Report of the Australian Malaria Register for 1992 and 1993

Joan Bryan, Elekana Fa'afoi & Simon Forsyth,
Australian Centre for International & Tropical Health & Nutrition, The University of Queensland, Mayne Medical School, Herston Road, Herston Qld 4006

Abstract

Australia is free from endemic malaria but several hundred imported cases occur each year. Notification and screening data on malaria cases are collected by State and Territory health authorities and laboratories and forwarded to the Australian Malaria Register (AMR) for national collation and analysis. This report provides information on 758 malaria cases with 5 deaths reported in Australia in 1992 and 712 cases with 1 death in 1993. In both years, just over 70% of cases were male and the modal age group was 20 to 29 years. Cases were reported from all States and Territories, with Queensland reporting the greatest number of cases in both years. The predominant species was *Plasmodium vivax*, although *P. falciparum* accounted for just over a quarter of the cases each year. Papua New Guinea (PNG) was the most common source of cases in both years, reflecting the number of people who move between Australia and PNG and the high endemicity of malaria in PNG. The incidence of malaria was also high in travellers from the Solomon Islands in both years and from Ghana in 1992 and Nigeria in 1993. The six deaths over two years highlight the need for medical practitioners to consider malaria as a diagnosis in patients with a history of travel to malarious countries and to provide appropriate advice on malaria prophylaxis to intending travellers. *Commun Dis Intell* 1998;22:237-244

Introduction

Malaria is still a major health problem in tropical areas of the world. Although free from endemic malaria, Australia imports several hundred cases of malaria from these countries each year. The Australian Malaria Register collates case data nationally and has previously published reports for the years 1990 and 1991.[1,2,3,4] This paper reports on malaria cases in Australia in 1992 and 1993.

Materials and Methods

This report is based on information supplied by the State and Territory health authority malaria registers, screening programs and laboratories to the Australian Malaria Register which is managed by the Tropical Health Program of the Australian

ISSN 0725-3141
Volume 22
Number 11
29 October 1998

Contents

Cont'd next page

Contents, *continued*

Centre for International and Tropical Health and Nutrition.

The data were entered and managed using EpiInfo Version 6, in accordance with the coding and editing rules described in Appendix 1 of Sleigh et al. [3] Duplicate entries, which occurred due to errors of entry or to the same episode being reported through both a private practitioner and a hospital, were identified and eliminated where possible.

Cases with clinical onset or, in the absence of clinical data, with a first laboratory report in 1992 and 1993 were included in the analysis. The definition and classification of cases as new or relapsed and imported or introduced were the same as those used previously. [1,2,3,4] Relapse was defined as occurring when the same species of parasite was identified from a patient more than 28 days from the onset of a primary attack. Reports of episodes involving the same parasite and occurring within 28 days in the one individual were counted as a single episode. Cases were classified as imported when infection was acquired outside Australia.

In many cases occupation was not recorded, but the person was stated to have entered Australia for education. Such persons were classified as students and those under 15 years of age as minors. For many people no reason was given for their being in the country in which they acquired malaria; however, each year more than 100 of these people acquired malaria in the country in which they were born and the new category 'birth country' was made for such cases.

The incidence of malaria in arrivals from various countries was calculated using all arrivals from the relevant country as the denominator. The unpublished arrivals data was provided by the Australian Bureau of Statistics.

Results

Sex and age distribution

In 1992, 758 cases of malaria were reported, of which 554 (73.1%) occurred in males. In 1993, the total number of cases was 715. Sex was not recorded for 5 of these (2 in the 0-9 age group, 1 in the 10-19 year age group and 2 whose ages were not recorded). Of the remaining cases, 510 (71.8%) were male. (Figures 1 and 2).

Age was not recorded for 18 cases (5 female and 13 male) in 1992 and 22 cases (7 female, 13 male and 2 unknown sex) in 1993. For those cases in which age was recorded, the modal age group was 20 to 29 years in both years (Figures 1 and 2).

Geographic and seasonal distribution

In both years, the greatest number of cases was recorded in Queensland; 338 (44.6%) in 1992 and 294 (41.1%) in 1993. New South Wales accounted for 163 (21.5%) of cases in 1992 and 206 (28.8%) in 1993. Victoria had 130 cases (17.2%) in 1992 and 90 (12.6%) in 1993 (Tables 1 and 2).

Cases for which a date of onset of symptoms is recorded occurred fairly evenly throughout the year with the highest number of cases in February in both years and the lowest in August in 1992 and December in 1993 (Figure 3).

Figure 1. Malaria notifications, Australia, 1992, by age and sex.

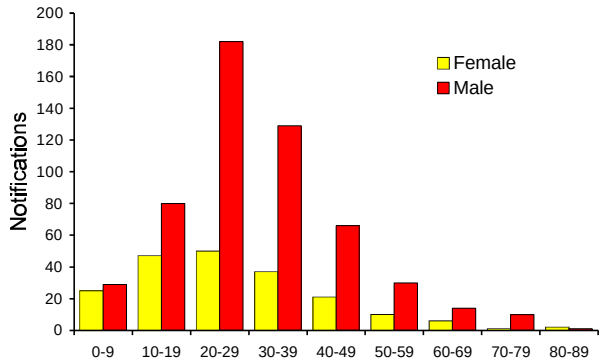


Figure 2. Malaria notifications, Australia, 1993, by age and sex.

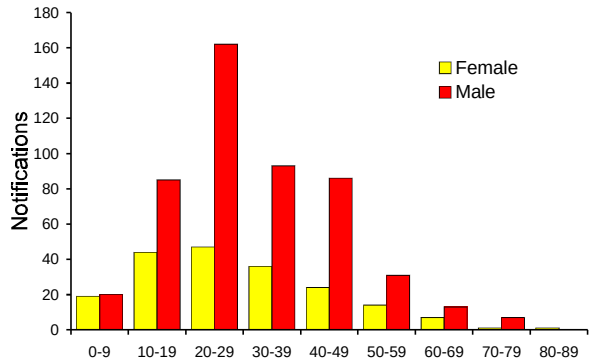
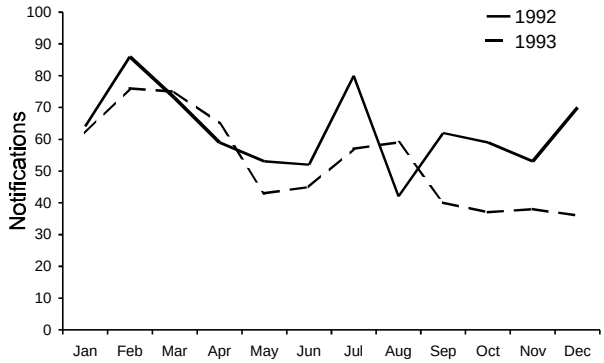


Figure 3. Malaria notifications, Australia, 1992 and 1993, by month of onset.



Species of malaria parasite

In 1992, the species was unknown for 7 of the cases. Of the remaining 751 cases, 214 (28.5%) were *Plasmodium falciparum* only, 512 (68.2%) were *P. vivax* and 10 (1.3%) were mixed infections of both species. *P. malariae* and *P.*

Table 1. Malaria notifications, Australia, 1992, by State or Territory of residence and species of *Plasmodium*.

State / Territory	*Plasmodium* species							
	falciparum	*vivax*	*falciparum/ vivax*	*malariae*	*ovale*	Unknown	Total	Percent
ACT	6	20	0	0	0	0	26	3.4
NSW	29	124	0	3	5	2	163	21.5
NT	8	14	0	0	0	0	22	2.9
Qld	111	219	4	3	0	1	338	44.6
SA	10	21	0	0	0	1	32	4.2
Tas	0	3	0	0	0	0	3	0.4
Vic	40	80	5	1	1	3	130	17.2
WA	10	31	1	0	2	0	44	5.8
Total	214	512	10	7	8	7	758	100.0

ovale were both relatively rare, together accounting for less than 2% of cases (Table 1).

In 1993, species was unknown for 2 cases. Of the remaining 713, there were 184 cases (25.8%) of *P. falciparum* alone, 497 cases (70.0%) of *P. vivax* alone and 9 (1.3%) cases were infected with both species. Again, *P. ovale* and *P. malariae* were rare (Table 2).

Table 2. Malaria notifications, Australia, 1993, by State or Territory of residence and species of *Plasmodium*.

State / Territory	*Plasmodium* species								
	falciparum	*vivax*	*falciparum/ vivax*	*malariae*	*ovale*	*ovale/ falciparum*	Unknown	Total	Percent
ACT	8	13	0	1	1	0	0	23	3.2
NSW	36	159	1	4	6	0	0	206	28.8
NT	11	22	0	0	0	0	0	33	4.6
Qld	101	185	3	3	2	0	0	294	41.2
SA	5	18	2	0	0	0	0	25	3.5
Tas	3	3	0	0	0	0	0	6	0.8
Vic	18	65	0	1	2	1	2	90	12.6
WA	2	32	3	0	0	1	0	38	5.3
Total	184	497	9	9	11	2	2	715	100.0

Accuracy of diagnosis

P. falciparum is the only species with a high case fatality rate. Because of differing patterns of resistance to drugs between the malaria species, case management is dependent on correct species identification. In 1992, 643 slides were re-read at a reference laboratory and parasite identification differed in 63 cases, including 22 cases in which the parasite species was originally recorded as unknown. Fifteen cases of *P. falciparum* were incorrectly classified as *P. vivax* and one as *P. malariae*. In the 640 slides examined at a reference laboratory in 1993, 46 differences occurred, including seven slides of *P. falciparum* which were initially diagnosed as *P. vivax*. No fatal cases were amongst those misdiagnosed.

Onset of illness

In 1992, 18 cases of parasitaemia were recorded as a result of screening people without symptoms. The infection of one of these was acquired in India, and Papua New Guinea (PNG) was the origin of the infection of all other symptomless cases. Of the 650 clinical cases, 518 (79.7%) became ill in Australia, 113 (17.4%) became ill overseas and no data were given for 19 (2.9%) cases.

In 1993, the country of acquisition of all 18 cases diagnosed on screening was PNG. The proportion who became ill in Australia and overseas was very similar to the previous year with an Australian onset for 482 of the 605 clinical cases (79.7%), an overseas onset for 114 (18.8%) and no data for 9 (14.9%) cases.

Delay in diagnosis

Data on onset date and date of diagnosis were available for 679 cases (89.6%) in 1992 and 630 cases (88.5%) in 1993. The number of cases diagnosed on the day of symptom onset was 97 in 1992 and 129 in 1993, respectively 14.3% and 25.7% of the cases for which this data were available.

In 1992, over 50% of cases were diagnosed within 3 days of the onset of symptoms. Diagnosis was on average slightly quicker if symptoms began in Australia. In cases with an Australian onset, 56.6% of cases being diagnosed by day 3, whereas 50% of cases in which symptoms started overseas were not diagnosed until the fifth day of illness. In 1993, overall 54.6% were diagnosed by day 3 after onset of symptoms. This included 55.3% of cases with an Australian onset and 51.4% of cases in which the person became ill overseas. The numbers of undiagnosed cases remaining each day after the onset of symptoms are shown in Figures 4 and 5.

Figure 4. Malaria notifications, Australia, 1992, number of cases remaining undiagnosed each day after onset of symptoms.

