Volume 15, Number 11–12
November–December 2004

NSW Public Health *Bulletin*

NSW HEALTH
Working as a Team

ISSN 1034 7674
State Health Publication PH 040271

UNWANTED GUESTS: THE MISERIES, THE DANGERS AND THE GLORIOUS FUTURE OF BITING INSECTS AND VECTOR-BORNE DISEASES IN NEW SOUTH WALES

GUEST EDITORIAL

Krishna Hort
Wentworth Public Health Unit
Wentworth Area Health Service

Stephen Corbett
Centre for Public Health
Western Sydney Area Health Service

Insects have an infinite talent to annoy, and as vectors of disease they have had a decisive effect on human affairs, determining the fates of cities and armies,[12] religions and nations. The outbreak of bubonic plague in Europe during the 14th century is considered an important contributor to the demise of feudalism. Convocations of the College of Cardinals in Rome were regularly disrupted by mortality from the *mal aria* (literally, the 'bad air') of the Pontine Marshes.[3] Napoleon lured an English army into the malarial swamps of *Les Pays-Bas* (The Netherlands) to effect a famous victory.[3] In history, vector-borne diseases have been the constant and unwanted companions of new settlers, the adventurous, the poor, and marching armies and pilgrims.

Among the greatest achievements of the revolutions in microbiology and entomology at the end of the 19th century were the identification of the life cycle and vectors of malaria, typhus, yellow fever, and bubonic plague. These efforts quickly led to effective measures of control and dramatic reductions in mortality from these dreaded diseases.

The first President of the Board of Health and Chief Medical Adviser of New South Wales, John Ashburton Thompson, played an important role in confirming the role of the rat flea in the transmission of bubonic plague. His careful synthesis of epidemiological, entomological and microbiological data from the outbreak of bubonic plague in Sydney in 1900 was presented to great acclaim at the 14th International Conference on Hygiene and Demography, which was held in Berlin in 1907.[4]

continued on page 192

CONTENTS

Since then, expertise in medical entomology has been a vital part of the public health infrastructure. Mosquito-borne illness remains an important public health issue in New South Wales. Over the last 8 years there have been 8,000 notified cases of disease caused by Ross River virus and Barmah Forest virus. The number of cases not diagnosed or not notified is likely to be much higher.

The contemporary relevance of insects to public health endeavours—both as vectors of disease and as purveyors of ordinary human misery—is well illustrated in this edition of the *NSW Public Health Bulletin*.

We begin with 3 articles on mosquito-borne disease. Doggett provides an overview of mosquito-borne viruses (arboviruses) in New South Wales, with a focus on Ross River virus and the newly emerging Barmah Forest virus. Harvey and Dwyer examine the recent increases in notifications of Barmah Forest virus; and Heuston reviews the epidemiology of dengue fever in New South Wales.

These are followed by a collection of articles on the irritating and infuriating problems of lice, cockroaches, bedbugs and ticks. These bugs often have a commercial and emotional impact that far outweighs their significance for physical health. Nowhere is this better illustrated than in the history of the Nitbusters program. This modest but popular public health program has provided desperate parents and school principals with an effective method of dealing with this scourge of our school population.

Miller and Peters follow with a summary of what is known about some other common houseguests: the cockroaches. Torres and Carey remind us of the lifecycle of the tick and of potential tick-borne illness, and discuss their experience in developing an evidence-based approach to the removal of ticks. Ryan, Peters and Miller give us a fascinating account of their investigations of bedbugs in short-stay accommodation in the City of Sydney.

And finally, in the article by Geary and Russell, the maggot sets out on its long march towards rehabilitation as a force for good in public health—and as a potential export industry.

Globally, mosquito-borne illnesses, particularly malaria and dengue, are major public health problems. Malaria kills more than 1 million people each year, most of them children. Since 1998, the World Health Organization has coordinated the Roll Back Malaria Campaign to combat this disease. Like the Australian population, mosquitoes and mosquito-borne viruses are good travellers. There is an ever-present and perhaps ever-increasing threat (for example, through global warming) that these or other vector-borne diseases will gain a major foothold in Australia. The ingress of the dengue vector *Aedes albopictus* and the West Nile virus into the continental United States is the most dramatic recent example of the need for vigilance and the maintenance of high levels of surveillance and expertise in vector-borne disease.

A number of key messages emerge from this collection of articles on contemporary insect pests and vectors:

- climate change, increases in population, international travel and the movement of goods all heighten the risk of importation of insects and insect-borne disease;
- we need to maintain the capacity for surveillance and response to insect vectors of public health significance, especially mosquito-borne illness;
- we need to increase the awareness of clinicians and the general public of the significance of insect-borne disease, and foster appropriate habits of protection from attacks by mosquitoes;
- we need to actively monitor the effectiveness and potentially toxic effects of chemicals used to control insect pests.

ACKNOWLEDGEMENTS

The guest editors would like to acknowledge the efforts of all the contributors in the preparation of their manuscripts, and in responding to reviewers' comments, and in particular to Glenis Lloyd, from the Environmental Health Branch of the NSW Department of Health, who originally conceived the idea of this special 'bug' issue of the *NSW Public Health Bulletin*.

