing laboratory for the reporting period 18 April to 1 May 1996

STATE OR TERRITORY	LABORATORY	REPORTS
Australian Capital Territory	Woden Valley Hospital, Canberra	31
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	158
	Royal North Shore Hospital, St Leonards	15
	South West Area Pathology Service, Liverpool	75
Queensland	State Health Laboratory, Brisbane	574
Tasmania	Northern Tasmanian Pathology Service, Launceston	3
Victoria	Monash Medical Centre, Melbourne	30
Western Australia	PathCentre Virology, Perth	159
	Royal Perth Hospital	35
TOTAL		1180