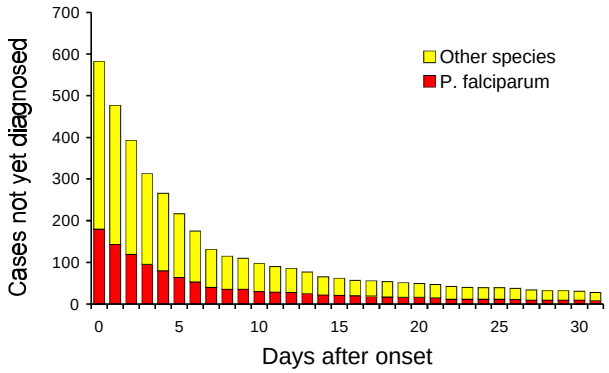
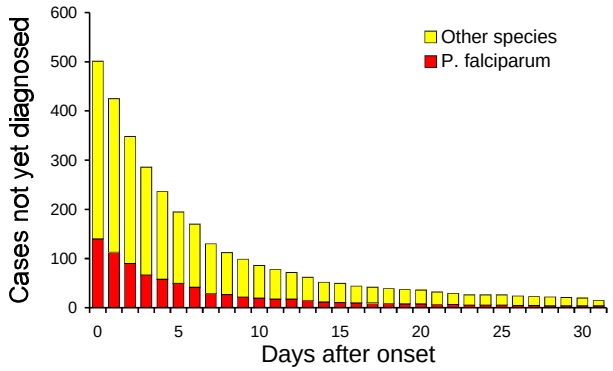


Figure 5. Malaria notifications, Australia, 1993, number of cases remaining undiagnosed each day after onset of symptoms.



Deaths

Five deaths were recorded in 1992, all in Australian residents. Two cases were acquired in PNG, one in the Solomon Islands and one in Nigeria. One case was acquired in Australia, as a result of a blood transfusion. *P. falciparum* was the parasite in all fatal cases. All cases became ill in Australia and the delay between onset of symptoms and diagnosis was 3 days, 5 days, 6 days (2 cases) and 7 days respectively.

The one death recorded in 1993 occurred in a 46 year old male who had a *P. vivax* infection acquired in Indonesia. The species was confirmed in a central reference laboratory. There was a two week diagnostic delay in this case, but even with such delays *P. vivax* infections do not normally cause fatalities. It is possible that this was an infection with both *P. falciparum* and *P. vivax* but with few *P. falciparum* infected red blood cells in the peripheral blood stream. No information was available on whether the patient had any underlying medical condition.

Case classification and origin of cases

In 1992, two cases were acquired in Australia. One was from a blood transfusion and the other was in a Torres Strait Island resident who was diagnosed on Thursday Island. In 1993, no cases were acquired in Australia. In 1992, 26 cases were classified as relapsing and in 1993, there were 39. Origin was unknown in 45 cases in 1992 and 51 cases in 1993. All other cases (90.6% in 1992 and 87.4% in 1993) were classified as imported (Table 3).

Table 3. Case classifications for malaria notifications, Australia, 1992 and 1993.

Case classification	1992		1993	
	Frequency	Percent	Frequency	Percent
Imported	686	90.5	625	87.4
Acquired in Australia	2	0.3	0	0.0
Relapsing	26	3.4	39	5.5
Unknown	44	5.8	51	7.1
Total	758	100.0	715	100.0

PNG was the most common source of cases in both years, 361 cases in 1992 and 323 cases in 1993. Other important source countries were: the Solomon Islands (86 cases in 1992 and 74 cases in 1993), Vanuatu (18 in 1992 and 30 in 1993), Indonesia (86 cases in 1992 and 67 in 1993), Thailand (11 cases in 1992 and 17 in 1993), India (50 cases in 1992 and 58 in 1993). Vietnam was the source of 17 cases in 1992 but only 2 in 1993. Ghana, Nigeria, Kenya and Pakistan also contributed 5 to 11 cases annually. Fiji, a malaria-free country without malaria vectors was reported as the country of acquisition for one case of malaria in 1992. This case was not followed up. The countries in which malaria was acquired, and the species involved, are shown in Tables 4 and 5.

The incidence of malaria in arrivals from selected countries is presented in Table 6. As in previous years, the incidence was high for the Solomon Islands (11.4/1000 and 11.1/1000). However, this figure was exceeded by Ghana in 1992 (14.0/1000) and Nigeria in both 1992 (11.6/1000) and 1993 (12.7/1000). As fewer than a thousand travellers entered Australia annually from either of these countries, the absolute numbers of malaria cases acquired there were small. The incidence in travellers from PNG was lower than in 1991, and this was responsible for the reduction in the total number of cases in both 1992 and 1993 compared to 1991 when 939 cases were reported. [3.4]

The species of malaria differed according to region in which the parasites were acquired (Tables 7 and 8). In 1992, 72.7% of cases from Africa were due to *P. falciparum* but this percentage dropped to 52.4% in 1993. The proportion of cases due to *P. falciparum* was lower and relatively stable in other regions being about 28% for the SW Pacific 13% to 18% in SE Asia and less than 10% in South Asia.

Occupation and reason for travel

The occupation of almost half of the cases was not provided (Table 9). Amongst the rest, about 40% were students. The most common reason for presence in the malarious country in which infection occurred was 'holiday', accounting for 237 cases in 1992 and 190 cases in 1993 (Table 9).

Table 4. Malaria notifications, Australia, 1992, by country in which malaria was acquired and species of *Plasmodium*.

	Plasmodium species						
Country	*falciparum*	*vivax*	*falciparum/ vivax*	*malariae*	*ovale*	Unknown	Total
Angola	0	0	0	0	1	0	1
Australia	1	1	0	0	0	0	2
Benin	1	0	0	0	0	0	1
Brazil	1	0	0	0	0	0	1
Cameroon	1	0	0	0	0	0	1
China	1	0	0	0	0	0	1
East Timor	0	1	0	0	0	0	1
Egypt	1	0	0	0	0	0	1
Fiji[1]	0	1	0	0	0	0	1
Ghana	6	2	0	0	1	0	9
India	5	44	1	0	0	0	50
Indonesia	17	67	0	1	0	1	86
Kenya	8	1	0	0	2	0	11
Malawi	2	0	0	0	0	0	2
Mexico	1	0	0	0	0	0	1
Myanmar	0	4	0	0	0	0	4
Nepal	0	1	0	0	0	0	1
Nigeria	5	0	0	1	1	0	7
Pakistan	0	4	0	0	0	1	5
Philippines	0	1	0	0	0	0	1
Papua New Guinea	108	239	6	5	0	3	361
Solomon Islands	21	61	3	0	0	1	86
Sri Lanka	0	3	0	0	0	0	3
Sudan	1	0	0	0	0	0	1
Thailand	4	7	0	0	0	0	11
Uganda	1	0	0	0	0	0	1
Vanuatu	5	13	0	0	0	0	18
Vietnam	0	17	0	0	0	0	17
Zaire	0	1	0	0	0	0	1
Zambia	3	0	0	0	0	0	3
Zimbabwe	2	0	0	0	0	0	2
Other	9	6	0	0	3	0	18
Unknown	0	0	0	0	0	49	49
Total	204	474	10	7	8	55	758

1. Reported source of infection. However, Fiji is a malaria free country with no malaria vectors.

Of the cases for whom a reason for visit was recorded, between 20 and 30% each year acquired their infection in their country of birth. The source of infection was the birth country in 203 cases in 1992 and 123 cases in 1993 (Table 10). In 1992, 50 people who acquired their infection in their country of birth entered Australia for education, as did 43 in 1993.

Malaria in the receptive zone

In Australia, areas north of 19°S are considered receptive to malaria.[5] The number of cases reported by doctors within the receptive zone was 139 in 1992 and 135 in 1993. *P. falciparum* accounted for 47.5% of these cases in 1992 and for 47.5% in 1993. In 1992, 75% of *P. falciparum* cases in the receptive zone had been diagnosed by day 5, whereas seven days elapsed before 75% of *P. falciparum* cases were diagnosed in the non-receptive zones. Diagnosis of *P. falciparum* in the receptive zone was quicker in 1993 with 75% of cases diagnosed by day 4, compared to day 6 in other areas.

Discussion

The six deaths from malaria in two years highlight the need for general practitioners to be aware of the possibility of malaria in travellers from tropical countries and regions, particularly PNG, the Solomon Islands and Africa, and to arrange for the appropriate diagnostic tests immediately symptoms develop. Misdiagnosis was not implicated in any of the deaths, but the demonstration that 21 cases of the potentially fatal *P. falciparum* were originally diagnosed as the usually nonfatal *P. vivax* indicates that misidentification can occur. When treating patients with malaria, medical

Table 5. Malaria notifications, Australia, 1993, by country in which malaria was acquired and species of *Plasmodium*.

	Plasmodium species							
Country	*falciparum*	*vivax*	*falciparum/ vivax*	*malariae*	*ovale*	*ovale/ falciparum*	Unknown	Total
Belize	0	1	0	0	0	0	0	1
Burma	0	3	0	0	0	0	0	3
Cambodia	0	3	0	0	0	0	0	3
Central African Republic	0	0	0	0	1	0	0	1
Equador	0	1	0	0	0	0	0	1
Ethiopia	0	0	1	0	0	0	0	1
Ghana	3	0	0	2	0	1	0	6
India	4	53	1	0	0	0	0	58
Indonesia	10	57	0	0	0	0	0	67
Ivory Coast	0	0	0	0	1	0	0	1
Kenya	3	0	0	0	2	0	0	5
Laos	0	1	0	0	0	0	0	1
Malawi	1	1	0	0	0	0	0	2
Malaysia	0	0	1	0	0	0	0	1
Nigeria	5	0	0	0	0	0	0	5
Pakistan	0	8	0	0	0	0	0	8
Philippines	0	1	0	0	0	0	0	1
Papua New Guinea	104	210	4	3	1	0	1	323
Singapore	0	1	0	0	0	0	0	1
Solomon Islands	14	59	1	0	0	0	0	74
Somalia	1	3	0	0	0	0	0	4
South Africa	0	1	0	0	1	0	0	2
Sri Lanka	0	1	0	0	0	0	0	1
Sudan	1	0	0	0	0	0	0	1
Tanzania	5	0	0	0	0	0	0	5
Thailand	1	16	0	0	0	0	0	17
Timor	1	0	0	0	0	0	0	1
Uganda	2	0	0	0	1	0	0	3
Vanuatu	3	25	1	1	0	0	0	30
Vietnam	0	2	0	0	0	0	0	2
Zaire	0	0	0	1	2	0	0	3
Zambia	2	1	0	0	0	0	1	4
Zimbabwe	3	2	0	0	0	1	0	6
Other	8	6	0	1	2	0	0	17
Unknown	0	0	0	0	0	0	56	56
Total	171	456	9	8	11	2	58	715

practitioners should consider the possibility of misdiagnosis, especially when the blood slide has been examined in laboratories which would seldom see malaria cases. New diagnostic tests which are highly sensitive and specific for *P. falciparum* now enable doctors to establish more accurately whether or not their patient is infected with this species.

Delay in diagnosis has implications both for the individual patient and for public health in the malaria receptive zone. Diagnostic delay of more than three days occurred in five of the six patients who died (83.3%), but for fewer than 50% of malaria cases overall.

Delays in diagnosis and treatment allow time for gametocytes to develop. The gametocytes of *P. falciparum* require about 10-12 days to mature before they can infect mosquitoes whereas those of *P. Vivax* take 2-4 days.[6] The presence of gametocytes in a patient in the malaria receptive zone provides a risk of transmission of malaria within Australia. No secondary cases arose as a result of the 274 cases reported in the receptive zone during 1992 and 1993, however continuing vigilance is needed to ensure that any outbreak can be quickly detected and remedial action taken.