REFERENCES

1. Zinsser H. *Rats, lice, and history*. New York: Little, Brown and Company, 1984.
2. McNeill WH. *Plagues and peoples*. New York: Anchor, 1998.
3. Rocco F. *The miraculous fever tree*. New York: HarperCollins, 2003.
4. Thompson JA. Report to Section V of the XIVth International Conference on Hygiene and Demography on the mode of spread and prevention of plague in Australia, Berlin, 1907 (unpublished).

POPULATION HEALTH ASPECTS OF MOSQUITO-BORNE DISEASE IN NEW SOUTH WALES

Stephen Doggett
Department of Medical Entomology
Institute of Clinical Pathology and Medical Research, Westmead

Human pathogens transmitted by mosquitoes pose a significant threat to population health in New South Wales. Within the State, there is annual activity of Ross River virus and Barmah Forest virus, occasional activity from Kunjin virus and Sindbis virus, and rare epidemics of Murray Valley encephalitis virus. For the traveller, dengue and malaria are constant threats. The focus of this article will be the mosquito-borne viruses (the 'arboviruses'), including the factors that influence their activity in New South Wales, how they affect the community, and the future threats they pose to population health in the State.

ARBOVIRUS ACTIVITY IN NEW SOUTH WALES

Arbovirus activity is dependent on numerous factors: the availability of water (especially rainfall and tidal amplitude), temperature, mosquito vectors, reservoir hosts, past activity, geography, and population demographics.

Mosquitoes require water to breed; more water means more mosquitoes and disease. Mosquito activity is linked to temperature and therefore more cases of arbovirus infection occur in the warmer north of the State with its longer mosquito season. Competent vectors (that is, mosquitoes able to transmit the virus) are present in most of New South Wales.

Arboviruses cycle naturally between mosquitoes and vertebrate hosts. The distribution and movements of the host will also limit the virus distribution. For example, waterbirds, which are the natural hosts of Murray Valley encephalitis virus, do not disperse to the coast and hence the virus does not occur there.

In any year, arbovirus activity also depends on immunity in the population. Recent epidemics mean that levels of antibodies are high, which confers some protection to both natural hosts and humans. Conversely, little activity means antibodies are low and the population is highly susceptible.

The geography of New South Wales has defined 3 broad 'virogeographic' zones for arbovirus activity: the inland, tablelands and coast. The inland has low and inconsistent rainfall, with infrequent flooding resulting in occasional large outbreaks of activity. The irregular rainfall means that seasonal activity is highly variable. Much of the ongoing activity has arisen through human land uses, particularly that of irrigation, which often result in massive mosquito breeding.

The tablelands have little vector breeding and arbovirus activity, and many of the cases in this zone are probably acquired elsewhere. The Great Dividing Range, which forms the tablelands, provides a climatic and physical barrier that helps to maintain moisture levels along the coast and restrict the distribution and movements of certain natural hosts such as waterbirds.

On the coast, rainfall is more consistent and mosquito activity more regular. Tidal inundation also promotes breeding of mosquitoes in the saltmarshes. A combination of high tides and heavy rainfall has resulted in some of the largest outbreaks in the State. This includes the Barmah Forest virus epidemics of 1995 (south coast) and 2001 (mid-north coast),[1,2] and the combined Ross River virus and Barmah Forest virus epidemic of 2003 (northern rivers).[3] Freshwater breeding mosquitoes may breed in large numbers after rain, with arbovirus activity ensuing. The Ross River virus outbreaks of 1996 (northern rivers) and 1997 (western Sydney),[4,5] and the Barmah Forest virus cases in 2002 (western Sydney),[6] were all probably the result of transmission via freshwater mosquitoes.

Cities, particularly Sydney, have lost large areas of natural habitat along with the native fauna. The lack of hosts means that there is little urban arbovirus transmission, except on the outskirts of the city. Thus, most notifications from Sydney (except some from the outskirts) have been acquired elsewhere.

ARBOVIRUS SURVEILLANCE IN NEW SOUTH WALES

The methods employed for monitoring arbovirus activity within the State include mosquito surveillance, the use of sentinel animals, and the notification of human disease. Mosquito populations are routinely monitored at up to 30 locations across the State, through the months of November to April, in order to detect unusual densities that may indicate increased arbovirus activity. At inland monitoring locations, the mosquitoes are also tested for the presence of virus. Sentinel chickens located at inland locations are bled weekly during the mosquito season to detect the transmission of Murray Valley encephalitis virus and Kunjin virus. NSW Health funds these activities and the results are publicly available on the NSW Arbovirus Surveillance and Mosquito Monitoring Program website at **www.arbovirus.health.nsw.gov.au**.

