

Isolation of β -lactamase positive vancomycin resistant *Enterococcus faecalis*; first case in Australia

Thomas McAlister,¹ Narelle George,¹ Joan Faoagali¹ and Jan Bell²

Abstract

An increasing number of clinical isolates of vancomycin resistant enterococci (VRE) have been reported in the literature since 1988. Only a few cases of β -lactamase producing VRE have been described worldwide. This article reports the first case of β -lactamase positive VRE in Australia. This strain of *Enterococcus faecalis* was isolated from a patient with non-Hodgkin's lymphoma who subsequently underwent a bone marrow transplant. Screening of all ward contacts did not detect any further case of β -lactamase producing VRE. With the development of multiple antibiotic resistance in enterococci, additional infection surveillance protocols have been implemented in the hospital. These include routine screening of 'at risk' patients, instigating relevant infection control measures for management of VRE positive patients and controlling the usage of vancomycin in order to limit the development of resistant isolates. *Commun Dis Intell* 1999;23:237-239.

Introduction

Enterococci are intrinsically resistant to a wide range of antibiotics. Traditionally, vancomycin and amoxicillin are the drugs of choice for treatment of enterococcal disease, however, the choice of therapeutic options has been markedly reduced with the emergence of β -lactamase producing, vancomycin resistant strains of

enterococci.¹ Since the first reported case of vancomycin resistance in enterococci in Britain² in 1988, further cases have occurred throughout Europe and America. In Australia, vancomycin resistant enterococci (VRE)³ were first reported in 1994.³ It was not until 1996⁴ that VRE were first detected at the Royal Brisbane Hospital (RBH).

1. Division of Microbiology, Queensland Health Pathology Service, Royal Brisbane Hospitals Campus, Herston, Queensland, 4029
2. Microbiology and Infectious Diseases, Women's and Children's Hospital, Adelaide, South Australia, 5006

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This article documents the first reported case of β -lactamase production in a vancomycin resistant isolate of enterococcus in Australia.

Case report

A 28 year old male was diagnosed with non-Hodgkin's lymphoma in April 1996. He received a bone marrow transplant in March 1997. Subsequently the patient developed a number of febrile episodes (up to 40.2°C) in the period preceding his death on 25 March 1997.

During March 1997, the laboratory investigations revealed severe neutropaenia and thrombocytopaenia.

Staphylococcus aureus was cultured from peripheral blood and the lumen of his Hickman's catheter. Cytomegalovirus was cultured from his urine. Faecal specimens were positive for *Clostridium difficile* toxin A. A β -lactamase positive *Enterococcus faecalis* (*E. faecalis*; *VanB* phenotype) was isolated from his faeces on 23 March 1997.

In the four months from December 1996 to March 1997 the patient was treated with acyclovir, amphotericin B, ceftazidime, co-trimoxazole, cyclosporin, fluconazole, imipenem, metronidazole, norfloxacin, piperacillin, tetracycline, ticarcillin-clavulanic acid and tobramycin. During the same period, five courses of vancomycin were administered, three of these between January and March.

Methods and Results

Laboratory investigations

Enterococcus faecalis was grown following routine screening of his faeces on Enterococcosel (ESEL) agar (BBL) which contained 6mg/L vancomycin. Identification tests included aesculin hydrolysis, gram stain, motility, pyrrolidonyl arylamidase (PYR) activity and Strep API (bioMérieux). Vancomycin resistance was determined by the E Test™ (AB Biodisk, Sweden) method. The minimum inhibitory concentration (MIC) of vancomycin was 48 mg/L. The isolate was susceptible to teicoplanin with an MIC of 0.15 mg/L. These results classified the organism as a VRE with a *VanB* phenotype. The organism was referred to the Women's and Children's Hospital, Adelaide, Australia for genotypic analysis. The identification of the organism was confirmed using a multiplex polymerase chain reaction (PCR) assay based on specific detection of genes encoding D-alanine: D-alanine ligases; and PCR primers to 330 base pair fragments following direct amplification were used to confirm the *VanB* genotype.⁵ Additional antibiotic susceptibility testing using agar dilution also showed the organism to be resistant to 500 mg/L gentamicin. The Nitrocefin test (Oxoid) for β -lactamase was positive after 15 minutes.

Infection control

Following the bone marrow transplant, the positive patient was nursed in a single room in the Oncology ward. Barrier nursing techniques were implemented. The patient subsequently died two days after the specimen was sent to the laboratory, before the isolate was fully identified. Rectal swabs collected from all patients within the ward were culture negative for VRE. Environmental samples were collected from the walls, cupboards, bed frame, mattress and pillowcase in the single room. No VRE were isolated from any of the environmental samples. All

horizontal surfaces, walls and the bed frame in the patient's room were cleaned with neutral detergent solution.

Discussion

With the emergence of vancomycin resistance in enterococci, the choice of antibiotics for treatment of VRE infections is severely restricted.¹ However, amoxycillin still remains a therapeutic option in cases of disease due to vancomycin resistant strains of *E. faecalis*. With the emergence of β -lactamase producing strains of enterococci, amoxycillin becomes ineffective. Only a few cases of β -lactamase producing, vancomycin resistant *E. faecalis* have been reported worldwide.⁶ Two other β -lactamase positive strains of enterococci have been isolated in Australia, however, this is the first documented case of β -lactamase production in a vancomycin resistant isolate.

The clinical history of the positive patient highlights many of the key 'at risk' criteria that one would expect for development of VRE infection. Firstly, the patient was diagnosed with non-Hodgkins lymphoma and was severely immunosuppressed prior to and following a bone marrow transplant. Secondly, during the course of his illness, he had been exposed to a wide range of broad spectrum antibiotics which would have the potential to promote colonisation by organisms such as enterococci. In addition to this, he had received five courses of vancomycin, three of which were in the two month period immediately preceding the detection of vancomycin resistant enterococci. The reason for the selection for β -lactamase production is not as clear cut. However, piperacillin and ceftazidime were used as prophylactic antibiotic cover during the period preceding his bone marrow transplant.

Since 1996, rectal swabs have been routinely collected on a weekly basis to screen all haematology/oncology and intensive care patients at Royal Brisbane Hospital for VRE. Similarly, any faecal sample submitted for routine microbiological investigation from patients in the Bone Marrow Transplant Unit are also screened for VRE. The samples are screened by plating on ESEL vancomycin medium. Colonies showing evidence of aesculin hydrolysis are subcultured for further identification tests. All enterococci are tested by the agar dilution method against a number of antibiotics including amoxycillin 2 mg/L, vancomycin 4 mg/L, gentamicin 500 mg/L and streptomycin 2,000 mg/L.

β -lactamase tests are routinely performed on all enterococcal isolates. Any strain of enterococcus which is resistant to vancomycin at 4 mg/L has vancomycin and teicoplanin minimum inhibitory concentrations (MICs) determined using the Etest™ method.

On detection of VRE, the infection control unit is notified and screening of close ward contacts is undertaken. In this instance, all other patients within the ward were culture negative for VRE. Because enterococci can survive for long periods of time in the environment, it is recommended that environmental sampling be undertaken prior to cleaning to establish the extent of the potential contamination.

When VRE is isolated from a patient at RBH, he/she is transferred to a single room in the Infectious Diseases Unit. Rectal swabs are then collected weekly and screened for VRE until there are three consecutive negative samples. Meanwhile, his/her previous room is cleaned with a neutral detergent solution and the bedding and drapes are replaced. Swabs taken from various cleaned horizontal surfaces are then collected and screened for VRE prior to reuse.

Outbreaks of VRE infection are usually a result of dissemination of a single clone either via hospital personnel or contact with contaminated fomites. Implementation of strict infection control practices involving barrier nursing and isolation of positive patients is required to limit the outbreak.^{7,8} Failure to implement such precautions may result in the establishment of multiple endemic strains. Greatly increased usage of broad spectrum antibiotics including third generation cephalosporins and vancomycin have also contributed to escalating numbers of VRE isolates. With the emergence of β -lactamase producing VRE, therapeutic options are diminishing even further. Vancomycin usage should be restricted to limit the potential proliferation of VRE.⁸

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Notice

Update of subscription details

Tenders have been invited for the printing and mailing of *CDI*.

As part of the process a review of the current mailing list is currently in progress.

Readers are asked to provide current subscription details by filling out the detachable form on page 257 of this issue, and returning it to either:

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This form will also appear in the next issue of *CDI*. At the end of October the mailing list will be updated to include only those who have returned forms.