The continuing occurrence of imported malaria cases in Australia is a reminder to doctors of the need to provide patients who are intending to travel to malarious countries with accurate and up to date advice on the risks of malaria in the countries they intend to visit and on the measures

Table 6. Malaria notifications, Australia, 1992 and 1993, incidence of malaria in arrivals from selected countries.

Country of exposure	Arrivals		Cases		Rate/1000	
	1992	1993	1992	1993	1992	1993
Papua New Guinea	76545	77640	361	323	4.7	4.2
Solomon Islands	7545	6641	86	74	11.4	11.1
Vanuatu	25856	24236	18	30	0.7	1.2
Indonesia	230392	270172	86	67	0.4	0.2
Thailand	106361	123262	11	17	0.1	0.1
Vietnam	26479	31475	17	2	0.6	0.1
India	34120	36145	50	58	1.5	1.6
Ghana	645	984	9	6	14.0	6.1
Nigeria	605	393	7	5	11.6	12.7
Kenya	2526	2801	11	5	4.4	1.8
Pakistan	5019	4487	5	8	1.0	1.8

Table 7. Malaria notifications, Australia, 1992, by region of exposure and species of *Plasmodium*.

Region of exposure	*Plasmodium* species						
	falciparum	*vivax*	*falciparum/ vivax*	*malariae*	*ovale*	Unknown	Total
Africa	40	6	0	1	8	0	55
Australia	1	1	0	0	0	0	2
Central America	1	0	0	0	0	0	1
Southeast Asia	22	101	0	1	0	1	125
Southwest Pacific	134	314	9	5	0	4	466
South America	1	0	0	0	0	0	1
South Asia	5	52	1	0	0	1	59
Unknown	0	0	0	0	0	49	49
Total	204	474	10	7	8	55	758

Table 8. Malaria notifications, Australia, 1993, by region of exposure and species of *Plasmodium*.

Region of exposure	*Plasmodium* species								
	falciparum	*vivax*	*falciparum/ vivax*	*malariae*	*ovale*	*ovale/ falciparum*	*vivax/ovale*	Unknown	Total
Africa	31	9	1	4	9	2	1	1	63
Central America	0	1	0	0	0	0	0	0	1
Southeast Asia	6	68	1	0	0	0	0	0	100
SW Pacific	60	226	5	4	1	0	0	1	427
South America	0	1	0	0	0	0	0	0	1
South Asia	3	58	1	0	0	0	0	0	67
Unknown	0	0	0	0	0	0	0	56	56
Total	100	363	8	8	10	2	1	58	715

they can take to protect themselves against this potentially fatal disease.

References

1. Forsyth S, Loeskow K, Pearce M, Riley I, Sleigh A and Srinivasa M. Final report of the Australian Malaria Register for 1990. Tropical Health Program, The University of Queensland. Herston (Brisbane):1991.
2. Forsyth S, Loeskow K, Pearce M, Riley I, Sleigh A and Srinivasa M. Report of the Australian Malaria Register for 1990. *Commun Dis Intell* 1991;15:400-408
3. Sleigh A, Srinivasa M, Cooper A, Forsyth S, and Riley I. Report of the Australian Malaria Register for 1991. Tropical Health Program, The University of Queensland. Herston (Brisbane):1992.
4. Sleigh A, Srinivasa M, Cooper A, Forsyth S, and Riley I. Report of the Australian Malaria Register for 1991. *Commun Dis Intell* 1993;17:134-142

5. Black RH. Malaria in Australia. Commonwealth Department of Health School of Public Health and Tropical Medicine, The University of Sydney, Service Publication No.9. Australian Government Printing Service. Canberra 1972

6. Carter R and Graves PM. Gametocytes in malaria. In Wernsdorfer WH and McGregor I (Eds) Principles and practice of malariology. Churchill Livingstone. Edinburgh 1988

Table 9. Malaria notifications, Australia, 1992 and 1993, by occupation.

Occupation	1992 Number of cases	1992 Percent	1993 Number of cases	1993 Percent
Clerk	3	0.4	9	1.3
Labourer and related worker	29	3.8	20	2.8
Machine operator, driver	15	2.0	7	1.0
Manager, administrative	15	2.0	18	2.5
Minor (years old)	53	7.0	49	6.9
Para-professional	23	3.0	22	3.1
Professional	72	9.5	36	5.0
Salesperson, personal service worker	20	2.6	17	2.4
Student	160	21.1	149	20.8
Tradesperson	30	4.0	31	4.3
Unknown	348	45.9	357	49.9
Total	758	100	715	100.0

Table 10. Malaria notifications, Australia, 1992 and 1993, by reason for presence in country in which malaria exposure occurred.

Reason	1992 Number of cases	1992 Percent	1993 Number of cases	1993 Percent
Business	57	8.3	56	10.0
Business companion	2	0.3	2	0.4
Education	7	1.0	3	0.5
Employment	90	13.1	104	18.5
Holiday	237	34.4	190	33.9
Other	51	7.4	27	4.8
Birth country	203	29.5	123	21.9
Student vacation	5	0.7	9	1.6
Visiting relatives	36	5.2	47	8.4
Total[1]	688	100.0	561	100.0

1. Excludes 70 cases in 1992 and 154 cases in 1993 for whom no data were recorded.

Editorial Comment

Bronwen Harvey

Apart from historical interest, what can a report on the malaria situation in Australia five years ago tell us? Firstly, the report highlights the potentially disastrous consequences of delays in diagnosis of malaria and misdiagnosis of malaria species, issues which are as important now as in 1993.

Secondly, the report serves as a reminder to clinicians, travellers and travel agents that travel to malarious countries carries with it the risk of exposure to infection with malaria. Clinicians need to be able to provide accurate and up to date travel health advice to intending travellers, or to refer them to someone who can (see Box). Travellers may not be aware of the need to seek such advice and travel agents can play an important role in educating travellers and ensuring that they seek advice several weeks before the date of travel. A travel history should become a routine part of clinical practice and anyone who has recently returned from a malarious country and presents with symptoms suggestive of malaria should be immediately tested for the infection, preferably through a pathology service which has experience in malaria diagnosis.

Thirdly, the report provides an opportunity to remind clinicians and laboratories of the importance of ensuring that all cases are notified to the relevant State or Territory health authority. Prompt notification enables public health authorities to establish the origin of each patient's infection and ensure the early identification of any cases acquired in Australia. In the malaria receptive area of Australia, prompt investigation of cases enables public health action to prevent local transmission of the disease.

Finally, the report provides an opportunity to consider the current status of national malaria surveillance in Australia and to look at possible future directions.

Malaria is one of the communicable diseases for which data are collected by State and Territory health authorities under their public health legislations. Notification data for a number of communicable diseases, including malaria, have been nationally collated since 1917.[1] Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided the framework for the continuation of this national collation. Malaria data are published regularly in the surveillance section of *CDI* and included in the annual reports of the NNDSS.

Data on only a small number of variables are collected within the NNDSS: age, sex, state and postcode of residence, aboriginality and onset and report dates. Since 1995, data on the *Plasmodium* species have also been

collected. Although there is a level of under-reporting of malaria to the NNDSS,[3] the data allow general trends in the incidence of malaria to be monitored (see report below). However, the absence of data on the origin of infection limits its usefulness for documenting Australia's continuing freedom from endemic disease.

To collate more extensive information than available through routine notifiable disease surveillance, a Central Register of Malaria Cases was established as a Commonwealth Department of Health function at the School of Public Health and Tropical Medicine, Sydney, in 1969. [2] Responsibility for the Australian Malaria Register (AMR) was transferred to the Tropical Health Program at the University of Queensland in 1990. AMR reports for 1990 and 1991 have been previously published in CDI. [3,4]

The data available through the AMR are both more complete and more extensive. The better case ascertainment through the AMR is illustrated by comparing the numbers of cases reported by the AMR and the NNDSS for the two years 1992 (AMR 758 cases, NNDSS 741 cases) and 1993 (AMR 712 cases, NNDSS 669 cases). The more extensive dataset enables reporting of place of acquisition and has the capacity to identify cases which have occurred due to local transmission. Data on reason for travel and occupation are useful adjuncts to the data on place of acquisition. The Register also provides important information on diagnostic delay and misdiagnosis and incorporates data on malaria deaths.

Although the AMR has the capacity to be a more useful dataset than the NNDSS, the timeliness of data analysis and reporting has been a problem which has limited the value of the data collection for those involved in providing travel advice and clinical services to patients with malaria. A meeting between the AMR and Commonwealth, State and Territory health authorities in October 1995, made a number of recommendations for improving malaria surveillance [6] but these have not been implemented. One State health authority has formally advised the Commonwealth that, while continuing to investigate each case for possible local transmission and collate data relevant for that purpose, the State is no longer collecting the extended data needed for reporting to the AMR.

What should be the role of national malaria surveillance? Should it be limited to ensuring and documenting Australia's malaria free status or should surveillance attempt to answer broader questions about why travellers continue to contract this disease? Some of the questions which could be addressed include: Is malaria in Australia the result of inadequate travel advice from clinicians, or travellers not seeking travel advice in the first place? Are travellers failing to comply with treatment regimens and or engaging in activities which increase their risk? Are the currently prescribed regimens effective?

While the risk that malaria will be reintroduced to Australia is low, [7] the primary focus of malaria surveillance must continue to be the identification of and rapid public health response to introduced cases. At the national level, this could be achieved through the NNDSS with the addition of a field for classifying cases. Whether this approach would constitute a sufficient level of malaria surveillance for Australia could be debated.

The current AMR is an "outsourced " surveillance program that fits well within the model for national communicable disease surveillance developed in the National Communicable Diseases Surveillance Strategy.[8] However, it is time to reconsider the purpose of malaria surveillance and determine the best approach to this complex issue. The review of the list of nationally notified disease and case definitions currently being undertaken through the Communicable Diseases Network Australia New Zealand could provide a suitable forum for reconsidering these issues.

References

1. Hall R. Notifiable diseases surveillance, 1917-1991. *Commun Dis Intell* 1993;17:226-236.
2. Black RH. Malaria in Australia. Commonwealth Department of Health School of Public Health and Tropical Medicine, The University of Sydney, Service Publication No.9. Australian Government Printing Service. Canberra 1972.
3. Anonymous. CDI Editorial Comment. *Commun Dis Intell* 1993;17:142-143.
4. Forsyth S, Loeskow K, Pearce M, Riley I, Sleigh A and Srinivasa M. Report of the Australian Malaria Register for 1990. *Commun Dis Intell* 1991;15:400-408.
5. Sleigh A, Srinivasa M, Cooper A, Forsyth S, and Riley I. Report of the Australian Malaria Register for 1991. *Commun Dis Intell* 1993;17:134-142.
6. Longbottom H. Epidemiology of Malaria in Australia1991-1995. *Commun Dis Intell* 1996;20:84-87.
7. Walker J. Malaria in a changing world: an Australian perspective. *Int J Parasit* 1998;28(6):947-53.
8. National Communicable Disease Surveillance Strategy.