Human infectious diseases are reported to the NSW Department of Health's Notifiable Diseases Database, with most arbovirus cases notified between December and the following May. However, information derived from this database (such as in Tables 1–2 and Figures 1–4) does have some limitations. There is no distinction between presumptive cases (single positive IgM serology) and confirmed cases (fourfold or greater increase in antibody titre between acute and convalescent sera), while the patient location is recorded as the residential address,

TABLE 1

NOTIFICATIONS OF MOSQUITO-BORNE DISEASES, NEW SOUTH WALES, 1995–96 TO 2002–03

Year	1995–96	1996–97	1997–98	1998–99	1999–00	2000–01	2001–02	2002–03	Total
Ross River virus	939	1537	344	1211	736	773	218	456	6214
Barmah Forest virus	155	188	118	242	188	375	404	428	2098
Sindbis virus	0	0	0	3	3	4	7	7	24
Murray Valley Encephalitis virus[1]	0	0	1	0	0	0	0	0	1
Kunjin virus	1	0	0	0	0	1	0	0	2
Kokobera virus	0	0	1	0	0	0	0	0	1
Dengue viruses[2]	15	18	36	32	23	25	66	73	288
Arbovirus notifications not otherwise specified	4	2	0	1	1	3	1	0	12
Malaria[2]	133	191	163	160	205	175	147	100	1274
Total	**1247**	**1936**	**663**	**1649**	**1156**	**1356**	**843**	**1064**	**9914**

1 The 1 case of Murray Valley Encephalitis virus was presumed to be acquired outside of NSW.

2 Both Dengue and Malaria are acquired outside of NSW.

Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health; and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health.

TABLE 2

NOTIFICATIONS AND RATES OF ROSS RIVER VIRUS DISEASE AND BARMAH FOREST VIRUS DISEASE BY AREA HEALTH SERVICE GROUPED ACCORDING TO VIROGEOGRAPHIC REGION, NEW SOUTH WALES, JANUARY 1995 TO FEBRUARY 2004

	Area health service	No. RRV disease cases	Crude rate per 100,000 per annum	No. BFV disease cases	Crude rate per 100,000 per annum
Sydney	CS	41	0.9	9	0.2
	NS	148	2.2	15	0.2
	WS	109	1.8	11	0.2
	WEN	181	6.5	8	0.3
	SWS	70	1.0	7	0.1
	SES	79	1.2	14	0.2
Coastal	NR	998	42.9	747	31.8
	MNC	803	34.8	984	42.1
	HUN	785	16.4	167	3.4
	CC	316	12.6	36	1.3
	ILL	275	9.0	114	3.7
	SA	230	14.1	218	13.5
Inland	NE	527	33.1	47	3.0
	MAC	440	47.5	18	1.9
	MW	202	13.5	10	0.7
	GM	928	40.3	51	2.2
	FW	391	88.5	35	8.0
All Sydney		**628**	**1.9**	**64**	**0.2**
All Coastal		**3,407**	**20.4**	**2,266**	**13.5**
All Inland		**2,488**	**31.8**	**161**	**2.4**

CS = Central Sydney, NS = Northern Sydney, WS = Western Sydney, WEN = Wentworth, SWS = South West Sydney, SES = South Eastern Sydney, NR = Northern Rivers, MNC = Mid-North Coast, HUN = Hunter, CC = Central Coast, ILL = Illawarra, SA = Southern Area, NE = New England, MAC = Macquarie, MW = MidWest, GM = Greater Murray, FW = Far West

RRV = Ross River virus, BFV = Barmah Forest virus

Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health; and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health.

which may not be where the infection occurred. It is likely that the latter information is more reliable in country areas where the viruses are endemic and most cases occur. The reported notification date is either the date of disease onset or the date of specimen collection, whichever is earlier. As the date of onset is not recorded in the vast majority of notifications (as case follow-up would be required to establish disease onset), the specimen collection date is mostly used. The incubation period of most arboviruses averages 7–10 days. Assuming a further delay of 5 days before the patient consults their general practitioner and has blood taken for testing, the notification date can be 2 weeks or longer after the patient was bitten by the vector mosquito.

NOTIFICATIONS OF MOSQUITO-BORNE DISEASE IN NEW SOUTH WALES

Table 1 lists the notifications of mosquito-borne diseases in New South Wales for the financial years July 1995 to June 2003. The dengue and malaria notifications are included to demonstrate the significant risks to the traveller. Excluding the latter 2, there were a total of 8,352 cases of arbovirus infections notified during this period of 8 seasons, with an average of 1,044 cases per season. A brief discussion of the 3 most significant locally-transmitted arboviruses follows.