An outbreak of measles in a rural Queensland town in 1997; an opportunity to assess vaccine effectiveness

Heather F. Gidding,^{1,2} Susan Hills,³ Linda Selvey,¹ Leslee A. Roberts,² Susan Johnston⁴

Abstract

This report describes a measles outbreak in a rural town in south-east Queensland and presents the results of a vaccine effectiveness (VE) study performed during this outbreak. It is important to assess the effectiveness of a vaccine in an outbreak to determine if the outbreak is due to failure of the vaccine or failure to vaccinate. There were 44 cases of measles amongst local residents, which represents a notification rate of 396.7 per 100,000 population. Case investigations identified a group of people who had been exposed to measles at a seminar. The attack rate for the seminar cohort was 18% (11/61). This presented an opportunity to conduct a VE study using data about children aged less than 16 years who attended the seminar. In this cohort of 23 attendees, all 7 children who had not received any measles vaccinations became cases whilst the 6 who were fully vaccinated for their age according to NHMRC guidelines were protected from measles illness. Although there were insufficient fully vaccinated cohort members to reliably estimate VE for this group, the vaccine was 84.6% (95% CI: 15.0-99.7%) effective for those who had received at least one validated dose of vaccine. Despite the sample size limitations, the results support the view that failure to vaccinate rather than vaccine failure contributed to the high infection rate in the seminar cohort. *Commun Dis Intell* 1999;23:240-245.

Introduction

On 9 September 1997, a case of measles in a resident of a rural town in south-east Queensland was notified to Queensland Health. Serologically confirmed cases of measles had already been reported from a neighbouring town. By 15 September 7 measles notifications had been received. Investigation of these cases revealed that some had attended a local education seminar on 28 August.

Investigation of the education seminar cohort identified an opportunity to assess the effectiveness of the measles vaccine. Assessing the effectiveness of a vaccine in an outbreak is important to determine if the outbreak is due to failure of the vaccine or failure to vaccinate. Vaccine efficacy is usually determined under controlled conditions prior to licensing, however the effectiveness of a vaccine under normal (field) conditions may vary and should be assessed when the opportunity arises. Vaccines may fail due to incorrect storage and administration of the vaccine, drug interference or the age at vaccination. Vaccine effectiveness may vary in different populations.¹ Establishing that a vaccine is effective during an outbreak provides support for continuing efforts to improve vaccination coverage levels and may allow the appraisal of current vaccination strategies.

This report describes a measles outbreak in a rural town in south-east Queensland during 1997 and provides the results of a vaccine effectiveness (VE) study. The relationship of this outbreak to the state-wide outbreak is also discussed.

Methods

Case definitions

The NHMRC measles case definitions for national surveillance and a confirmed case² (Box) were used to define a case as being notifiable to the Queensland

Box. Measles case definitions for notification to the Queensland Notifiable Diseases Registry²

Measles case definition for national surveillance

An illness characterised by all of the following features:

- (a) a generalised maculopapular rash lasting three or more days; and
- (b) a fever exceeding 38.3°C; and
- (c) cough or coryza or conjunctivitis or Koplik spots.

Measles confirmed case definition

A person with signs and symptoms consistent with measles and any one of the following:

- (a) measles virus detected in an appropriate specimen; or
- (b) the presence of measles specific IgM antibody; or
- (c) a fourfold rise in measles antibody titre in sera obtained at least two weeks apart; or
- (d) history of contact with a laboratory confirmed case.

1. Communicable Diseases Unit, Queensland Health, Brisbane, Queensland
2. Master of Applied Epidemiology Program, National Centre for Epidemiology and Population Health, Australian National University, Australian Capital Territory
3. Central Public Health Unit Network, Rockhampton, Queensland
4. Central Public Health Unit Network, Wide Bay, Queensland

Notifiable Diseases Registry (NODS). A presumptive case was defined as having an illness characterised by a morbilliform rash, cough, and fever present at the time of rash onset.²

Notified cases with onset dates between 15 August and 31 December 1997 were considered part of the state-wide outbreak. This time period was chosen because 15 August was the onset date of the first notified case in 1997 from the town where this outbreak was first recognised, and the number of State notifications returned to pre-outbreak levels by 31 December. Cases considered part of the local outbreak were those with onset dates during the outbreak who resided in the rural Queensland town, or were linked to the education seminar.

Outbreak notification rates were calculated using the 1996 census population supplied by the Australian Bureau of Statistics. The distributions of local cases by age, sex, date of onset, statistical local area of residency, and method of diagnosis (clinical or laboratory confirmed) were determined.

Active case finding

All possible measles cases reported to the Central Queensland Public Health Unit outbreak investigation team were questioned about whether they had the symptoms and signs that defined a presumptive or notifiable case. Cases identified as being presumptive or notifiable were asked about their contact with other people during the period 1 day prior to developing signs of measles to 4 days after developing a rash. This period was defined as the infectious period of a case.³ Individuals who had been in contact with an infectious case were interviewed to determine whether they had the symptoms and signs of measles. Presumptive or notifiable cases were also questioned about their contact with other people in the incubation period 7 to 18 days prior to developing signs of measles infection.³ People identified as having had contact with a case during this period were then interviewed about whether they had the symptoms and signs of measles. Serological confirmation was recommended for presumptive cases that did not have incubation periods consistent with contact with an infectious case, or when clinical symptoms did not conform to those used to define a notifiable case. Notifiable cases identified by these active case finding methods were allocated to one of the case types described in Table 1.

Vaccine effectiveness study

The cohort

Interviews with presumptive and notifiable primary cases about their attendance at any gatherings during their defined infectious period identified 11 cases who had attended the education seminar. Investigation of these cases identified one person who had coryzal symptoms and conjunctivitis at the seminar and developed a rash the next day. This case was therefore defined as being the probable source case for the seminar cohort.

The education seminar was investigated further to identify a suitable cohort for performing a VE study. Details about the seminar were obtained from the seminar coordinator.

The seminar was in a hall and there were two sessions with a tea break in between. The first session ran from 2 p.m. until approximately 5.30 p.m. and the second ran from 7 p.m. until 9 p.m. A list of people who attended the

education seminar was constructed by the seminar coordinator and telephone interviews were conducted with available attendees. Seminar attendees, or a parent of an attendee, were questioned at least 18 days after the seminar (that is, after the defined incubation period for exposure to an infectious case at the seminar). Questions were asked about attendance at each of the seminar sessions, the attendees' age, their measles vaccination status, and whether they had the symptoms and signs that defined a presumptive or notifiable case. Attendees were also asked if they could provide the names of any other attendees in order to establish the completeness of the attendance list. The interviews established that the probable source case only attended the afternoon session

Table 1. Case definitions used during the investigation of a measles outbreak in a rural Queensland town, and at an education seminar in that town, 15 August - 31 December 1997

Type of case	Definition
Primary case	A presumptive or notifiable case. ⁺
Secondary case	As for a primary case, <i>and</i> had an incubation period [#] consistent with exposure to an infectious* primary case.
Probable source case	As for a primary case, <i>and</i> whilst infectious,* they had contact with a primary case, <i>and</i> this contact was within the time frame for the incubation period [#] of the primary case.
Probable source case linked to the seminar	As for a primary case, <i>and</i> attended the seminar whilst infectious.*
Primary case linked to the seminar	As for a primary case, <i>and</i> attended the seminar, <i>and</i> had an incubation period between 7 and 18 days after the seminar.
Secondary case linked to the seminar	As for a secondary case, <i>and</i> their primary case attended the seminar.
Primary case indirectly linked to the seminar	As for a primary case linked to seminar, <i>except</i> a family member (not the case) attended the seminar.
Secondary case indirectly linked to the seminar	As for a primary case, <i>and</i> had an incubation period [#] consistent with exposure to an infectious* primary case indirectly linked to the seminar.

+ See Box for definitions.

[#]The incubation period was defined as between 7 and 18 days after contact with an infectious case.³

of the seminar and all primary cases linked to the seminar also attended this session. Therefore, attendees of the afternoon session of the seminar were a suitable cohort to perform a VE study.

The age distribution of attendees at the afternoon session of the seminar fell into two categories; children (aged between 2 and 15 years), and adults (aged at least 30 years). Vaccine effectiveness calculations were performed only for the children. Due to the measles vaccine not being widely available in Australia until 1970, adults aged at least 30 years are unlikely to be vaccinated against measles and most would be immune due to past exposure to the disease.⁴ Since vaccination status and disease risk in the adult group are likely to depend on whether an individual has had past exposure to measles, VE calculations including this group would give spuriously low results.¹ The attendee thought to be the source of infection for the seminar cohort was excluded from the VE study as they were not at risk of infection at the seminar. Attendees with an unknown age, unknown vaccination status, or a history of measles were also excluded (as recommended by Orenstein, Bernier and Hinman¹).