List of travel health advisory services

Travellers Medical and Vaccination Centre (TMVC). (Available in each capital city and some regional centres)

Health Services Australia (HSA). (Available in each capital city)

Medical Advisory Services to Travellers Abroad (MASTA). (Sydney)

Trends in Malaria in Australia,1991-1997

Bronwen Harvey

An analysis of the malaria cases in the current NNDSS dataset was undertaken for cases with onset dates in 1991 to 1997. To allow for reporting delays, the analysis for each year included cases with onset dates in that year which were reported up to and including 30 June in each subsequent calendar year. Data were downloaded from EpiInfo version 6 and analysed using SPSS for Windows version 8.0 Time series data for the period 1917- 1991 were downloaded from the NNDSS historic data collection in Microsoft Excel.

The number of malaria cases notified each year varied between a low of 622 in 1995 and a high of 866 in 1996. Each year, the state reporting the highest number of cases was Queensland (Table 1) . Males have predominated with a M:F ratio varying between 2.3 and 2.5. The highest number of cases has been in the young adult age groups, with peaks in the 20-24 years age group in 1991, 1993, 1994 and 1997 and in the 25-29 years age group in 1992, 1995 and 1996 (Table 2).

Data on malaria species have been included in the NNDSS since 1995. Although reporting was initially poor, with no species recorded for over 70% of cases in 1995, there has been a gradual improvement since then. Species data were recorded for 65% of cases in 1996 and 87% of cases in 1997. In each of these years, the predominant species was *Plasmodium vivax*, accounting for between 64% and 69% of the cases for whom species was reported. *P. falciparum* accounted for between 29% and 32% of cases for whom species was reported. Mixed *falciparum/vivax* infections occurred in a small proportion of cases (<1% to 2.3%) each year.

Data on Aboriginality are poorly reported in the NDSS and, in most years, aboriginality is not recorded for over half of the cases. Where status has been recorded, the number of cases reported as occurring in Aboriginal and Torres Strait Islander persons has varied from 0 (1995) to 12 (1997).

While the number of cases has fluctuated from year to year, there appears to have been a plateauing of the upward trend in cases that was seen in the 1970s and 1980s (Figure 1). There have also been no significant trends in the age, sex, geographic and seasonal distribution of cases. The proportions of cases due to *P. falciparum* in the 1995 - 1997 period are similar to those reported by the AMR for 1992 and 1993 (*Commun Dis Intell* 1998;22:11;237-244).

Figure 1. Malaria notifications, Australia, 1991-1997, by State or Territory

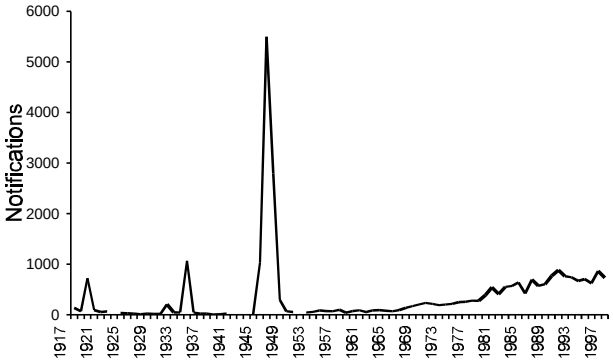


Table 1. Malaria notifications, Australia, 1991-1997, by State or Territory

	Year of onset						
State/ Territory	1991	1992	1993	1994	1995	1996	1997
ACT	22	26	18	24	21	27	17
NSW	139	125	174	185	98	213	162
NT	46	31	29	40	37	26	34
Qld	391	338	291	298	277	412	376
SA	40	35	25	30	24	21	22
Tas	10	14	11	14	1	5	5
Vic	79	132	81	83	127	107	83
WA	38	40	40	30	37	55	34
Total	765	741	669	704	622	866	733

Source: National Notifiable Diseases Surveillance System

Table 2. Malaria notifications, Australia, 1991-1997, by age group and sex

Age group (years)	Sex[1] 1991 M	1991 F	1992 M	1992 F	1993 M	1993 F	1994 M	1994 F	1995 M	1995 F	1996 M	1996 F	1997 M	1997 F
0-4	10	9	8	8	10	13	9	5	4	4	13	12	16	13
5-9	13	13	19	13	14	7	13	10	10	3	18	18	15	10
10-14	39	22	24	15	20	13	15	8	20	11	27	12	17	13
15-19	68	30	53	34	54	28	59	31	33	35	74	37	55	31
20-24	87	26	68	17	79	26	89	30	59	25	85	45	73	31
25-29	85	24	100	34	71	20	71	34	76	22	96	37	68	29
30-34	47	24	62	16	46	21	58	20	50	14	58	32	63	27
35-39	46	17	57	19	39	15	41	18	42	15	60	20	50	17
40-44	49	11	37	16	39	15	50	16	38	21	46	14	38	11
45-49	36	11	29	10	38	12	30	8	45	7	56	10	43	12
50-54	9	8	18	3	17	6	32	12	24	9	19	7	34	10
55-59	12	1	11	7	16	6	12	1	17	2	20	7	15	3
60-64	10	4	12	5	8	2	4	3	7	1	7	4	8	3
65-69	3	1	4	2	5	4	3	4	7	3	13	2	10	2
70-74	3	0	8	0	4	1	2	0	3		4	2	2	2
75-79	1	0	1	0	1	0	2	0	1	1	2	0	2	2
80-84	1	0	0	1	0	0	1	0	1	1	0	0	0	1
85+	2	0	1	1	0	0	0	0		1	0	0	0	0
NS	16	17	17	5	10	2	8	4	5	2	5	1	0	2
Total	537	218	529	206	471	191	499	204	442	177	603	260	509	219

Source: National Notifiable Diseases Surveillance System

1. Excludes the following cases for whom sex was not stated: 1991 = 10, 1992 = 6, 1993 = 7, 1994 = 1, 1995 = 3, 1996 = 3, 1997 = 5.

VRE; a public health context

Alexandra Geue, Senior Microbiologist, National Centre for Disease Control

In recent weeks newspapers around Australia have reported outbreaks of vancomycin-resistant enterococci (VRE) in five Victorian hospitals. To date, VRE has not been a major cause of hospital infection in Australia, unlike the United States where it has become a major nosocomial pathogen. Since 1994, when Australia's first case of VRE was detected, the National Antimicrobial Resistance Surveillance Program (NARSP) has collected data on 69 cases of VRE from around Australia which are presented in a timely report for this issue of *CDI*.[1]

The outbreaks in Victoria serve as a reminder that we must constantly examine and improve our public health practices. Vancomycin is currently the last line of treatment against methicillin resistant *Staphylococcus aureus ,* a common cause of nosocomial infection in Australia. Japan and the United States have already seen nosocomial infections with *S. aureus* that have resistance to vancomycin. Australia needs to heed this warning and re-examine current practice. Community use of antibiotics in Australia is one of the highest in the developed world.[2] Antibiotics are also used in animal feeds. The extent to which these practices are contributing to the increasing antibiotic resistant pathogens in humans is largely unknown, but this question is being examined by the Joint Expert Technical Advisory Committee on Antimicrobial Resistance (JETACAR), a joint initiative of the Federal Ministers for Health and Industries. This committee is expected to complete its work before 1999.

Effective infection control in health care settings remains a vital strategy in containing and preventing nosocomial infections. In the National Centre for Disease Control a review of the national infection control guidelines *Infection Control in the Health Care Setting* is under way. This will utilise experts from across the health care professions in drafting a new national infection control policy. The revised document will include more comprehensive and up-to-date information for controlling antibiotic resistant organisms in health care settings.

Last but not least, outbreaks of VRE remind us of the importance of surveillance. Early detection of resistant organisms can provide important early warnings of changes in our environment that may impact on public health. Without comprehensive active surveillance we can not develop effective or well targeted infection control policies. Surveillance of nosocomial infections and antibiotic resistance in animals and humans has been identified as a high public health priority by the Communicable Disease Network Australia New Zealand.

1. Bell J, Turnidge J, Coombs G, O'Brien F. Emergence and epidemiology of vancomycin-resistant enterococci in Australia. *Commun Dis Intell* 1998;22:249-252.
2. McManus P, Hammond ML, Whicker SD, Primrose JG, Mant A, and Fairall SR. Antibiotic use in the Australian community, 1990-1995. *MJA* 1997;167:124-127.

Emergence and epidemiology of vancomycin-resistant enterococci in Australia

Jan Bell,[1] John Turnidge,[1] Geoffrey Coombs,[2] Frances O'Brien[3]

Abstract

Enterococci with acquired resistance to vancomycin and other glycopeptides (VRE) have emerged and spread rapidly through Europe and the United States since 1988. The first isolate of VRE in Australia occurred in 1994. Only one case was noted in 1995. Since March 1996 there has been a steady increase in the number of reports of VRE throughout the country. To August 1998 there have been 69 documented strains or clusters of strains detected in patients with documented infection, and about 3 times as many strains have been detected through screening procedures of contacts or in risk groups. 19% of strains whose source was known were blood isolates, while 34% came from urine and 47% came from other specimens. The strains have been found in 26 institutions in 10 widely separated cities or regions of the country (in 6/8 states or territories), without any obvious temporal associations in their appearance. All strains appear to have arisen locally except for one strain imported from the United Kingdom. Furthermore there was no direct evidence of interhospital transfer of strains. All clinical strains were examined by PCR to confirm species and to test for the presence of known vancomycin-resistance genes. Of the 69 strains, 42 were *vanB E. faecium*, 12 were *vanA E. faecium*, 9 were *vanB E. faecalis*, 3 were *vanA E. faecalis*. Three were negative for *vanA*, *vanB*, *vanC1*, *vanC2/C3* and *vanD*. PGFE profiles on 38 strains have revealed at least 8 types of *vanB E. faecium*, 6 of *vanA E. faecium*, 4 of *vanB E. faecalis* and 2 of *vanA E. faecalis*. Isolates containing *vanA* always had different profiles from those containing *vanB*. Clinical clustering was confirmed by PFGE, and supported by extended antibiogram. 14 of 15 *E. faecalis* were ampicillin susceptible compared to only 2 of 54 *E. faecium*. One *E. faecalis* strain was ß-lactamase positive. The epidemiology of VRE in Australia appears to be different from that of Europe or the United States, since *vanB E. faecium* predominates and strains have appeared in diverse locations independently and are highly polyclonal. *Commun Dis Intell* 1998;22:249-252.

Introduction

Vancomycin-resistant *Enterococcus faecium* and *E. faecalis* (VRE) were first described in Britain in 1988 and soon afterwards were reported from other European countries and the United States. In the United States they have become major nosocomial pathogens, rising in incidence from 0.3% in 1989 to 7.9% in 1993 as reported by the CDC,[1] and among patients in intensive-care units, now representing 14% of blood culture isolates of enterococci.[1] The rapid emergence of VRE in the United States has been attributed to the intensive clinical use of vancomycin in both parenteral and oral forms in that country,[2] on a background of high level usage of cephalosporins which promote enterococcal superinfection. In Europe, investigators have postulated an additional role for the use of the glycopeptide avoparcin as a growth promoter in intensive animal industries, resulting in colonisation with VanA *E. faecium* and subsequent transmission to humans via the food chain.[3] The first vancomycin-resistant *E. faecium* in Australia was isolated from a liver transplant recipient in Melbourne in 1994.[4] Since March 1996 multiple isolates of vancomycin-resistant *E. faecium* and vancomycin-resistant *E. faecalis* have occurred throughout Australia. Only a few of these strains have been reported in the literature.[5, 6, 7, 8] As a referral centre for antimicrobial resistance in Australia, we have collected isolates from virtually all known instances of VRE infection that have occurred since 1994. In order to characterise these strains further we have developed multiplex PCR assays for *vanA*, *vanB*, *vanC1* and *vanC2/3*,[9] and have used these to examine the genetic basis for vancomycin resistance in Australian isolates of VRE. Results have been compared to those obtained by conventional susceptibility testing against glycopeptides.