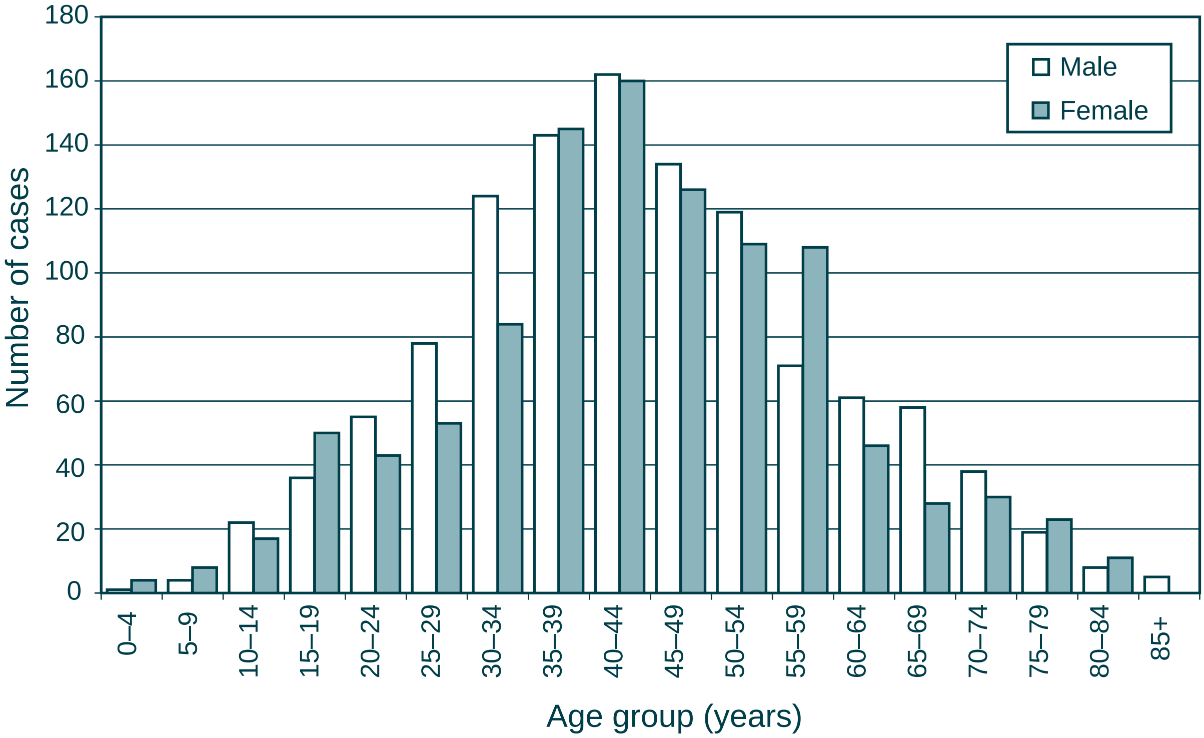
Ross River virus

Ross River virus is the most common arbovirus to infect humans within Australia. The disease is typified by rash, fever, arthralgia and arthritis. The disease occurs in all states, although notification rates are greater in the northern states. In New South Wales, there were 6,214 cases from July 1995 to June 2003 (Table 1). The virus is endemic in both coastal and inland regions, with the occasional disease outbreak in western Sydney.[5,7] Within the State, the north coast produces the greatest number of cases (Table 2), although the far west has the highest notification rates; generally the more rural the area the greater the number of cases and the higher the rate. The majority of cases occur in people aged 20 to 60 (Figure 1), with no significant difference between the sexes.

Notifications of Ross River virus cases peak very late in the season (Figure 2), with many still being reported in May when mosquito populations are well on the decline. It is difficult to determine the reason for this, but perhaps with the declining numbers people become less vigilant in their personal protection measures against mosquitoes.

FIGURE 1

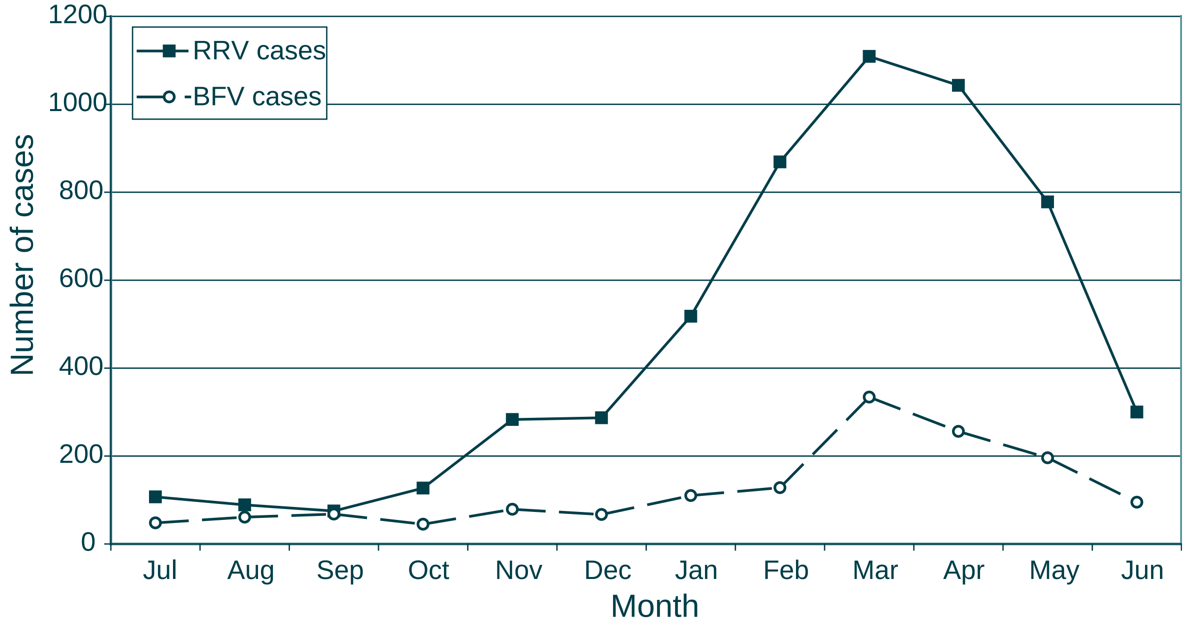
ROSS RIVER VIRUS DISEASE NOTIFICATIONS BY AGE AND SEX, NEW SOUTH WALES, JANUARY 2000 TO FEBRUARY 2004



Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health; and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health.