Determining the vaccination status

Measles vaccination dates were determined by asking parents of children who attended the seminar to read out the vaccination dates entered in childhood immunisation books. If a vaccination date could not be provided, consent to access the child's general practitioner and council records was requested. For the purposes of this study, a vaccination was considered to be 'validated' if the vaccination date could be obtained from any of the above sources. The vaccination status for children indicating they had a prior history of measles or who were not vaccinated was not confirmed. Children were defined to be 'fully vaccinated' if they had received all vaccinations recommended for their age⁵ (that is, children aged between 1 and 9 years required one dose of measles vaccine and children aged 10 years or over required two doses to be defined as fully vaccinated).

Vaccine effectiveness (VE) was calculated using the following formula:¹

$$VE(\%) = \{(ARU - ARV) / ARU\} \times 100$$

where ARU=attack rate in the unvaccinated

and ARV=attack rate in the vaccinated

Exact 95% Confidence Intervals for the VE estimates were calculated using STATA.⁶ All other analyses were performed using Epi Info 6.04b.⁷

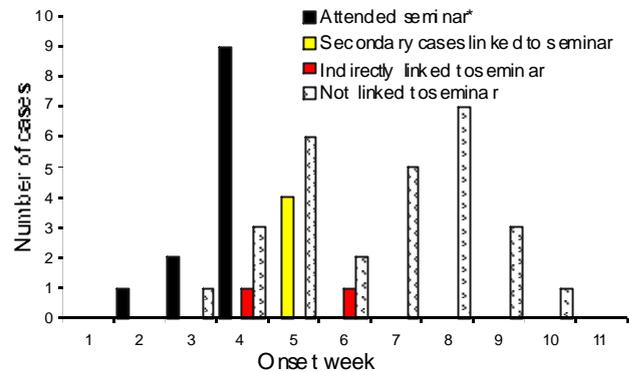
Results

The outbreak

Forty-six cases of measles were notified as part of the local outbreak (Figure 1). Two cases were visiting the area to attend the education seminar and 44 cases were local residents. This represents a notification rate for the area of 396.7 per 100,000 population (44/11,092). The local notification rate was 64 times the outbreak rate for Queensland (6.2/100,000 population).

During the outbreak period, more notifications were from this town than from any other area. Local cases accounted for 21% (44/208) of the State's outbreak notifications.

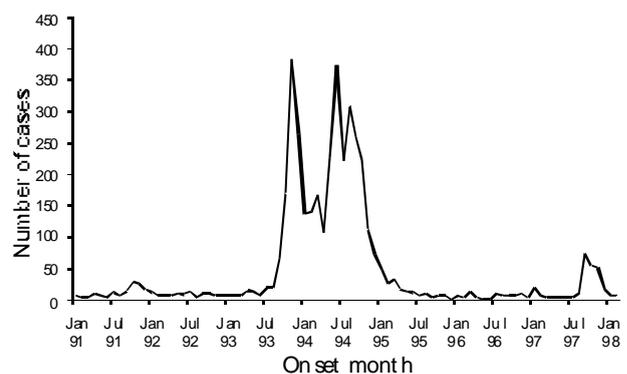
Figure 1. Measles notifications from a rural Queensland town and notifications linked to an education seminar in that town, by week of onset, 15 August-31 December 1997



*Probable source case for seminar cohort and primary cases linked to seminar

From this town, and a neighbouring town where the outbreak was first recognised, the outbreak appeared to spread across Queensland and interstate. This outbreak was the largest in Queensland since the epidemic of 1993 and 1994 (Figure 2).

Figure 2. Measles notifications for Queensland by month of onset, January 1991 to February 1998



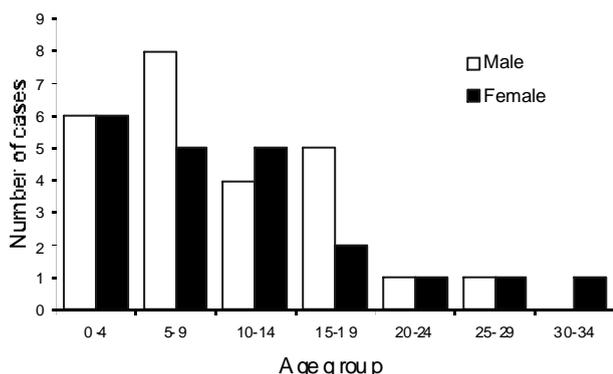
Eighteen measles notifications associated with the local outbreak were linked to the education seminar and 28 cases were local residents who were not linked to the seminar (Figure 1). Of the 18 cases linked to the seminar, one case was the defined probable source case for the seminar cohort, 11 were primary cases who had attended the seminar, 4 were secondary cases linked to the seminar, and 2 cases occurred in siblings whose well parents had attended the seminar. One of these siblings became ill 10 days after the seminar and was therefore a

primary case indirectly linked to the seminar. The other sibling was a secondary case indirectly linked to the seminar.

The probable source case for cases linked to the education seminar was an unvaccinated child who went to school in the town where the state-wide outbreak was first identified. Other cases had previously been identified who went to the same school. The source of infection for the 28 cases who were not linked to the education seminar is unknown.

The age range for notified cases associated with the local outbreak was 11 months to 34 years (median age 9 years). The number of notified cases generally decreased with increasing age (Figure 3). Only 3 cases were aged 1 year or younger. However, over half (54.3%) of the notified cases were aged less than 10 years. Thirty-nine out of the 44 local cases (88.6%) were aged less than 20 years. The notification rate for this age group was 1,090 per 100,000 population (39/3,579). There were slightly more male notifications (M:F ratio 1.2:1).

Figure 3. Measles notifications from a rural Queensland town and notifications linked with an education seminar in that town, by age and sex, 15 August-31 December 1997



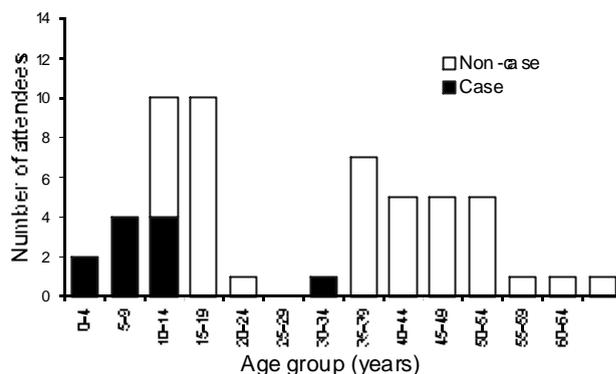
Ten of the 46 cases (21.7%) associated with the local outbreak were serologically confirmed, 2 of whom were primary cases linked to the education seminar. Two of the 3 children aged 1 year or younger were serologically confirmed.

The vaccine effectiveness study

The education seminar cohort

Sixty-two people were identified as having attended the afternoon session of the education seminar. The names of 57 attendees were on the list of attendees provided. Five extra attendees were identified by interviewing the listed attendees. Information was available to determine the disease status for all 62 seminar attendees. Excluding the probable source case for the seminar cohort, the measles attack rate was 18% (11/61). Of the 61 attendees exposed to the probable source case, 27 were defined as children and 26 as adults (Figure 4). Eight attendees (12.9%) refused to answer questions about their vaccination status and age.

Figure 4. Seminar attendees by measles status and age group



Of the 12 cases that had attended the seminar, only 7 had been notified. The other 5 (41.7%) were identified by the case investigation methods. Similarly, of the 6 secondary cases linked to the seminar, 5 (83.3%) were identified by the case investigation methods.

The vaccine effectiveness study cohort

Of the 27 seminar attendees who were known to be children and could have been exposed to measles at the seminar, 3 gave a history of past infection with measles and 1 child's vaccination status was unknown (Table 2).

Table 2. Vaccination status of children who attended the education seminar by age group

Age group	1-9 years	10-15 years
Not vaccinated	5	2
History of measles	0	3
1 dose of measles vaccine (% validated [#])	5 (100%)	10 (70%)
2 doses of measles vaccine	0	1*
Unknown vaccination status	1	0
Total	11	16

[#]Validated = vaccination date provided by parent, doctor or council

The VE study cohort consisted of 23 attendees excluding the 4 cases noted above. Of these attendees, 15 had received one dose of measles vaccine and one had received two doses. Thirteen of these 17 doses (76.5%) were supported by the report of a vaccination date. The date of vaccination was provided by the parent (8/13), doctor (2/13) or the council (3/13).