Methods

Bacterial Strains

Clinical isolates of *Enterococcus* spp. referred to the National Antimicrobial Resistance Surveillance Program were studied. For the purposes of analysis only strains isolated from clinical specimens were included (whether pathogenic or commensal at the site). Where there were clusters of epidemiologically related isolates with the same phenotype and genotype, this cluster was recorded as one isolate (index case). Although many additional isolates have been detected at several institutions around the country, these have not been included as there are insufficient data available to the Surveillance Program to determine whether these isolates are related or unrelated to the clinical isolates.

1. National Antimicrobial Resistance Surveillance Program, Department of Microbiology and Infectious Diseases, Women's and Children's Hospital, Adelaide.
2. Department of Microbiology, Royal Perth Hospital.
3. School of Biomedical Science, Curtin School of Technology, Perth.

Identification and antimicrobial susceptibility testing

Isolates were identified by a conventional test scheme.[10] A multiplex PCR assay based on specific detection of genes encoding D-alanine:D-alanine ligases (ddl)[11] was used to confirm identification of *E. faecalis* and *E. faecium*. MICs of vancomycin and teicoplanin were determined for each isolate by the E-test (AB Biodisk, Solna, Sweden) method on Mueller-Hinton agar. The interpretative criteria of the NCCLS[12] were used for determining susceptibility of the isolates.

Vancomycin Resistance Gene Typing by PCR

A multiplex PCR assay was used for the detection of *vanA*, *vanB*, *vanC1*, and v*anC2* or *vanC3* genes.[9] Genotype-negative VRE isolates, with vancomycin MICs 4 µg/ml, were also tested for the presence of *vanD* using primers described by Perichon et al.[13]

Pulsed-Field Gel Electrophoresis

Thirty eight strains were analysed by PFGE. Chromosomal DNA was digested with *Sma* 1 restriction endonuclease and patterns interpreted according to the criteria of Tenover.[14]

Results

Vancomycin-resistant genotypes

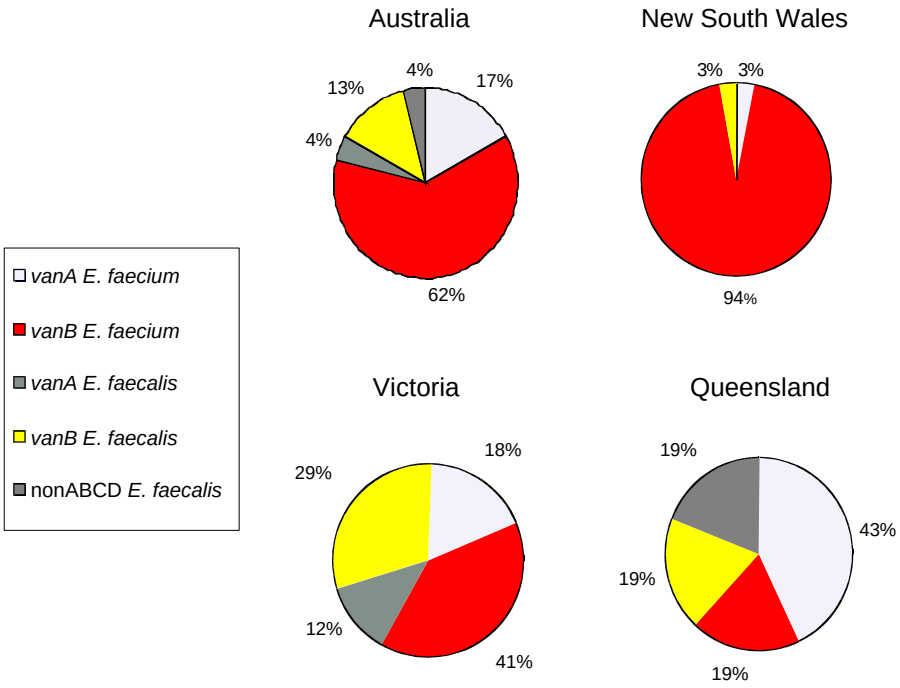
To the middle of September 1998, a total of 69 VRE isolates or clusters of isolates were found in 26 institutions in 10 cities throughout all mainland states of Australia (Figure 1). Results of PCR analysis of the van genotype are shown in Table 1. There were 42 (62%) *vanB E. faecium*, 12 (17%) *vanA E. faecium*, 9 (13%) *vanB E. faecalis*, three *vanA E. Faecalis* and three *van*-negative *E. faecalis* isolates. Two *E. faecium* and two *E. faecalis* isolates which were PCR-positive for *vanB* had intermediate resistance to vancomycin (MICs 8-16 µg/ml).

Figure 1. Evolution of clinical VRE isolates over time by institution.

Institution	Year and quarter																	Total for Institution
	1994		1995				1996				1997				1998			
- City*	q3	q4	q1	q2	q3	q4	q1	q2	q3	q4	q1	q2	q3	q4	q1	q2	q3	
A - Mel	1								1			3						5
B - Dar				1														1
C - New								1				2	1					4
D - Syd								1	1	4	3	2	1			1		13
E - Bri								1		1		1						3
F - Bri									1	1								2
G - Mel									1	1					2			4
H - Per									1									1
I - Bri									1	1	1	1				1	1	6
J - Syd									1							1		2
K - Mel										1								1
L - New										1		1	2	1	2			7
M - Mel												1						1
N - Syd												1						1
O - Ade													2					2
P - Mel														1				1
Q - New															1			1
R - Per															1			1
S - Mel															2		1	3
T - Vic																1		1
U – Bri																1	2	3
V – NSW																1		1
W – Bri																	2	2
X – Syd																	1	1
Y – NSW																	1	1
Z – Mel																	1	1
Total for quarters	1	0	0	1	0	0	0	3	7	10	4	12	6	2	8	6	9	69

Each institution designated by a letter. City abbreviations: Mel = Melbourne, Dar = Darwin, New = Newcastle, Syd = Sydney, Bri = Brisbane, Per = Perth, Vic = Victorian city not Melbourne, NSW = New South Wales city not Sydney or Newcastle

Figure 2. Prevalence of different VRE genotypes in Australia and different states.



One *vanA E. faecalis* isolate had a VanB phenotype (teicoplanin MIC of 4 µg/ml). One VanA *E. faecium* isolate which was vanB-positive had a teicoplanin MIC of 256 µg/ml. Three *E. faecalis* isolates were consistently negative for *vanA*, *vanB*, *vanC1*, *vanC2/3* and *vanD* in spite of exhibiting a VanB phenotype (vancomycin MICs 12-16 µg/ml). All three isolates were from the same institution. There was significant variation in both the species distribution and genotype of VRE isolates between States (Figure 2).

Table 1. Resistance Genotypes.

Species	*vanA*	*vanB*	nonABC	Total
E. faecium	12	42	-	54
E. faecalis	3	9	3	15
Total	15	51	3	69

Isolate sources

A high proportion of the 'index' case isolates were from blood (17%) (Table 2). The types of specimens from which VRE were isolated were otherwise typical of enterococci.

Other susceptibilities

Fourteen of 15 *E. faecalis* were ampicillin sensitive. The ampicillin resistant *vanB E. faecalis* was ß-lactamase positive. Similarly only 2 of 52 *E. faecium* were ampicillin sensitive. Both were *vanA* genotype.

Pulsed-Field Gel Electrophoresis

PFGE studies demonstrated at least 16 types of *E. faecium* (n=29) and at least 7 types of *E. faecalis* (n=9). The studies confirmed outbreak clusters. However, few institutions had strains in common, with only 4 PFGE types (*E. faecium* 3, *E. faecalis* 1) being detected in more than one institution. This finding was consistent with the fact that there is has been no known transfer of VRE between institutions. Moreover multiple unrelated were strains found even in a single institution.

Table 2. Specimen Source.

Source	*E. faecalis*	*E. faecium*	Total	
Urine	3	18	21	30%
Blood	3	9	12	17%
Other	4	25	29	42%
Stool, rectal, perianal	4	12	16	
Intra-abdominal		3	3	
Bile		4	4	
CAPD fluid		1	1	
Skin		5	5	
Unknown	5	2	7	10%
Total	15	54	69	

Discussion

The VRE isolated in Australia to date show considerable diversity in phenotype, genotype and geographic location. Cases have largely arisen sporadically and there has been no obvious geographic evolution, unlike in the USA where VRE strains have progressed from the North Eastern seaboard to the South East over several years. All four combinations of genotype and species have been found, with the commonest being *vanB E. faecium*. While the clinical profiles of VRE-affected patients appear to be similar to that recorded in the US and elsewhere,[2] the predominance of *vanB E. faecium* rather than *vanA E.*

faecium suggests different epidemiology from either Europe or the USA.

The origin of VRE in Australia remains unclear. One strain definitely appears to have been imported from the UK. Another strain occurred in a liver transplant recipient who was a New Zealand-born resident of Taiwan. This patient had entered Australia specifically for transplantation a few days prior to the procedure. *E. faecalis* of VanB phenotype was initially isolated from blood cultures after surgery. The patient was treated with teicoplanin, but several days later VRE was again isolated from blood cultures, with the isolate identified as *E. faecalis* of VanA phenotype. Genotyping showed both isolates to possess the *vanB* gene, and subsequent ribotyping confirmed the strains to be identical. Emergence of resistance to teicoplanin has been recorded previously, albeit rarely.[15]

Vancomycin usage in Australia is relatively high and has been increasing over the last decade (Eli Lilly Australia Pty Limited, personal communication). There is significant regional variation in its use due to the variation in prevalence of multi-resistant *Staphylococcus aureus*. Australia is also a high user of avoparcin as a growth promoter in the intensive animal industries. It is possible that the novel epidemiology of VRE in Australia may result from a combination of high usage vancomycin and avoparcin in humans and animals, respectively.

All three strains with the VanB phenotype, but lacking *vanA, vanB, vanC1, vanC2* or *vanC3*, or *vanD* came from a single institution and gave two distinct pulsed-field gel electrophoresis patterns. Our results are consistent with either the existence of a significant variant of a current *van* genotype or a novel one. The *van* loci of these strains are undergoing further analysis.

Acknowledgements

We wish to thank all the contributing laboratories throughout Australia who provided enterococci for this study. The National Antimicrobial Resistance Surveillance Program (NARSP) is supported by a grant from the National Centre for Disease Control.