FIGURE 2

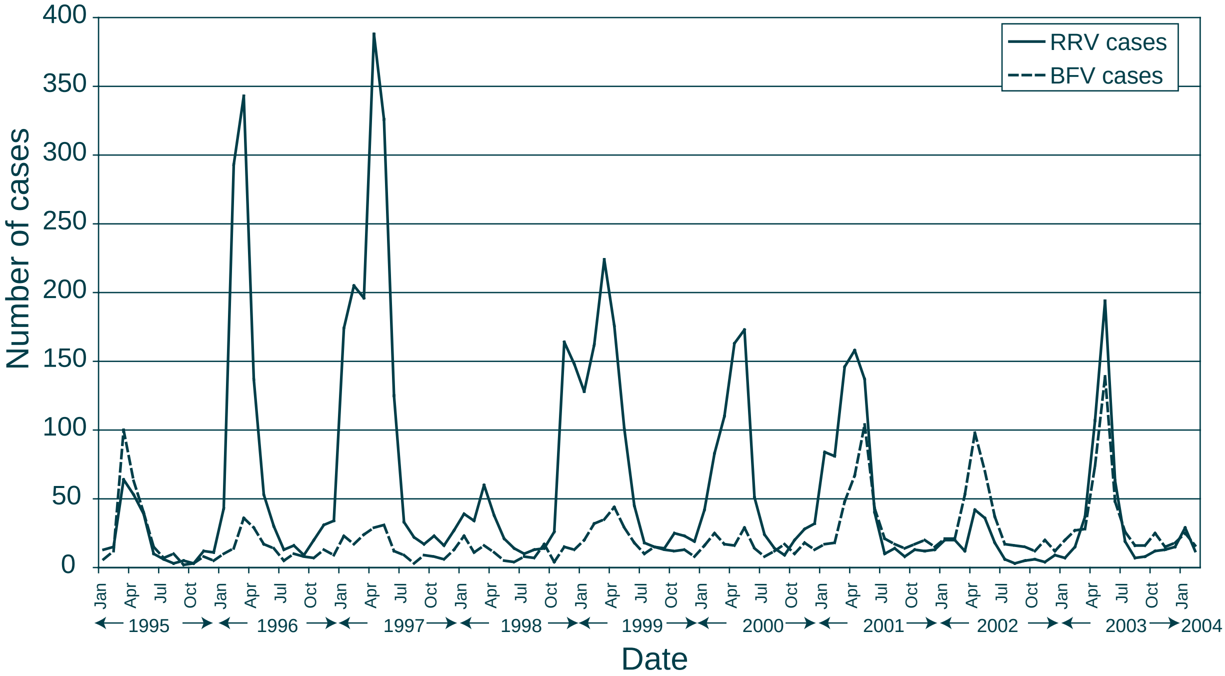
NOTIFICATIONS OF ROSS RIVER VIRUS DISEASE AND BARMAH FOREST VIRUS DISEASE BY MONTH, NEW SOUTH WALES, JANUARY 1995 TO FEBRUARY 2004



Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health; and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health.

FIGURE 3

SEASONAL NOTIFICATIONS OF ROSS RIVER VIRUS DISEASE AND BARMAH FOREST VIRUS DISEASE, NEW SOUTH WALES, JANUARY 1995 TO FEBRUARY 2004



Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health; and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health.

Activity of the virus and the number of Ross River virus cases is quite variable from season to season (Figure 3) and for rural areas there is annual endemic activity. Major outbreaks are associated with extreme rainfall, such as the 1983–84 epidemic, which was widespread across the inland with 1,196 cases.[8] During outbreaks, notification rates can be extraordinarily high. During March 1996, rates of 1006.2 per 100,000 were recorded in the Far West Area Health Service. Occasionally outbreaks can be very localised and intense. The outbreak in western Sydney during 1997 resulted in 69 cases over a small geographic area.[5] It would appear that these types of outbreaks occur after a considerable time of absence of local activity.