In this cohort, all children who had not received any vaccinations against measles (n=7) became cases, whilst those who were fully vaccinated (n=6) were protected from disease. However, the small sample size of this

Table 3. Measles vaccine effectiveness for children who attended the education seminar

Comparison	Attack rate in vaccinated	Attack rate in unvaccinated	Vaccine effectiveness % (95% CI*)
Received at least one dose vs never vaccinated	3/16	7/7	81.3 (17.0-98.8)
Received at least one validated dose* vs never vaccinated	2/13	7/7	84.6 (15.0-99.7)

* Validated = vaccination date provided by parent, doctor or council

* CI = Confidence Interval

comparison meant that the VE could not be estimated with any precision. For children who had received at least one dose of measles vaccine, the point estimate VE was 81.3% (Table 3). When considering only validated doses, the VE was 84.6%. There were insufficient cohort members to stratify VE calculations by age.

Discussion

The outbreak

The outbreak notification rate in this town was high, especially compared to rates for the rest of Queensland. The rate is still likely to be an underestimate of the true incidence. A large proportion of cases associated with the education seminar were only identified by active case finding during the investigation. Reasons for the high rates in this town compared to the rest of Queensland are unclear. Data recorded on the Queensland vaccination register does not suggest that vaccination coverage levels for local children are lower than for the rest of the State (assuming the completeness of the vaccination register is consistent across Queensland). However, the vaccination register only provides vaccination coverage levels for children up to the age of 2 years at present, therefore population coverage levels cannot be determined. For children aged 2 years or younger, the available vaccination coverage levels do indicate a degree of protection which is consistent with there being few local cases in this age group. Prior to the outbreak, only one measles notification had been received from this town since notifications were first recorded on the NODS data base in 1991. If notifications are equated to the disease incidence and hence to exposure, past exposure is likely to be minimal, which may explain the high local rates during this outbreak.

Local cases were distributed evenly among children aged less than 10 years. This is unlike the age distribution of national notifications for 1996⁸ and State notifications during the outbreak, where the most susceptible age group was infants too young to be vaccinated. Locally, infants may have been under-represented as contacts with the virus, especially at the education seminar which was for school aged children and their parents. The seminar was the most likely source of measles infection for a significant proportion (23.9%) of the local outbreak cases.

Transmission of measles at the seminar was probably by airborne droplet nuclei.⁹ As the measles virus is known to be viable for several hours in droplet form,⁹ people who attend the same room within 2 hours of an infectious patient are considered 'at risk' of infection.² Therefore the

high attack rate amongst afternoon seminar attendees is not unexpected. Transmission also occurred indirectly to one child whose well parents had attended the seminar.

This child may have been infected via contact with articles freshly soiled with nasal or throat secretions containing the measles virus.³

Vaccine effectiveness

The results of this study support the view that the measles vaccine was effective in preventing infection in the study cohort. All children who attended the education seminar and were fully vaccinated were protected against disease. In addition, the point estimates of VE for children having received at least one dose of vaccine are consistent with the findings of other VE studies^{10,11} that indicate that the vaccine was effective in other settings. However, the outbreak investigation could only identify a small cohort to determine the VE, hence the VE estimates have wide confidence intervals due to sample size limitations. The inability to obtain precise VE estimates highlights one of the difficulties encountered when trying to estimate VE during an outbreak. Despite these limitations, it was important to have performed the study as the results do not suggest the vaccine failed and provide support for ongoing efforts to improve vaccination coverage.

Selection and misclassification biases can affect VE estimates. Problems with the sensitivity and specificity of the case definition, case ascertainment, validity of vaccination and disease histories, comparability of vaccinated and unvaccinated cohort members, and non participation can bias the VE estimate. Vaccine effectiveness calculations using the seminar cohort minimise many of the biases that can be encountered when estimating VE.

The definitions used to define a notifiable case were applied to all cohort members equally by asking each attendee, or their parent, whether they had the symptoms and signs that defined a presumptive or notifiable measles case. Two primary cases linked to the seminar were serologically confirmed. These two measures should have minimised misclassification of the disease status of cohort members. All known seminar attendees were followed for a time that would have identified the cases in this group. Therefore unequal case ascertainment is unlikely to affect the VE estimates. It is possible that not all attendees were identified, and the effect this would have on the VE estimates is unknown. This possibility was minimised by obtaining a list of attendees and asking identified attendees if they knew anyone else who attended. Only one VE cohort member had an unknown vaccination

status, further minimising the possibility of non participation bias. Eight attendees were excluded because they refused to provide information about their age and vaccination status. Orenstein, Bernier and Hinman recommend these unknowns be excluded as it is difficult to predict how they would distribute themselves with regard to vaccination status.¹

The VE estimates are unlikely to be affected by differences in past exposure to measles between vaccinated and unvaccinated cohort members. Firstly, cohort members with a stated history of measles were excluded regardless of vaccination status. Secondly, as previously discussed, past exposure is likely to be minimal in the age groups used for the calculations (assuming most children have been long term residents of the area). Exposure during the current outbreak is also relatively uniform as all attendees were in the same hall as the source case for a similar amount of time.

A high proportion of vaccinations could be validated by report of a vaccination date. Council and general practitioner records are likely to be accurate, and would not be biased by a knowledge of the attendee's disease status. Parental recall has been found to be an unreliable method of determining vaccination status.¹² To minimise recall bias, parents were asked to read out the dates of vaccination from written records. Similar methods have been used in previous Australian studies^{10,11} and it is unlikely that parents would fabricate a vaccination date. However the validity of this method remains unproven. No attempt was made to validate a history of measles or vaccination records for VE cohort members stating they had not been vaccinated. Therefore the results of this study need to be interpreted with caution. Some members who stated they were unvaccinated may have been vaccinated, and this would lead to a biased estimate of VE if their reported vaccination status was biased by whether they were a case.

Conclusion

This outbreak investigation identified an opportunity to assess the effectiveness of the measles vaccine under field conditions. There is an ongoing need to assess vaccine effectiveness in order to establish that a vaccine is effective in a given situation, and to provide support for efforts to improve vaccination coverage levels. This vaccine effectiveness study supports the view that failure to vaccinate rather than vaccine failure contributed to the high infection rate in the education seminar cohort.

Acknowledgments

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New publications

Hepatitis C

The Communicable Diseases Network Australia New Zealand (CDNANZ) has recently released two new publications on hepatitis C.

CDNANZ recently endorsed the *Australian Hepatitis C Surveillance Strategy* developed by the Hepatitis C Surveillance Committee of CDNANZ. This strategy builds on existing surveillance activities and proposes an implementation plan that is now under discussion between the relevant agencies.

Additional copies can be obtained from:

National Centre in HIV Epidemiology and Clinical Research
University of New South Wales
376 Victoria Street
DARLINGHURST NSW 2010
AUSTRALIA

The third publication in the *Communicable Diseases Intelligence Technical Report Series (No. 3)* is *Epidemiology of the hepatitis C virus* by Nick Crofts, Sandy Thompson and John Kaldor.

This report summarises the state of knowledge of the epidemiology of hepatitis C as at the end of 1998, both internationally and in Australia. It includes:

- estimates and projections developed by the Australian National Council on AIDS and Related Diseases Hepatitis C Virus Projections Working Group as part of the 1998 review of Australia's response to the hepatitis C epidemic; and
- detailed discussion on the main routes of transmission and the natural history of hepatitis C.

A Framework for an Australian Influenza Pandemic Plan

A framework for an Australian Influenza Pandemic Plan, Version 1 June 1999, endorsed by the Communicable Diseases Network Australia New Zealand, is the fourth report in the *Communicable Diseases Intelligence Report Series*.

The Plan provides a strategic framework for the detection and management of an influenza pandemic in Australia. The aim of the Plan is to reduce the mortality, morbidity, social disruption and economic losses associated with an influenza pandemic. The Plan provides a national framework and direction for the development of plans at the State/Territory and local level, enabling State/Territories to link their own pandemic contingency plans (either existing or future) to the national Plan.

Key elements of the Plan include recommendations for:

- surveillance for the detection of a novel strain of influenza virus;
- surveillance measures to monitor the impact of a pandemic strain including evaluation on antiviral drug resistance in the event of a pandemic;
- preventative measures to reduce the impact of the spread of a pandemic strain;
- a communication strategy for rapid dissemination of information; and
- promotion of planning for the provision of medical care and the maintenance of essential community services.

The Plan is a working document rather than a final solution. It will continue to be revised, added to, altered and refined as knowledge grows and changes.