References

1. Centers for Disease Control and Prevention. 1993. Nosocomial enterococci resistant to vancomycin - United States, 1989-1993. *MMWR Morbid. Mortal. Weekly Rep*. 42:597-599.
2. Leclercq R., and P. Courvalin. 1997. Resistance to glycopeptides in enterococci. *Clin. Infect. Dis.* 24:545-546.
3. Aarestrup F. M., P. Ahrens, M. Madsen, L. V. Pallesen, R. L. Poulsen, and H. Westh. 1996. Glycopeptide susceptibility among Danish *Enterococcus faecium* and *Enterococcus faecalis* isolates of animal and human origin and PCR identification of genes within VanA cluster. *Antimicrob. Agents Chemother*. 40:1938-1940.
4. Kamarulzaman A., F. A. Tosolini, A. L. Boquest, J. E. Geddes, and M. J. Richards. 1995. Vancomycin-resistant *Enterococcus faecium* in a liver transplant recipient [abstract]. *Aust. NZ J. Med.* 25:560.
5. Branley J., B. Yan, and R. A. V. Benn. Vancomycin-resistant *Enterococcus faecalis* [letter]. *Med. J. Aust.* 1996;165:292.
6. Faoagali J., J. Bodman, and A. Geary. 1996. Isolation of vancomycin-resistant enterococci in Queensland, case 2. *Commun Dis Intell* 1996;20:402-403.
7. Ferguson J., H. Butt, C. Johnson, and M. Boyle. 1996. Vancomycin-resistant *Enterococcus faecium* colonisations [letter]. *Med. J. Aus*t. 165:292-293.
8. Paterson D., A. Jennings, A. Allen, K. Sherlock, and M. Whitby. 1996. Isolation of vancomycin-resistant enterococci in Queensland, case 1. *Commun Dis Intell* 1996;20:400-401.
9. Bell J. M., J. C. Paton, and J. Turnidge. 1998. Emergence of Vancomycin-Resistant Enterococci in Australia: Phenotypic and Genotypic Characteristics of Isolates. *J. Clin. Microbiol*. 36:2187-2190.
10. Facklam R. R., and M. D. Collins. 1989. Identification of *Enterococcus* species isolated from human infections by a conventional test scheme. *J. Clin. Microbiol*. 27:731-734
11. Dutka-Malen S., S. Evers, and P. Courvalin. 1995. Detection of glycopeptide resistance genotypes and identification to the species level of clinically relevant enterococci by PCR. *J. Clin. Microbiol.* 33:24-27. (Erratum, 33:1434)
12. National Committee for Clinical Laboratory Standards. 1997. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically - 4th ed. Approved standard M7-A4. National Committee for Clinical Laboratory Standards, Villanova, Pa.
13. Perichon B., P. Reynolds and P. Courvalin. 1997. VanD-type glycopeptide-resistant *Enterococcus faecium* BM4339. *Antimicrob. Agents Chemother*. 41:2016-2018.
14. Tenover, F.C., R.D. Arbeit, R.V. Goering, P. Mickelsen, B.E. Murray, D.H. Persing, and B. Swaminathan. 1995. Interpreting chromosomal DNA restriction patterns produced by pulsed-field gel electrophoresis: criteria for bacterial strain typing. *J. Clin. Microbiol*. 33:2233-2239.
15. Hayden, M. K., G. M. Trenholme, J. E. Schultz, and D. F. Sahm. 1993. In vivo development of teicoplanin resistance in a VanB *Enterococcus faecium* isolate. *J. Infect. Dis*. 167:1224-1227.

Measles Control Campaign Update

During the three month period of the Campaign, the uptake of measles-mumps-rubella (MMR) vaccine given at primary school clinics and the number of adverse events following MMR vaccination are being monitored. Data are forwarded to the National Centre for Disease Control for collation and publication in *CDI*.

Measles Control Campaign activity data, cumulative to 25 September 1998[1]

Sum total students	902,261
Total forms returned	832,214
Consents to vaccinate	697,287
Total students immunised	651,615
Percentages are:	
Of total students	93% returned their forms
Of total forms returned	84% consented to vaccination
Of total consents to vaccination	95% have been vaccinated
Of total students	72% have been vaccinated.

1. Victorian data are not included in the activity data report, but are included in the adverse events report.

Adverse events

Faints/syncopy	17
Syncopal fits	13
Anaphylaxis	4
Hyperventilation	3
Rash	2
Local allergic reaction	2
Severe immediate local reaction	1
Arthropathy	1
Fever	1
Anxiety	1
Lymphadenopathy	1
Myalgia/Lymphadenopathy/ headache/stiff neck/rash	1
Immediate acute unilateral parotitis	1

Enquiries can be directed to Sue Campbell-Lloyd, National Manager of the Measles Control Campaign, Sydney Office, Commonwealth Department of Health and Aged Care, PO Box 9848, Sydney 2000, phone (02) 9263 3990, email Sue.Campbell-Lloyd@health.gov.au.

CDI Instructions for authors

Communicable Diseases Intelligence (CDI) is a four weekly publication of the National Centre for Disease Control, Commonwealth Department of Health and Family Services and the Communicable Diseases Network Australia. Its aim is to provide timely information about communicable diseases in Australia to those with responsibility for their control. *CDI* has a particular emphasis on public health issues.

CDI invites contributions dealing with any aspect of communicable disease incidence, risk factors, surveillance or control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence.

On receipt of an article, *CDI* sends a brief acknowledgment indicating that it will be considered for publication. The article will then undergo a review process which may include peer review by two experts in the topic area. Articles may be rejected without peer review. Occasionally reports of urgent public health importance may be published immediately, at the discretion of the Editor. Authors may be asked to revise articles as a result of the review process and the final decision about publication is made by the Editor.

CDI is published on every fourth Thursday of the year. It is finalised for printing on the Monday prior to the publication date. Very topical brief contributions (for example reports of current outbreaks) may be published in the period of receipt, by arrangement with the editorial staff.

Submission procedure

A single copy of the contribution should be submitted to The Deputy Editor, *Communicable Diseases Intelligence*, at the address below. A covering letter should identify the corresponding author and be signed by all authors agreeing to possible publication.

The contribution should be provided in hard copy and on diskette (3.5 inch disks). Microsoft Word for Windows Version 6 (or earlier version) or Rich Text Format (RTF) files should be used. Either Times New Roman or Arial font is preferred. Short contributions may also be sent by email.

Authors

Authors of articles should be identified by their first name, last name, institution and address, with phone and fax contacts for the corresponding author. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Articles and short reports

The text of articles should be structured to contain abstract, introduction, methods, results, discussion, acknowledgments and references, as far as is possible. Short contributions may need fewer subsections. There is no strict word limit for articles but manuscripts of 2,000 words or less are preferred. A word count should be included with the contribution.

Tables and figures

All tables and figures should be referred to within the results section and should not duplicate information in the text. Graphs published are produced in Microsoft Excel. If graphs are to be included, the numerical data on which these are based should also be provided to enable production in house style. Black and white illustrations or photographs can be included if required.

References

References should be identified consecutively in the text by the use of superscript numbers. The Vancouver reference style is used by *CDI* (see International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Ann Intern Med* 1997;1126:36-47). All unpublished material should be referred to within the text (instead of the reference list) as personal communication or unpublished observation. The only exception is material which has been accepted for publication (in press).

Protection of patients' rights to privacy

Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity. Complete anonymity may be difficult to achieve, and written informed consent should be obtained if there is any doubt. Informed consent for this purpose requires that the patient be shown the manuscript to be published.

When informed consent has been obtained it should be included in the article.

Contact details

Contributions and requests for further information should be sent to: The Deputy Editor, *Communicable Diseases Intelligence*, National Centre for Disease Control, MDP 6, GPO Box 9848, Canberra, ACT 2601.
Telephone: (06) 289 6895 Fax: (06) 289 7791
Email: alison.milton@health.gov.au

Notice to authors

Citations referring to this journal should use the abbreviation *Commun Dis Intell* to be consistent with that used by Medline citation.

Communicable Diseases Surveillance

Highlights

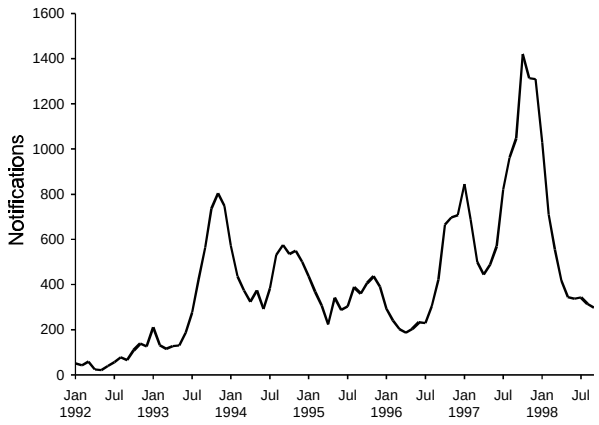
Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) is a 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

Pertussis notifications continue to fall slightly when examined by date of onset (Figure 1). The downward trend has continued for each month of this year. This is encouraging, given that the rise towards the peak in late 1997 began in May 1997. Most notifications having onset in 1998 are in children aged 5 to 9 years (17%), 10 to 14 years (16%) and 0 to 4 years (11%).

Numbers of notifications for other vaccine preventable diseases also remain low.

Figure 1. Notifications of Pertussis, January 1992 to September 1998, by month of onset.



Arboviruses

9 notifications of dengue have been recorded for the current reporting period compared with 24 in the previous reporting period. This brings the total reported in 1998 to 390.

The numbers of new notifications for Ross River virus infection have also continued to decline for several months as expected for the time of year.

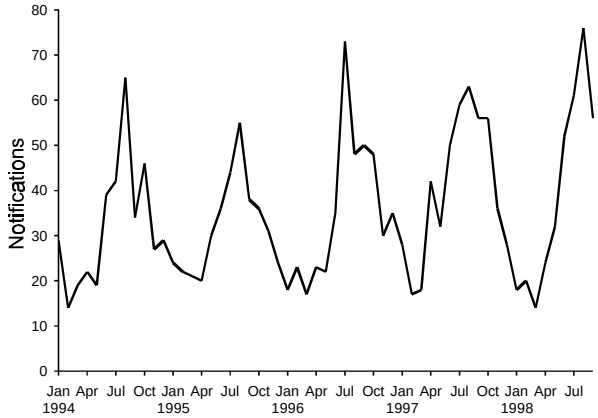
Hepatitis A

Numbers of notifications of hepatitis A remain lower than those seen in the first 6 months of 1998.

Meningococcal infection

The number of notifications of meningococcal infection reflects the higher level usually recorded in Australia during Winter and Spring (Figure 2).

Figure 2. Notifications of meningococcal disease, Australia, January 1994 to September 1998, by month of onset.



SLTEC infections, HUS and TTP

Reporting of these conditions commenced in the previous issue of CDI which includes the case definitions (Commun Dis Intell 1998;22:223). No cases have been reported for the current period.

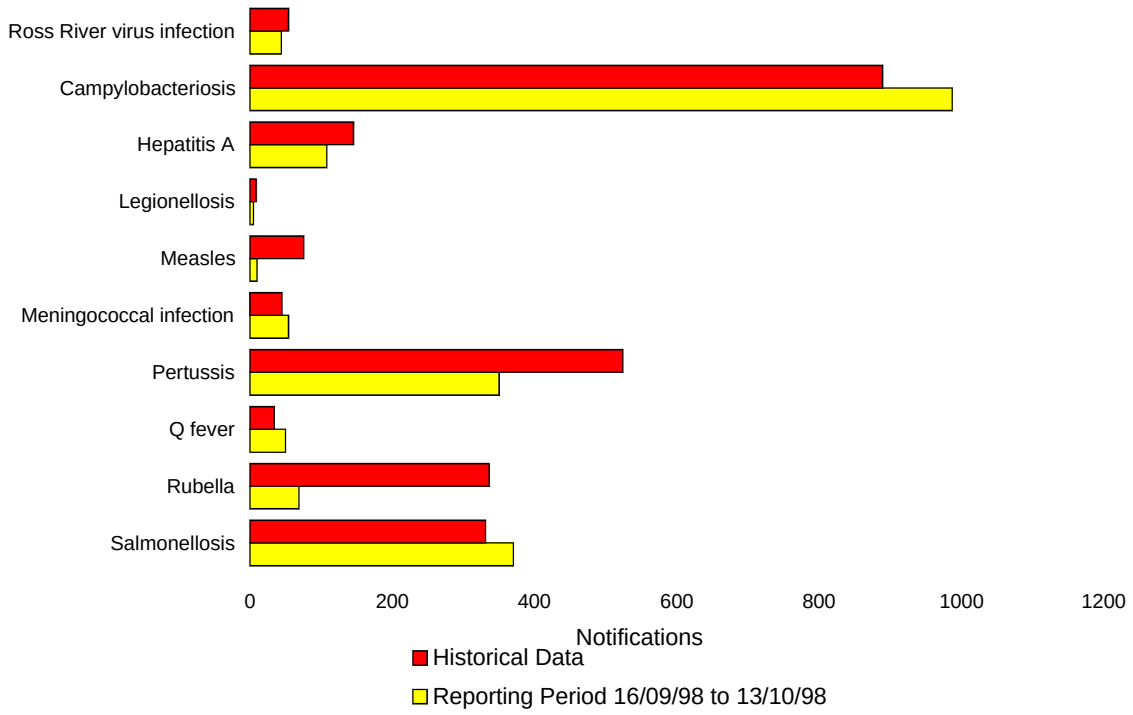
Tables

There were 3,932 notifications to the National Notifiable Diseases Surveillance System (NNDSS) in the four week period, 16 September to 13 October 1998 (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 3).