Barmah Forest virus

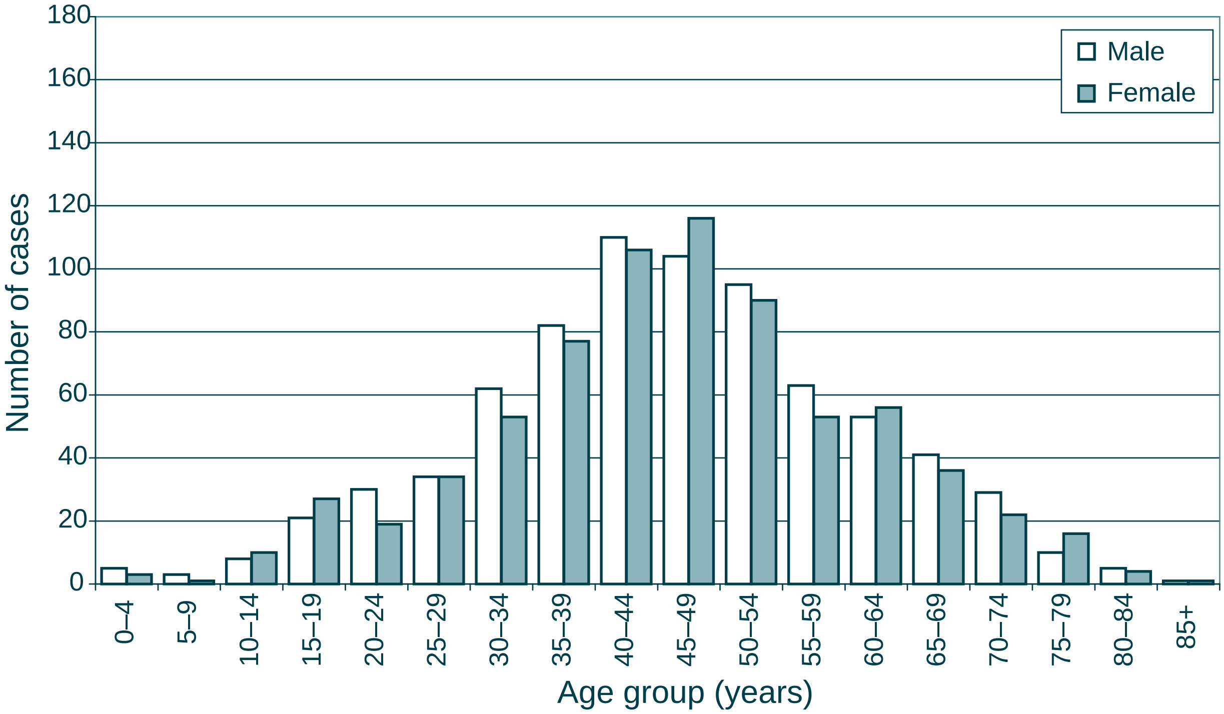
Barmah Forest virus shows many similarities to Ross River virus: similar disease symptoms (albeit less prolonged), similar seasonal activity (Figure 2, but note this is unlike Queensland, which has a secondary peak of Barmah Forest virus in November),[9] similar age group affected (Figure 4), and similar male-to-female disease ratio (Figure 4). The big difference for New South Wales is that Barmah Forest virus appears to be largely confined to the coastal region. Most notifications are from the coast (Table 2) and only 1 instance of Barmah Forest virus has been isolated from inland-trapped mosquitoes (collected from Menindee in 1993) and processed by the NSW Arbovirus Surveillance Program.[10] It is possible that many cases reported from the inland region were acquired from coastal districts. Why the virus is largely confined to this region is not known, but there is evidence to suggest that *Culex annulirostris*, the main inland arbovirus vector mosquito, is an inefficient vector of Barmah Forest virus.[11] Currently, the reservoir hosts (that is, the vertebrate hosts involved in endemic arbovirus cycles) are not known, but perhaps the distribution of these is helping to confine activity to the coast.

The seasonal (Figure 3) and spatial activity of Barmah Forest virus is highly variable. On the south coast, there are relatively few cases annually and the disease is largely epidemic in nature, with 1 large outbreak in 1995 with 135 cases.[1] Many more cases with higher notification rates occur along the north coast, and there have been large recent outbreaks over the 3 consecutive seasons of 2000–01 to 2002–03.[2,3,6]

Elsewhere in the country, Barmah Forest virus disease shows a similar trend, with most human cases occurring in coastal regions. Likewise, the disease tends to show epidemic patterns in most states.[1,12]

FIGURE 4

BARMAH FOREST VIRUS DISEASE NOTIFICATIONS BY AGE AND SEX, NEW SOUTH WALES, JANUARY 2000 TO FEBRUARY 2004



Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health; and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health.

Murray Valley encephalitis virus

Murray Valley encephalitis virus is one of the most important arboviruses as the disease has a high fatality rate and many survivors are left with severe permanent neurological damage. The virus is endemic in the northwest of Australia where activity occurs in most years.[13] For the southeast, Murray Valley encephalitis is epidemic, and previous disease activity has followed 2 wet years.[14] Past outbreaks in New South Wales have occurred in 1917 (70 cases), 1918 (49 cases), 1925 (10 cases), 1951 (10 cases), 1956 (3 cases) and 1974 (5 cases).[14] The last outbreak involved some 58 cases Australia-wide with 13 deaths, and the majority were from the Murray Valley. The cases within New South Wales were widely dispersed both temporally and spatially over a 10-week period from the first to the last, with cases from Albury in the east to Broken Hill in the west. In 2001, the virus was widely active along the Darling River,[2] however, no human disease cases were reported. More recently in late 2003, there was some activity at Menindee,[15] and again no cases were recognised.

POTENTIAL THREATS TO POPULATION HEALTH IN NEW SOUTH WALES

Since notifications began in 1991, New South Wales has experienced a period of exceptionally low rainfall, with the 1990s being the driest recorded decade. This suggests that arbovirus activity has been well below normal. A return to regular rainfall patterns may see a return to higher levels of activity in inland areas of the State, as there has been no epidemic Murray Valley encephalitis since 1974, and on the south coast, as there has been little arbovirus activity there since the 1995 Barmah Forest virus outbreak.