Copies are available on the Internet website: <http://www.health.gov.au/pubhlth/strateg/communic/tech/influenza.htm>

The *Communicable Diseases Intelligence Technical Report Series* can be obtained from:

The Publications Officer
Publications Unit (MDP 129)
Department of Health and Aged Care
GPO Box 9848
CANBERRA ACT 2601
AUSTRALIA

or Toll free telephone number: 1800 020 103 ext 8654

Communicable Diseases Surveillance

Highlights

Communicable Diseases Surveillance consists of data from various 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' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Vaccine preventable diseases

While the number of notifications of vaccine preventable diseases remains low when compared with historical data (Figure 1), a significant number of notifications of pertussis continues to be received.

For the 245 pertussis notifications in this reporting period, the male to female ratio is 1:1.8 and the majority of cases (17%) are in the 10-14 age group.

Meningococcal infection

The number of notifications of meningococcal infection is comparable with historical data with 64 notifications being received in this reporting period compared with 64 in the same period last year. Serogroup information is available for 45% of notifications received in 1999 and, of these, 62% are serogroup B and 32% are serogroup C. This is comparable with the proportions reported in the most recent Annual Report of the Australian Meningococcal Surveillance Programme. The male to female ratio for the current reporting period is 1.25:1 while the majority of cases (37%) are in the 0 to 4 years age group, 14% are in the 15-19 age group and 12% are in the 20-24 age group.

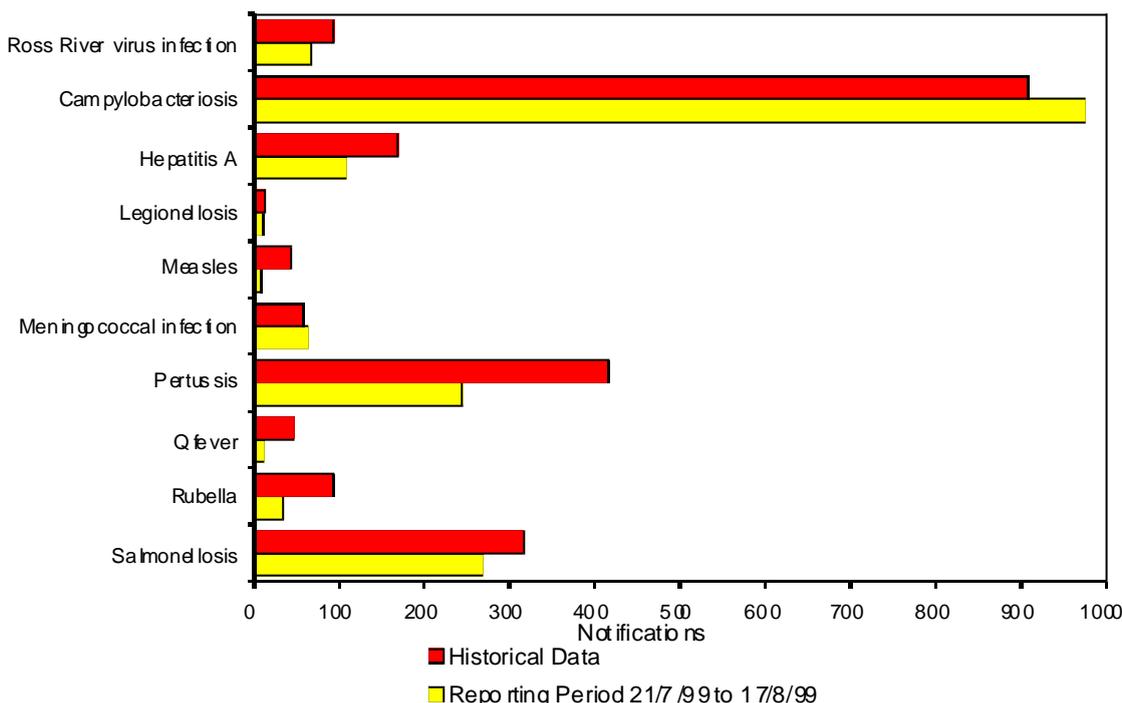
Tables

There were 5,209 notifications to the National Notifiable Diseases Surveillance System (NNDSS) in the four week period, 21 July to 17 August 1999 (Tables 1 and 2). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 1).

There were 3,222 reports received by the *CDI*/Virology and Serology Laboratory Reporting Scheme (LabVISE) in the four week period, 15 July to 11 August 1999 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 29 to 32, ending 15 August 1999, are included in this issue of *CDI* (Table 5).

Figure 1. Selected National Notifiable Diseases Surveillance System reports, and historical data¹



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 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 21 July to 17 August 1999

Disease ¹	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999 ²	Year to date 1998
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
<i>H. influenzae</i> type b infection	0	1	0	0	0	0	0	0	1	2	26	20
Measles	0	3	0	2	0	1	3	0	9	29	169	224
Mumps	0	3	0	2	1	0	6	4	16	19	112	109
Pertussis	11	86	0	41	29	26	49	3	245	317	2,220	4,582
Rubella ³	1	4	0	14	0	0	14	1	34	67	228	474
Tetanus	0	0	0	0	0	0	0	0	0	0	1	3

NN. Not Notifiable

1. No notification of poliomyelitis has been received since 1978.
 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 2. Notifications of diseases received by State and Territory health authorities in the period 21 July to 17 August 1999

Disease ^{1,2,3}	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999 ⁴	Year to date 1998
Arbovirus infection (NEC)	0	0	0	0	0	0	0	0	0	6	70	50
Barmah Forest virus infection	0	14	1	10	0	0	0	1	26	21	487	404
Brucellosis	0	0	0	1	0	0	0	0	1	4	16	28
Campylobacteriosis ⁵	16	-	19	67	224	24	487	139	976	948	7,877	7,204
Chancroid ⁶	0	0	0	0	0	0	0	0	0	0	0	1
Chlamydial infection (NEC) ^{6,7}	11	159	76	94	96	20	232	186	874	707	8,579	6,683
Cholera	0	0	0	0	0	0	0	0	0	0	2	3
Dengue	0	1	1	0	0	0	0	0	2	17	157	356
Donovanosis ⁶	0	0	2	1	NN	0	0	0	3	3	13	25
Gonococcal infection ⁸	6	78	100	38	22	2	57	80	383	384	3,526	3,308
Haemolytic uraemic syndrome ⁹	NN	0	1	0	0	0	NN	0	1	0	12	7
Hepatitis A	5	28	11	11	6	0	24	24	109	155	1,033	1,934
Hepatitis B incident	0	8	3	1	0	2	6	2	22	21	191	171
Hepatitis B unspecified ¹⁰	1	222	0	17	0	2	222	25	489	460	4,355	3,970
Hepatitis C incident	2	3	0	-	4	0	4	5	18	30	191	180
Hepatitis C unspecified ¹⁰	26	518	14	89	80	24	456	92	1,299	1,327	12,429	11,963
Hepatitis (NEC) ¹¹	0	2	0	0	0	0	0	NN	2	1	14	10
Hydatid infection	0	NN	0	0	0	0	2	0	2	5	20	25
Legionellosis	0	3	0	1	3	0	2	2	11	11	177	164
Leprosy	0	0	0	0	0	0	0	2	2	0	3	2
Leptospirosis	0	3	0	1	1	0	0	2	7	19	265	110
Listeriosis	0	0	0	0	0	0	2	0	2	4	29	38
Malaria	1	27	7	3	6	1	6	3	54	54	457	532
Meningococcal infection	0	30	2	4	6	3	15	4	64	64	311	246
Ornithosis	0	NN	0	0	1	0	3	0	4	4	52	26
Q Fever	0	5	0	4	0	0	2	1	12	38	300	337
Ross River virus infection	0	22	9	13	1	1	4	17	67	47	3,922	2,331
Salmonellosis (NEC)	6	58	19	27	32	4	72	51	269	294	5,327	5,300
Shigellosis ⁵	1	-	2	5	4	0	6	10	28	45	379	410
SLTEC, VTEC ¹²	NN	0	0	NN	2	0	NN	NN	2	0	17	8
Syphilis ¹³	1	41	23	36	0	1	1	3	106	135	1,195	907
TTP ¹⁴	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis	2	31	1	5	5	0	7	8	59	72	580	606
Typhoid ¹⁵	0	2	0	0	0	0	0	2	4	1	47	48
Yersiniosis (NEC) ⁵	0	-	0	4	1	0	1	0	6	11	100	157

1. Diseases preventable by routine childhood immunisation are presented in Table 1.

2. For HIV and AIDS, see Tables 6 and 7.

3. No notifications have been received during 1999 for the following rare diseases: lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.

4. 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.

5. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

6. Notifications from NSW have been received since September 1998, and were first reported in *CD* in Issue 23(9).

7. WA: genital only.

8. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.

9. Nationally reportable from August 1998.

10. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of testings being carried out.

11. Includes hepatitis D and E.

12. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC) became nationally reportable in August 1998.

13. Includes congenital syphilis.

14. Thrombotic thrombocytopenic purpura became nationally reportable in August 1998.

15. NSW, Qld: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Virology and serology laboratory reports by State or Territory¹ for the reporting period 15 July to 11 August 1999, and total reports for the year

	State or Territory ¹								Total this period	Total reported in 1999 ^{2,3,4}	
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
Measles, mumps, rubella											
Measles virus				2			3			5	140
Mumps virus							1	5		6	37
Rubella virus		1		15			5	2		23	86
Hepatitis viruses											
Hepatitis A virus		1	4	10			1	21		37	274
Hepatitis D virus				1						1	5
Arboviruses											
Barmah Forest virus				1					2	3	129
Dengue not typed									1	1	38
Flavivirus (unspecified)				1						1	15
Ross River virus		2	6	24			2	5	15	54	1,176
Adenoviruses											
Adenovirus not typed/pending		16	4	2			31	58		111	825
Adenovirus type 2							2			2	13
Adenovirus type 3							1			1	28
Adenovirus type 4							4			4	12
Adenovirus type 7							1			1	2
Adenovirus type 40									17	17	54
Herpes viruses											
Cytomegalovirus		20		14		1	27	19		81	780
Epstein-Barr virus		3	1	67			11	36		118	1,623
Herpes virus type 6								2		2	7
Varicella-zoster virus		7	2	39	2	2	46	49		147	1,117
Other DNA viruses											
Molluscum contagiosum								3		3	11
Papovavirus group								2		2	10
Parvovirus		2		10	2	2	26	15		57	303
Picornavirus family											
Coxsackievirus A16							3			3	12
Echovirus not typed/pending							1			1	1
Echovirus type 9		8								8	43
Echovirus type 11		4	1							5	95
Enterovirus not typed/pending		1	1	1	1		5	89		98	560
Enterovirus type 71 (BCR)							3			3	9
Poliovirus type 1 (uncharacterised)		4								4	18
Rhinovirus (all types)		15					4	19		38	291
Ortho/paramyxoviruses											
Influenza A virus		85	6	46			169	157		463	1,120
Influenza A virus H3N2							5			5	23
Influenza B virus		8	1	10			6	18		43	128
Parainfluenza virus type 1		3					2	1		6	32
Parainfluenza virus type 2		3					13	10		26	92
Parainfluenza virus type 3		3		4			7	108		122	479
Paramyxovirus (unspecified)							4			4	4
Respiratory syncytial virus		275		52		4	182	177		690	1,765

Table 3. Virology and serology laboratory reports by State or Territory¹ for the reporting period 15 July to 11 August, and total reports for the year (continued)

	State or Territory ¹								Total this period	Total reported in 1999 ^{2,3,4}
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Other RNA viruses										
HTLV-1								1	1	10
Norwalk agent							7		7	57
Rotavirus		244	1		1	5	50	90	391	1,106
Other										
<i>Chlamydia psittaci</i>							8	3	11	70
<i>Chlamydia trachomatis</i> not typed		26	23	112			7	133	301	2,070
<i>Chlamydia</i> species		2		1					3	14
<i>Coxiella burnetii</i> (Q fever)		1		16			2	2	21	127
<i>Mycoplasma pneumoniae</i>		6	1	23			37	12	79	772
<i>Rickettsia australis</i>							1		1	3
<i>Rickettsia tsutsugamushi</i>							2		2	2
<i>Rickettsia</i> spp - other								3	3	8
<i>Streptococcus</i> group A			4	23					27	98
<i>Bordetella pertussis</i>		2		51			22	6	81	472
<i>Brucella</i> species				1					1	3
<i>Legionella longbeachae</i>								1	1	29
<i>Yersinia enterocolitica</i>				1					1	10
<i>Leptospira</i> species				8				1	9	2
<i>Treponema pallidum</i>		9	45	23				4	86	201
Total	0	751	100	563	6	16	704	1,082	3,222	16,411

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. In 1999, data from the Institute of Clinical Pathology & Clinical Research, Westmead were under reported up to September.
3. Totals comprise data from all laboratories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
4. A new computer system for processing the virology and serology laboratory reporting scheme data, was implemented in September 1999. Consequently, cumulative figures may have decreased due to better duplicate processing.

Table 4. Virology and serology laboratory reports by contributing laboratories for the reporting period 15 July to 11 August 1999

State or Territory	Laboratory	Reports
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	100
	New Children's Hospital, Westmead	264
	Royal Prince Alfred Hospital, Camperdown	103
	South West Area Pathology Service, Liverpool	264
Queensland	Queensland Medical Laboratory, West End	648
	Townsville General Hospital	22
Tasmania	Northern Tasmanian Pathology Service, Launceston	14
Victoria	Royal Children's Hospital, Melbourne	380
	Victorian Infectious Diseases Reference Laboratory, Fairfield	325
Western Australia	PathCentre Virology, Perth	758
	Princess Margaret Hospital, Perth	344
Total		3,222

Table 5. Australian Sentinel Practice Research Network reports, weeks 29 to 32, 1999

Week number	29		30		31		32	
Week ending on	25 July 1999		1 August 1999		8 August 1999		15 August 1999	
Doctors reporting	55		53		57		53	
Total encounters	7,071		7,222		7,933		7,004	
Condition	Rate per 1,000							
	Reports	encounters	Reports	encounters	Reports	encounters	Reports	encounters
Influenza	109	15.4	97	13.4	116	14.6	115	16.4
Rubella	1	0.1	1	0.1	0	0.0	0	0.0
Measles	0	0.0	0	0.0	0	0.0	1	0.1
Chickenpox	19	2.7	9	1.2	17	2.1	14	2.0
New diagnosis of asthma	12	1.7	9	1.2	13	1.6	21	3.0
Post operative wound sepsis	7	1.0	6	0.8	7	0.9	8	1.1
Gastroenteritis	50	7.1	49	6.8	53	6.7	55	7.9

The 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 1999;23:55.

LabVISE is a sentinel reporting scheme. 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 1999;23:58.

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 in 1999. CDI reports the consultation rates for seven of these. For further information, including case definitions, see CDI 1999;23:55-56.

Additional Reports

National Influenza Surveillance, 1999

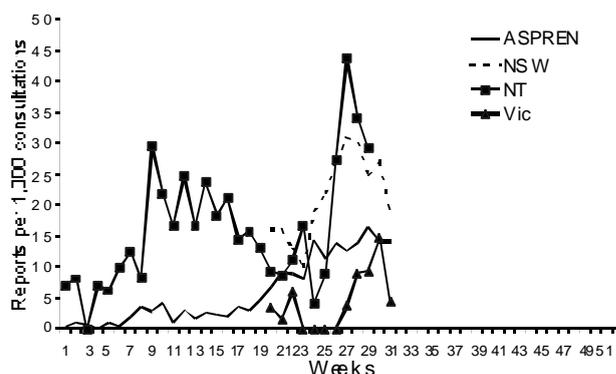
Three types of data are included in National Influenza Surveillance, 1999. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network, Department of Human Services (Victoria), Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health (Northern Territory); laboratory surveillance data from the Communicable Diseases Intelligence Virology and Serology Laboratory Reporting Scheme, LabVISE, and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. For further information about these schemes, see CDI 1999; 23:56.

Sentinel general practitioner surveillance

Over the last 4 week reporting period up until 11 August 1999, a peak in the rate of reports of influenza consultations occurred in all sentinel reporting schemes. This peak was a second peak of influenza consultations for the surveillance schemes in Victoria and the Northern Territory. The Tropical Influenza Surveillance Program

(NT) (45/1000) and NSW Sentinel Scheme (31/1000) reported the highest rates. These occurred in early to mid

Figure 2 Sentinel general practitioner influenza consultation rates, 1999, by scheme



August. The NSW peak rate was similar to the peak rate (42.9/1000) reported in the NSW Sentinel Surveillance Scheme in early August 1998.

Laboratory surveillance

For the year to date, a total of 1,130 laboratory reports of influenza have been received. Of these, 1,012 (90%) were influenza A and 118 (10%) were influenza B (Figure 3). The number of influenza A reports to date is less than the previously recorded high noted in 1998 (Figure 4). As the rates of clinical reporting through the sentinel surveillance schemes has not increased the laboratory figures represent a decrease in laboratory testing.