There were 2,201 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the four week period, 10 September to 7 October 1998 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 36 to 39, ending 4 October 1998, are included in this issue of *CDI* (Table 5).

Figure 3. Selected National Notifiable Diseases Surveillance System reports, and historical data.[1]



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

Table 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 16 September to 13 October 1998.

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
H. influenzae type b infection	0	1	0	2	0	0	0	0	3	4	24	41
Measles[3]	1	2	0	1	0	1	3	2	10	95	282	516
Mumps	1	1	0	1	0	0	2	2	7	13	147	158
Pertussis	10	158	1	75	29	2	67	8	350	824	5,321	6,555
Rubella[4]	2	7	1	44	0	0	13	2	69	161	666	1,147
Tetanus	0	0	0	1	0	0	0	0	1	0	5	7

NN. Not Notifiable

1. No notification of poliomyelitis has been received since 1986.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. The total number of measles notifications for 1998 has been revised downwards because of a reclassification of 79 cases previously notified as measles by Victoria. These cases have been reclassified as not measles following results of serology.
4. Includes congenital rubella.

Table 2. Notifications of diseases received by State and Territory health authorities in the period 16 September to 13 October 1998.

Disease[1,2,3,4]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998[5]	Year to date 1997
Arbovirus infection (NEC)[6]	0	2	0	3	0	0	0	1	6	6	65	114
Barmah Forest virus infection	0	13	3	14	0	0	0	0	30	30	476	568
Brucellosis												
Campylobacteriosis[7]	16	-	21	320	201	27	286	116	987	738	8,855	8,737
Chlamydial infection (NEC)[8]	20	NN	75	294	80	10	166	100	745	508	8,487	7,185
Cholera												
Dengue	0	4	0	4	1	0	0	0	9	1	390	196
Donovanosis	0	NN	0	0	NN	0	0	0	0	2	28	26
Gonococcal infection[9]	2	60	108	104	18	0	58	47	397	240	4,248	3,494
Hepatitis A	3	29	2	44	5	0	14	11	108	172	2,211	2,507
Hepatitis B incident[5]	0	5	2	1	1	0	4	0	13	18	184	203
Hepatitis C incident[10]	0	5	1	-	4	0	-	-	16	4	251	55
Hepatitis C unspecified[5]	17	NN	15	249	NN	22	13	70	386	611	6,724	7,498
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	0	4	15
Haemolytic uraemic syndrome[11]	NN	0	NN	0	0	0	NN	0	0	0	10	0
Hydatid infection												
Legionellosis	0	0	0	1	2	0	0	2	5	5	181	115
Leprosy												
Leptospirosis	0	5	0	7	0	0	3	0	15	7	131	96
Listeriosis	0	1	0	0	0	0	0	0	1	4	41	62
Malaria	1	3	3	14	0	0	6	1	28	58	596	670
Meningococcal infection	1	18	0	16	3	1	9	6	54	42	371	380
Ornithosis	0	NN	0	0	0	0	0	0	0	0	28	39
Q Fever	0	28	0	16	5	0	1	0	50	37	449	468
Ross River virus infection	0	16	1	24	0	0	0	3	44	40	2,482	6,404
Salmonellosis (NEC)	3	73	24	124	14	5	87	40	370	316	6,165	5,483
Shigellosis[7]	0	-	9	8	3	0	8	4	32	45	480	638
SLTEC, VTEC[12]	NN	0	NN	NN	0	0	NN	NN	0	0	14	0
Syphilis[13]	2	32	16	49	2	0	0	1	102	71	1,203	1,010
Tuberculosis	0	14	0	3	2	0	18	2	39	82	773	808
Typhoid[14]	0	1	0	0	0	0	2	1	4	2	61	59
Yersiniosis (NEC)[7]	0	-	0	6	2	0	2	1	11	9	180	199

1. Diseases preventable by routine childhood immunisation are presented in Table 1.
2. For HIV and AIDS, see Tables 6 and 7.
3. 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.
4. No notifications have been received during 1998 for the following rare diseases: botulism (foodborne), lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers. There have also been no cases of thromotic thrombocytopaenic purpura (TTP), which became nationally reportable in August 1998.
5. Data from Victoria for 1998 are incomplete.
6. NT: includes Barmah Forest virus.
7. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
8. WA: genital only.
9. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
10. Qld and Vic incident cases of Hepatitis C are not separately reported.
11. Nationally reportable from August 1998.
12. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC) became nationally reportable in August 1998.
13. Includes congenital syphilis.
14. NSW, Qld, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

NA Not applicable, as reporting for this condition did not commence until 1998.

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 10 September to 7 October 1998, and total reports for the year.

	State or Territory[1]								Total this period	Total reported in *CDI* in 1998
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles, mumps, rubella										
Measles virus					1				1	52
Mumps virus		2			1			3	6	37
Rubella virus				4	1		1		6	96
Hepatitis viruses										
Hepatitis A virus		1		4	4		1	6	16	327
Hepatitis D virus					1				1	5
Arboviruses										
Ross River virus				9	1		1	5	16	597
Barmah Forest virus				1				1	2	28
Dengue not typed			2					2	4	32
Flavivirus (unspecified)				1			3		4	60
Adenoviruses										
Adenovirus type 1					24				24	39
Adenovirus type 2					1		1		2	20
Adenovirus type 3					7		1		8	38
Adenovirus type 4							1		1	2
Adenovirus type 6					5				5	10
Adenovirus type 7					1				1	16
Adenovirus type 8							1		1	5
Adenovirus type 22							1		1	1
Adenovirus type 40							2		2	11
Adenovirus not typed/pending		16		3	49	1	6	12	87	647
Herpes viruses										
Cytomegalovirus		8		7	15		30	8	68	626
Varicella-zoster virus		2		14	16	1	25	21	79	998
Epstein-Barr virus		11	2	41	85		18	16	173	1,418
Other DNA viruses										
Papovavirus group							1		1	2
Parvovirus				3	4		8	7	22	188
Picorna virus family										
Coxsackievirus B4					1		1		2	6
Coxsackievirus B5							1		1	3
Echovirus type 18					1				1	6
Poliovirus type 1 (uncharacterised)							1		1	6
Rhinovirus (all types)		12						8	20	373
Enterovirus not typed/pending			3	4	1	1		20	29	397
Ortho/paramyxoviruses										
Influenza A virus		42	1	3	129	3	29	33	240	2,499
Influenza B virus					11		1		12	152
Parainfluenza virus type 1					5				5	270
Parainfluenza virus type 2					1				1	31
Parainfluenza virus type 3		4			16		2	14	36	280
Respiratory syncytial virus		85		7	283	80	267	49	771	3,850
Other RNA viruses										
HTLV-1			1					1	2	16
Rotavirus		38	3		52	17	87	13	210	896
Norwalk agent							5		5	30

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 10 September to 7 October 1998, and total reports for the year (continued).

	State or Territory[1]									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Total reported in *CDI* in 1998
Other										
Chlamydia trachomatis not typed		5	8	29	58	11	12	56	179	2,883
Chlamydia psittaci							3	1	4	40
Mycoplasma pneumoniae		13		16	35		36	6	106	1,089
Coxiella burnetii (Q fever)		1		2	1		1	2	7	97
Bordetella pertussis				15			19	2	36	832
Legionella longbeachae					1				1	28
TOTAL		240	20	163	811	114	566	287	2,201	19,047

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

Table 4. Virology and serology laboratory reports by contributing laboratories for the reporting period 10 September to 7 October 1998.

State or Territory	Laboratory	Reports
	New Children's Hospital, Westmead	119
New South Wales	South West Area Pathology Service, Liverpool	115
	Queensland Medical Laboratory, West End	167
Queensland	Townsville General Hospital	8
South Australia	Institute of Medical and Veterinary Science, Adelaide	811
Tasmania	Northern Tasmanian Pathology Service, Launceston	27
	Royal Hobart Hospital, Hobart	85
	Monash Medical Centre, Melbourne	48
	Royal Children's Hospital, Melbourne	399
Victoria	Victorian Infectious Diseases Reference Laboratory, Fairfield	123
	PathCentre Virology, Perth	258
	Princess Margaret Hospital, Perth	25
Western Australia	Western Diagnostic Pathology	16
TOTAL		2,201

Table 5. Australian Sentinel Practice Research Network reports, weeks 36 to 39, 1998.

Week number	36		37		38		39	
Week ending on	13 September 1998		20 September 1998		27 September 1998		4 October 1998	
Doctors reporting	62		60		56		48	
Total encounters	8136		7639		6773		5996	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	96	11.8	69	9.0	59	8.7	41	6.8
Rubella	1	0.1	1	0.1	0	0.0	4	0.7
Measles	1	0.1	1	0.1	1	0.1	1	0.2
Chickenpox	9	1.1	9	1.2	11	1.6	9	1.5
Pertussis	1	0.1	7	0.9	2	0.3	1	0.2
HIV testing (patient initiated)	17	2.1	11	1.4	20	3.0	8	1.3
HIV testing (doctor initiated)	6	0.7	5	0.7	4	0.6	4	0.7
Td (ADT) vaccine	57	7.0	44	5.8	42	6.2	38	6.3
Pertussis vaccination	35	4.3	41	5.4	41	6.1	33	5.5
Reaction to pertussis vaccine	0	0.0	1	0.1	1	0.1	1	0.2
Ross River virus infection	0	0.0	1	0.1	0	0.0	0	0.0
Gastroenteritis	73	9.0	80	10.5	91	13.4	78	13.0

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 1998;22:4-5.

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 1998;22:8.

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 1998. CDI reports the consultation rates for all of these. For further information, including case definitions, see CDI 1998;22:5-6.

Additional Reports

National Influenza Surveillance, 1998

Three types of data are included in National Influenza Surveillance, 1998. 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 1998; 22:83.

Sentinel General Practitioner Surveillance

Reports of influenza-like illness reported by the ASPREN, Tropical Influenza Surveillance Scheme (Northern Territory, Top End) and the Victorian and New South Wales Sentinel Practitioner Schemes have declined over the month of September. Peak activity was reported by the ASPREN and the Victorian Sentinel Practitioner Schemes in July, and by the New South Wales and Tropical Surveillance Schemes in August (Figure 4). The peak number of reports for this year has been lower across all schemes compared to 1997.