Physical changes to the environment through human endeavours may lead to more mosquitoes and arbovirus activity. For example, a recent trend has been to construct wetlands as a means of dealing with stormwater and wastewater. If these wetlands are not constructed to minimise vector breeding or not maintained appropriately, then mosquito production may become a significant issue, particularly for inland communities.[16] Likewise, the re-establishment of water flows to major river systems for environmental protection may result in increased flooding and enhanced disease activity.

As less land is available for development, especially along the coast, there is pressure on local councils to approve the building of residential or industrial estates close to problematic mosquito areas, especially saltmarshes. Adequate ‘buffer zones’ need to be defined to reduce the disease risk to the community.

A constant threat is the introduction of exotic vector mosquitoes, especially the dengue vector *Aedes albopictus*. If introduced, this species has the potential to become established in urban communities across most of southern Australia and dramatically extend the current dengue receptive zone. Government agencies must remain vigilant and adequately resourced to keep this species out and to eliminate it if introduced.

CONCLUSION

Mosquito-borne viruses pose a significant current and potential threat to the population health of New South Wales. Reduction in the burden of mosquito-borne disease can only come about through a concerted effort involving a multidisciplinary approach encompassing education, surveillance and mosquito control, and this challenge needs to be met not only by all levels of government but by the community has a whole. Current mosquito education programs target health warnings usually before and at the peak of mosquito breeding, yet most human cases appear to occur in the latter part of the season and health warnings should not be discontinued at this time.

ACKNOWLEDGEMENTS

All notification data were obtained from the Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health, and GODSEND (Graphical Online Data Surveillance and Evaluation of Notifiable Diseases), Centre for Epidemiology and Research, NSW Department of Health. Mr Mark Bartlett, Manager Surveillance, Communicable Diseases Branch, NSW Department of Health, provided the explanation of the surveillance data. Associate Professor Richard Russell, Department of Medical Entomology, Institute of Clinical Pathology and Medical Research, Westmead, critically reviewed the manuscript.

REFERENCES

1. Doggett SL, Russell RC, Clancy J, Haniotis J, Cloonan MJ. Barmah Forest virus epidemic on the south coast of New South Wales, Australia, 1994–1995: viruses, vectors, human cases, and environmental factors. *J Med Entomol* 1999; 36: 861–8.
2. Doggett S, Clancy J, Haniotis J, Russell RC, Hueston L, Marchetti M, et al. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program. 2000–2001 Annual Report*. Sydney: Department of Medical Entomology, Westmead, 2001.
3. Doggett S, Clancy J, Haniotis J, Russell RC, Hueston L, Marchetti M, et al. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program. 2002–2003 Annual Report*. Sydney: Department of Medical Entomology, Westmead, 2003.
4. Russell RC, Haniotis J, Doggett SL, Clancy J, Cloonan MJ. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program 1995–1996*. Sydney: Department of Medical Entomology, Westmead, 1996.
5. Amin J, Hueston L, Dwyer DE, Capon A. Ross River virus infection in the north-west outskirts of the Sydney basin. *Commun Dis Intell* 1998; 11: 101–2.
6. Doggett S, Clancy J, Haniotis J, Russell RC, Hueston L, Marchetti M, et al. *The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program. 2001–2002 Annual Report*. Sydney: Department of Medical Entomology, Westmead, 2002.

7. Brokenshire T, Symonds D, Reynolds R, Doggett S, Geary M, Russell R. A cluster of locally-acquired Ross River virus infections in outer Western Sydney. *N S W Public Health Bull* 2000; 11: 132–4.
8. Hawkes RA, Boughton CR, Naim HM, Stallman ND. A major outbreak of epidemic polyarthritis in New South Wales during the summer of 1983–1984. *Med J Aust* 1985; 143: 330–33.
9. Hills SL, Sheriden JW. The epidemiology of Barmah Forest virus in Queensland. *Arbo Res Aust* 1997; 7: 95–99.
10. Russell RC, Cloonan MJ, Doggett SL, Clancy J, Haniotis J, Wells P, et al. Surveillance of arboviruses and vectors in NSW, 1993–1996. *Arbo Res Aust* 1997; 7: 228–34.
11. Ryan PA, Kay BH. Vector competence of mosquitoes (Diptera: Culicidae) from Maroochy Shire, Australia, for Barmah Forest virus. *J Med Entomol* 1999; 36: 856–60.
12. Passmore J, O'Grady KA, Moran R, Wishart W. An outbreak of Barmah Forest virus disease in Victoria. *Commun Dis Intell* 2002; 26: 600–4.
13. Broom A, Sturrock K, vanHeuzen B, Lindsay M, Smith D. Seroconversions in sentinel chickens provide an early warning of Murray Valley Encephalitis virus activity in Western Australia. *Arbo Res Aust* 2001; 8: 43–7.
14. Forbes JA. *Murray Valley encephalitis 1974: also the epidemic variance since 1914 and predisposing rainfall patterns*. Sydney: Australasian Medical Publishing Company, 1978.
15. The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program website. Menindee Results. Available at www.arbovirus.health.nsw.gov.au/areas/arbovirus/results/menindee/menindee.htm. Accessed February 2004.
16. Russell RC. Constructed wetlands in Australia: concerns and constraints, compromises and complements for effective mosquito management. *Arbo Res Aust* 2000; 8: 314–23.