Figure 3. Laboratory reports of influenza, 1999, by type and by week of specimen collection

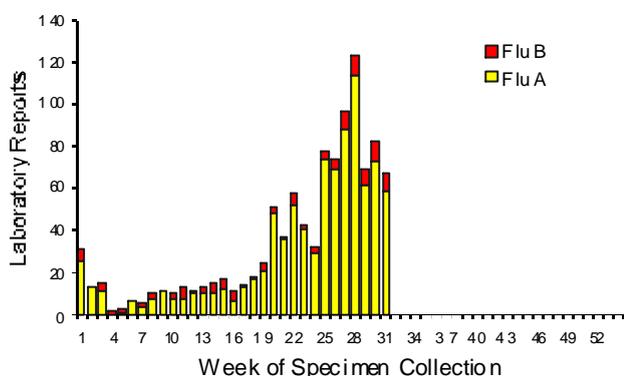
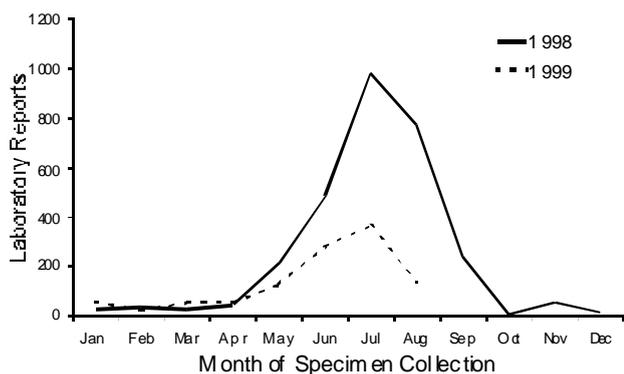


Figure 4 Laboratory reports of influenza, 1999, by month of specimen collection

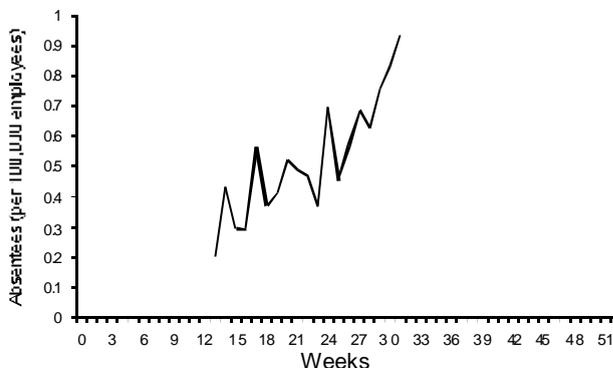


Absenteeism surveillance

The average rates for the last 4 week reporting period were 0.86% and the maximum rate was 0.95%. These rates represent a marked increase compared to a similar

period in 1998 in which the maximum reported rate was 0.29%. This reflects an ongoing trend noted in the previous report. These rates were greater than the previously reported period in May 1999 of 0.45% (Figure 5).

Figure 5. Absenteeism rates in Australia Post, 1999



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, and annually in HIV/AIDS and related diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648; Facsimile: (02) 9332 1837; <http://www.med.unsw.edu.au/ncheccr>.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 30 April 1999, as reported to 31 July 1999, are included in this issue of CDI (Tables 6 and 7).

Table 6. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 30 April 1999, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998
HIV diagnoses	Female	0	1	0	3	0	0	1	0	5	6	23	26
	Male	0	21	1	11	2	0	14	4	53	56	191	240
	Sex not reported	0	0	0	0	0	0	0	0	0	2	1	4
	Total ¹	0	22	1	14	2	0	15	4	58	64	215	270
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	2	3	5
	Male	0	5	1	0	1	0	1	0	8	23	29	95
	Total ¹	0	5	1	0	1	0	1	0	8	25	32	100
AIDS deaths	Female	1	0	0	0	0	0	0	0	1	0	1	2
	Male	1	1	0	0	1	0	0	0	3	11	32	46
	Total ¹	2	1	0	0	1	0	0	0	4	11	34	48

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

Table 7. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 April 1999, by sex and State or Territory

		State or Territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	23	587	8	137	57	5	203	108	1,128
	Male	188	10,589	106	1,899	654	77	3,790	882	18,185
	Sex not reported	0	258	0	0	0	0	25	0	283
	Total ¹	211	11,433	113	2,029	709	82	4,016	988	19,581
AIDS diagnoses	Female	8	173	0	46	21	3	67	26	344
	Male	86	4,534	35	792	328	44	1,591	344	7,754
	Total ¹	94	4,719	35	840	349	47	1,665	372	8,121
AIDS deaths	Female	3	113	0	30	15	2	47	16	226
	Male	65	3,129	24	556	226	28	1,248	245	5,521
	Total ¹	68	3,250	24	588	241	30	1,301	262	5,764

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

Serious Adverse Events Following Vaccination Surveillance Scheme

The Serious Adverse Events Following Vaccination Surveillance Scheme is a national surveillance scheme which monitors the serious adverse events that occur rarely following vaccination. More details of the scheme were published in *CDI* 1999;23;58.

Acceptance of a report does not imply a causal relationship between administration of the vaccine and the medical outcome, or that the report has been verified as to the accuracy of its contents.

It is estimated that 250,000 doses of vaccines are administered every month to Australian children under the age of six years.

Results for the reporting period 1 May to 31 August 1999.

There were 55 reports of serious adverse events following vaccination for this reporting period (Table 8). Onset dates were from 1996 to 1999, the majority (80%) being in 1999. Reports were received from the Australian Capital Territory (2), New South Wales (5), the Northern Territory (8), Queensland (19), South Australia (8), Victoria (8) and Western Australia (5) for this period.

The most frequently reported events following vaccination were other reactions (16 cases, 29%) followed by persistent screaming (14 cases, 26%), convulsions (12 cases, 22%), hypotonic/hyporesponsive episodes (6 cases, 11%), temperature of 40.5°C or more (5 cases, 9%). There was one case of acute flaccid paralysis reported and the diagnosis of the child was confirmed as Guillain-Barré Syndrome based on nerve conduction tests. The child had recovered 3 weeks after onset of symptoms. One death within 30 days of immunisation was reported from Victoria. The baby was 3 months old, and the cause of death was determined to be sudden infant death syndrome (SIDS) by the coroner.

Thirty-seven (67%) cases were associated with Diphtheria-Tetanus-Pertussis (DTP) vaccine, either alone or in combination with other vaccines. The number of adverse events reported during this period were lower than reported in the previous 2 years. One possible explanation could be the introduction of acellular pertussis vaccine. This could have resulted in the decrease in the persistent screaming reaction which used to be reported predominantly with the whole cell pertussis vaccine.

Seventeen of the 55 cases were hospitalised, of which 16 had recovered at the time of reporting. There was incomplete information on the recovery status of 6 cases, while all the other cases had recovered at the time of reporting.

Table 8. Adverse events following vaccination reported in the period 1 May to 31 August 1999¹

Event	Vaccines											Reporting States or Territories	Total reports for this period
	DTP	DTP, Hib	DTP, OPV, Hib	DTP, OPV, HEB	DTP, OPV	OPV, Other	MMR	Hib, OPV	Hib	Hep B	Other ²		
Persistent screaming	3	1	8	1				1				ACT, NSW, NT, Qld, Vic, WA	14
Hypotonic/hyporesponsive episode		1	2			1	1					Qld, Vic	5
Temperature of 40.5°C or more	1	1	2		1							ACT, NT, WA	5
Convulsions	4	2	1				4			1		NSW, Qld, SA, Vic, WA	12
Acute flaccid paralysis ³					1							NSW	1
Death			1									Vic	1
Other	3		3			1			1	2	6	NT, Qld, SA, Vic	16
TOTAL	11	5	17	1	2	2	5	1	1	3	6		54⁴

1. Events with onset dates from 1996 to 1999 were reported in this period.

2. Includes influenza vaccination, DTPa, CDT, OPV, Hepatitis B vaccine, pneumococcal vaccination, BCG, ADT and rabies immunoglobulin (HRIG)

3. This was a case of Guillain-Barré Syndrome

4. 1 child with an adverse event had no vaccine specified.

Bulletin Board

The Public Health Association of Australia Inc.