Laboratory Surveillance

There have been 2540 laboratory reports of influenza for the year to date. Of these, 2415 (95%) are influenza A and 125 (5 %) influenza B. Weekly reports of influenza A peaked in late July and early August (Figure 5). The number of influenza A reports for this year is greater than those reported over the same period for all years dating

Figure 4. Sentinel general practitioner consultation rates 1998, by week and scheme.

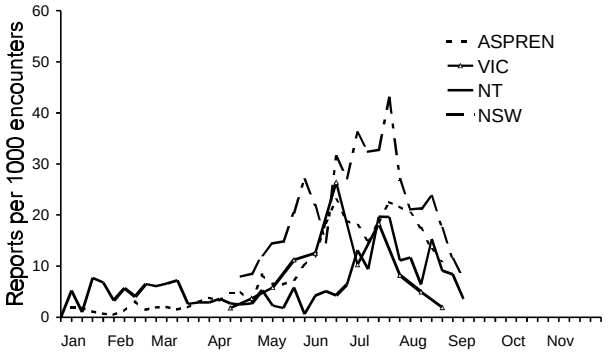


Figure 5. Influenza laboratory reports, 1998, by virus type and week of specimen collection.

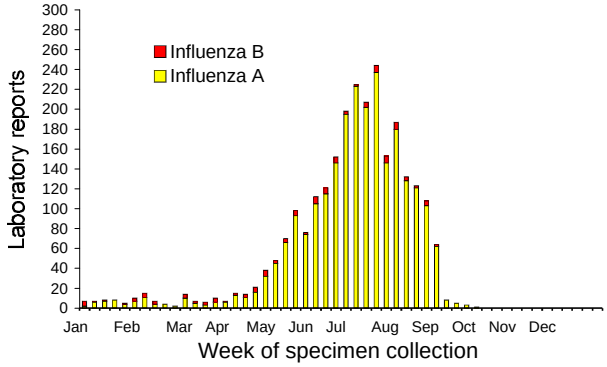
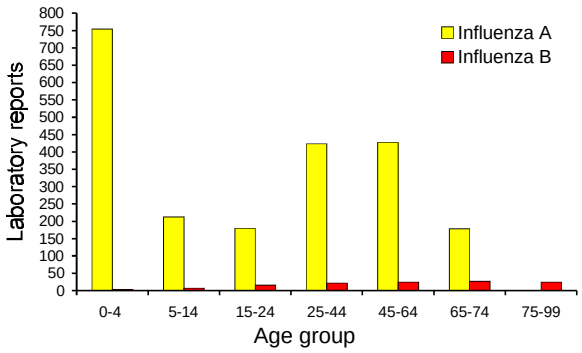
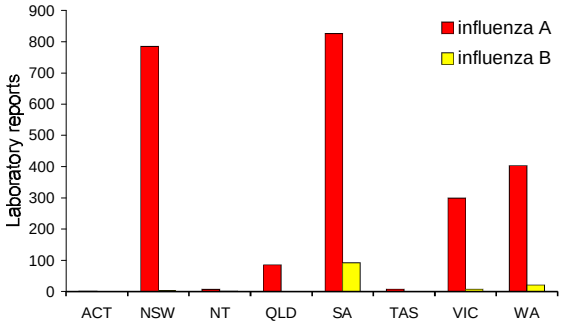


Figure 6. Influenza A and B laboratory reports, 1998, by age group.



back to 1993. 754 (31%) of influenza A and 3 (2.4%) of influenza B laboratory reports have been in children less than 4 years of age (Figure 6). 52 (41%) of all influenza B reports have been in those over the age of 65 years. Ninety-two (74%) of influenza B reports are from South Australia and 21(16%) from Western Australia, with relatively few influenza B reports from laboratories in the east and north of Australia (Figure 7).

Figure 7. Influenza A and B laboratory reports, 1998, by State and Territory.



The WHO Centre for Reference and Research has received 678 isolates of influenza A and 16 of influenza B for the year to date. All the influenza A viruses analysed have been H3N2 strains related to A/Sydney /5/97. One influenza B strain has been identified as B/Beijing/184/93-like. Thirty percent of influenza A strains analysed have shown reduced reactivity with A/Sydney/5/97 antiserum but this does not appear to indicate antigenic shift. A representative sample of these low avidity cell cultured strains regained reactivity with specific antisera when directly isolated in eggs.

Absenteeism surveillance

Rates of absenteeism in Australia Post employees for three consecutive days of each week have been reported on a weekly basis since late April. Absenteeism rates for the year have averaged 0.23% per week. Rates for September have averaged 0.15% which is slightly lower than what has been observed since April.

HIV and AIDS Surveillance

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

date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

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

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 31 May 1998, as reported to 31 August 1998, 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 31 May 1998, by sex and State or Territory of diagnosis.

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
HIV diagnoses	Female	1	2	0	2	0	0	1	1	7	5	33	33
	Male	1	23	2	7	5	0	9	1	48	52	287	320
	Sex not reported	0	2	0	0	0	0	0	0	2	1	8	10
	Total[1]	2	27	2	9	5	0	10	2	57	58	328	364
AIDS diagnoses	Female	1	0	0	0	0	0	0	0	1	2	5	15
	Male	1	9	1	1	1	0	0	0	13	20	76	139
	Total[1]	2	9	1	1	1	0	0	0	14	22	81	154
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	2	2	7
	Male	0	3	0	1	2	0	1	0	7	17	35	106
	Total[1]	0	3	0	1	2	0	1	0	7	19	37	113

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 31 May 1998, by sex and State or Territory.

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	22	552	7	128	52	4	194	88	1,047
	Male	183	10,318	96	1,821	633	77	3,728	857	17,713
	Sex not reported	0	263	0	0	0	0	25	0	288
	Total[1]	205	11,152	103	1,955	685	81	3,958	948	19,087
AIDS diagnoses	Female	8	159	0	45	19	2	64	23	320
	Male	82	4,373	32	766	321	41	1,527	337	7,479
	Total[1]	90	4,543	32	813	340	43	1,598	362	7,821
AIDS deaths	Female	2	112	0	28	14	2	45	16	219
	Male	62	3,049	23	529	219	27	1,205	241	5,355
	Total[1]	64	3,168	23	559	233	29	1,256	258	5,590

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

Bulletin Board

The Public Health Association of Australia Inc.,
6th National Conference on Immunisation
Immunisation; Beyond 2000
4-5 November 1998
Hilton on the Park, Melbourne
Phone: 02 6285 2373
Email: conference@pha.org.au

Centers for Disease Control and Prevention (USA) and the World Health Organization
2nd International Conference on Emerging Zoonoses
5-9 November 1998
Strasbourg, France
Phone: +33 1 4742 2016
Fax: +33 1 426 5 1725
Email: trgt@netvision.net.il

Communicable Diseases Network Australia New Zealand
Conference: *Control of Communicable Diseases in Australia*
10 November 1998
Australian National University, Canberra
Phone: 02 6289 8245
Fax: 02 6289 7791
Email: ccd.conf@health.gov.au

National Centre for Epidemiology and Population Health
Conference: *Developing Health*
11-12 November 1998
Canberra
Phone: 02 6249 5627
Fax: 02 6249 0740
Email: dev.health@nceph.anu.edu.au

The Australasian Society for HIV Medicine
19th Annual Conference
18-21 November 1998
Newcastle, Town Hall
Phone: 02 9382 1656
Fax: 02 9382 3699
Email: B.Pearlman@unsw.edu.au

The Australian Society for Microbiology Inc.
The 11th International Conference
International Congress of Virology
9-13 August 1999
International Congress of Bacteriology and Applied Microbiology
9-13 August 1999
International Congress of Mycology
16-20 August 1999
Sydney, New South Wales
Fax: 03 9262 3135
Email: tourhosts@tourhosts.com.au

The International Leptospirosis Society
2nd International Scientific Conference
22-25 August 1999
Kooringa Lodge, Marysville, Victoria
Phone: 03 9905 4815
Fax: 03 9905 4811
Web page and conference registration:
http://www.med.monash.edu.au/micro/department/leptconf/ils99.htm

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

Advance notice

Royal North Shore Hospital
Conference: *Outpatient Parenteral Therapy - beyond 2000*
17-22 September 2000
Fairmont Resort
Luera, New South Wales
Phone: 02 9956 8333
Fax: 02 0056 5154
Email: confact@conferenceaction.com.au

Health education resource

Talking about HIV/AIDS in the Kimberley, written by clinical psychologist, Pat Lowe, and illustrated by Carol Tang Wei, is a health education and counselling guide for use by health professionals working with Kimberley Aboriginal people. It can be purchased for $60 (postage and packing included) through Ms Ros Cain, of the Kimberley Public Health Unit, PMB 912, Derby WA 6728. Phone 08 9191 1144 or fax 08 9193 13 78

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 Family Services.

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

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 'Related sites' on the CDI homepage.

Diarrhoeal diseases

Bangladesh

The devastating floods which are sweeping the country began in early July this year and 52 districts out of a total of 64 with nearly 20 million people (25% of the total population) are affected. The death toll was reported as 730 up to 7 September 1998. Most of the districts currently affected are also endemic for diarrhoeal diseases and some outbreaks of acute diarrhoea have occurred. Over 185,000 people have so far been affected and 151 have died.

Cholera

Somalia

The cholera season in Somalia is now approaching. It is expected to be particularly deadly in Mogadishu this year, due to a variety of factors including internal displacement of people and poor facilities.

Cameroon

Since June when the outbreak began, cholera cases have been reported in the provinces of Extrême-Nord, Nord and Littoral, constituting a total of 1106 cases and 113 deaths. In the province of Extrême-Nord, 13 districts out of 22 have been affected by the outbreak. The 2 most affected are Kousseri, to the west of Djamena (104 cases and 24 deaths), and urban Maroua, the provincial capital (154 cases and 12 deaths). Since the start of the outbreak, this province has reported a total of 633 cases and 107 deaths (case-fatality rate, 17%). In the province of Nord, only Garoua, the provincial capital 200 km from Maroua, has reported cholera cases (4 cases and 2 deaths).

Cholera is endemic in the province of Littoral, especially in Douala, the country's main economic centre, where since the beginning of the year, 469 cases and 2 deaths have been reported. The Ministry of Health has taken the necessary measures to control the outbreak.

West Africa

There has been a recrudescence of cholera in the West Africa region since September 1998. Some countries in the region have been reporting cases of cholera since the beginning of the year. As from September, however, there has been a considerable increase in the number of countries reporting from the region and in the number of cases. Eleven countries are currently reporting cases of cholera with, for the time being, a relatively acceptable case fatality rate. The ministries of health of the countries affected are taking steps to ensure adequate case management and prevent further spread. These measures have been quite successful so far. WHO is concerned about the potential deterioration of the situation in the region and therefore strongly recommends the strengthening of epidemic preparedness and response activities for the countries in West Africa.

Correction

Vol 22:10:232. **Table 7. Cummulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introductionof HIV antibody testing to 30 April 1998, by sex and State or Territory.** Total HIV diagnoses should read 19015.

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

This journal is indexed by *Index Medicus* and Medline.

Subscriptions

Canberra Mailing, PO Box 650, Fyshwick ACT 2609,
fax (02) 6269 1212

Website

Http://www.health.gov.au/pubhlth/cdi/cdihtml.htm

Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereering process. Instructions to authors can be found in this issue of *CDI* 1998;22:11.