31st Annual Conference
26-29 September 1999
Carlton Hotel
Darwin, Northern Territory
Details: PO Box 319
Curtin ACT 2605
Email: conference@pha.org.au

The Queensland Institute of Medical Research

Symposium on Q Fever
13-14 October 1999
Brisbane, Queensland
Phone: 07 3844 1138
Fax: 07 3844 0909
Email: qfever@icms.com.au

Institute of Nanotechnology

The Surgery Room of the 21st Century
1-2 November 1999
The Diagnostic Centre of the 21st Century
3-4 November 1999
Glasgow, Scotland
Phone: 44 1786 447520
Fax: 44 1786 447530
Email: julie@nano.org.uk

The theme of the conference is taken from the Royal Academy of Engineering's publication *Medical Engineering - A Field With Potential*. The keynote address: *Lab-on-a-Chip Technologies - The Future* will be given by Professor Andreas Manz, Imperial College of Science, Technology and Medicine.

Some of the topics are of interest to communicable diseases. For details see contacts above.

Australasian Society for HIV Medicine Inc

11th Annual Conference
9-11 December 1999
Perth, Western Australia
Contact: ASHM Conference Secretariat
C/- ICMS Australasia Pty Ltd, GPO Box 2609,
Sydney, NSW, 2001
Phone: 02 9241 1478
Fax: 02 9251 3552

Advance notice

The First Pacific Rim Biomedical Seminar

Transportation of Infectious and Diagnostic Substances
3 March 2000
Sheraton on the Park
Sydney, NSW
Contact: Christine Sherwood
Phone: 1800 023 560; or
Sydney: 9693 2988
Email: sherwood@worldcourier.com.au

International Society of Travel Medicine/WHO/CDC

2nd European Conference of Travel Medicine
29-31 March 2000
Venice, Italy
Contact: Dr Walter Pasini, Italy
Phone: 390-541-24301
Fax: 390-541-25748
Email: wpasini@rimini.com

Australian Society for Infectious Diseases Meeting

April 16-19, 2000
Fairmont Resort Leura
Organisers: Dart Associates:
Phone: 02 94189396
For scientific content: Contact Tom Gottlieb,
Concord Hospital
Phone: 02-97677533
Fax: 02-97677868 or
Email: Tom@micr.crg.cs.nsw.gov.au

Australian Infection Control Association

First Biennial Conference
Infection Control Beyond 2000
3-5 May 2000
Hilton Adelaide International, South Australia
Contact: AICA 2000 Secretariat
PO Box 1280, Milton, Queensland 4064
Phone: 07 3369 0477
Fax: 07 3369 1512
Email: aica2000@im.com.au
Website: <http://www.aica.org.au/aica2000.htm>

Australian School of Environmental Studies

Arbovirus Research in Australia
3-7 July 2000
Couran Cove Nature Resort, Gold Coast, Queensland
Contact Dr Michael Brown, Queensland Institute of
Medical Research, PO Box Royal Brisbane Hospital,
Herston, Queensland, 4029
Website: <http://www.mcaa.org.au>

Royal North Shore Hospital

Outpatient Parenteral Therapy - beyond 2000
17-22 September 2000
Fairmont Resort
Leura, New South Wales
Phone: 02 9956 8333
Fax: 02 0056 5154
Email: confact@conferenceaction.com.au

The Australasian Society for HIV Medicine

12th Annual Conference
16-19 November 2000
The Carlton Crest, Melbourne, Victoria
Phone: 02 9382 1656
Fax: 02 9382 3699
Email: B.Pearlman@unsw.edu.au

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

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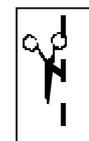


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Notice

The Australian Sentinel Practices Research Network (ASPREN) is considering the conditions to be recorded during 2000. If there are any researchers, epidemiologists or other readers of *CDI* who would like specific conditions to be recorded, could they please contact:

Dr Ian Wilson
Research & Health Promotion Unit
215 Payneham Road
ST PETERS SA 5069

There may be a small cost associated depending on the data to be recorded. There are limitations on the data that can be collected, in particular in relation to the distribution of recorders and the denominator used.

Proposals need to be submitted to the above address by Friday 8 October 1999.

Please direct any enquiries to Ian Wilson. He can be contacted by phone on 0419 801 787.

Overseas briefs

Source: World Health Organization (WHO)
This material has been condensed from information on the WHO Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the CDI homepage.

Suspected haemorrhagic fever, Germany

On 5 August, the Robert Koch Institute in Berlin provided WHO with more details on the previously reported case of a man hospitalised with suspected haemorrhagic fever, after returning from a trip to Côte d'Ivoire. He left Germany on 17 July for Abidjan, then travelled to Bouaké to spend 2 weeks at a scientific research camp located in the Komoe National Park. He returned to Germany on 1 August.

The patient did not show any symptoms until he reached his home, so the Institute has concluded that the risk of person-to-person transmission to other passengers is minimal. All those who are known to have had direct contact with him (either during the trip or after his arrival in Berlin) are under surveillance by the health authorities.

A range of tests were conducted to identify the infectious agent. Tests for the following were negative: Ebola, Hantavirus infection, Lassa fever, Marburg and malaria. The patient died on 6 August. The diagnosis of yellow fever by culture and PCR was confirmed today.

Haemorrhagic fever with renal syndrome, Kosovo

A case of haemorrhagic fever with renal syndrome (HFRS) has been confirmed in Kosovo. The case was in a 19 year old woman who lived in a mountainous, forested area near the Albanian border. The diagnosis, suspected on clinical grounds, has been supported by laboratory tests, which confirm hantavirus infection. The tests were performed by the WHO Collaborating Centre for Arboviruses and Haemorrhagic Fevers in Thessaloniki, Greece.

Polio in Afghanistan

An outbreak of poliomyelitis has been reported from Kunduz province in Northern Afghanistan. Since early May, a total of 26 cases of children with paralysis have been reported, of which 6 cases have already been confirmed as poliomyelitis through laboratory analysis. Fifteen of the 26 cases were reported from Kunduz town itself, with 11 cases from the districts surrounding Kunduz. The outbreak was identified only because special disease reporting for suspected polio cases, including the capacity for laboratory confirmation, was established in May 1999 in the north as part of the nation-wide initiative to eradicate polio.

Since all immunisation activities in Northern Afghanistan had nearly ceased in mid-1997 and are just now being re-established, the outbreak of poliomyelitis is not unexpected. To determine the full extent of the outbreak, all health facilities and NGOs providing health care in the north have been alerted to the outbreak and requested to report all suspected cases to the Ministry of Public Health. A large scale house-to-house immunisation campaign,

targeting more than 130,000 children aged less than 5 years has been launched this week in the outbreak area as a collaborative effort.

Poliomyelitis is endemic in Afghanistan and the best way to prevent the disease is to immunise children with at least 3 doses of polio vaccine during their first year of life. The global strategy to eradicate polio includes supplementary country-wide campaigns called National Immunisation Days (NIDs) when 2 drops of oral polio vaccine are given to all children under age 5 years, in 2 consecutive months. Country-wide NIDs were held in Afghanistan in May and June 1999 and are scheduled again for October and November. Unfortunately the outbreak in Kunduz started before the May NIDs.

Cholera

Madagascar

The cholera outbreak which began in late March has stabilised in the two provinces affected, and the number of reported cases has declined considerably during recent weeks. In Antananarivo province, the situation has been less acute than in Mahajanga province. There is a risk that some districts in Mahajanga province (Mahajanga I and II, Marovoay and Mitsinjo) may become endemic. Some cases have also been reported recently in Antsiranana province, Nosy Be district.

Strict control measures were implemented during the early stages of the outbreak, including: care of suspect cases in health centres and hospitals, and treatment of contacts,

disinfection and other hygiene measures in treatment centres and patients' homes, widespread campaigns to educate and inform the public, and epidemiological surveillance.

Notification of cases to WHO and information reports on the areas affected have been very regular and in conformity with the requirements of the International Health Regulations. There is no reason for travellers/tourists to postpone travel to Madagascar as the risk of infection is negligible if basic hygiene measures are taken.

Niger

An outbreak has occurred in Boboyé District, Dosso Department, which is approximately 100 km east of Niamey. The first cases were reported in June, and as of 30 July the total number was 169 cases with 10 deaths. Thirty-six villages were affected. Control measures which were taken at the early stages of the outbreak have helped to decrease the number of cases and since 28 July only 1 or 2 cases have been occurring daily. Various materials and medicines were supplied by UNICEF, WHO and "Italian Cooperation" both in the districts affected and in other regions in case of spread.

Cholera outbreaks occur in Niger fairly regularly although no cases were reported in 1998. A total of 259 cases with 13 deaths was reported in 1997 and a larger outbreak with 3,957 cases and 206 deaths in 1996.

Editor: Eddie O'Brien

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Subscriptions

Deputy Editor, *CDI*, MDP 6, Department of Health and Aged Care, GPO Box 9848, Canberra ACT 2601
Fax (02) 6289 7735

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Contributions

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