RECENT INCREASES IN THE NOTIFICATION OF BARMAH FOREST VIRUS INFECTIONS IN NEW SOUTH WALES

Lara Harvey
NSW Public Health Officer Training Program
NSW Department of Health

Dominic Dwyer
Centre for Infectious Diseases and Microbiology Laboratory Services
Institute of Clinical Pathology and Medical Research, Westmead

Infection due to Barmah Forest virus (BFV) is an emerging problem in Australia,[1] with increased numbers of cases being reported.[2–5] BFV is a mosquito-borne arbovirus from the *Togaviridae* family. The virus was first isolated in 1974 from the Barmah State Forest in the Murray Valley region of the Victoria–New South Wales border,[6] and was first shown to be pathogenic to humans in 1988.[7] Symptoms of acute human infection may include rash, arthralgia, myalgia, lethargy and fever,[3–5,8] and are similar to symptoms caused by Ross River virus infection. However, rash is more common and florid, and joint disease is less severe, in BFV disease than in Ross River virus disease.[9] In a study of BFV cases on the mid-north coast of New South Wales,[3] over half of all cases reported time off work and an illness that lasted more than 6 months. BFV disease is therefore associated with a significant burden of illness and is of public health concern. This article describes trends in the notification rates for BFV disease in New South Wales since it was made notifiable in 1991.

METHODS

Under the *NSW Public Health Act 1991*, all laboratories must notify suspected cases of BFV infection to the local public health unit. The case definition for a suspected case is a person in whom there are demonstrated specific IgM antibodies to BFV in cerebral spinal fluid or in serum collected within 14 days of onset of symptoms.[10] Public health unit staff record case details on a confidential statewide database. All cases notified from 1991 to 2003 were geocoded and entered into MapInfo Professional version 7.0 software,[11] to highlight geographical location of the disease. Only cases notified between 1995 and 2003 were used in the analysis of case characteristics, because of the probability of underreporting and poor data quality in earlier years.[12] Incidence rates were calculated using the average of the estimated mid-year population for each of the years 1995 to 2003. National data was obtained from the National Notifiable Diseases Surveillance System,[13] which is available on the Australian Government Department of Health and Ageing website at **www.cda.gov.au/surveil**.

RESULTS

For the period 1991–2003 there were 2,527 notifications of BFV infection in New South Wales residents. Before 1995, there were few BFV notifications in the State each year, with 6 cases in 1991, 6 cases in 1992, 25 cases in 1993, and 40 cases in 1994.

In 1995, the number of notifications increased to 271. Of these cases, 122 were resident in the Southern Area Health Service, with 30 per cent of these living in Batemans Bay.[2]

Between 1995 and 2000, there has been continuous BFV activity reported on the north coast of New South Wales, and in the Mid North Coast and Northern Rivers Area Health Services. In 1999, there was a small increase in number of cases reported on the south coast in the Illawarra and Southern Area Health Services.

The numbers of notifications rose again in 2001 (402 cases), mainly involving residents of the Mid North Coast and Northern Rivers Area Health Services (Table 1). Since 2001, the number of notifications of BFV infection has been steadily increasing, with 309 cases reported in the Northern Rivers Area Health Service and 303 cases in the Mid North Coast Area Health Service to the end of 2003. There was a large increase in notifications from the Hunter Area Health Service in 2002 (101 cases).

For the period 1995 to 2003, the average annual incidence of BFV infection was 4.2 per 100,000 persons in New South Wales. BFV infection is predominantly rural in distribution, with the average annual incidence in rural health areas being 9.9/100,000 compared with 0.2/100,000 in the metropolitan health areas. The highest incident rates were reported from Southern Area Health Service (68.8 /100,000) in 1995, the Mid North Coast Area Health Service (81.7/100,000) in 2001, and the Northern Rivers Area Health Service (96.4/100,000) in 2003 (Table 1).

There is a coastal distribution of cases, with the majority of cases restricted to regions east of the Great Dividing Range as shown in Figure 1. However, despite the coastal predominance, there were reported cases in the Far West

TABLE 1

BARMAH FOREST VIRUS INFECTION, NUMBER OF NOTIFICATIONS AND INCIDENCE RATES PER 100,000 PERSONS BY AREA HEALTH SERVICE OF RESIDENCE, NEW SOUTH WALES, 1996–2003

Health area		1995	1996	1997	1998	1999	2000	2001	2002	2003	Total
CSA	No.	4	0	1	0	0	1	0	1	2	9
	Rate	0.9	0	0.2	0	0	0.2	0	0.2	0.4	
NSA	No.	2	2	3	2	2	0	1	2	1	15
	Rate	0.3	0.3	0.4	0.3	0.3	0	0.1	0.3	0.1	
SES	No.	1	1	7	0	1	3	0	0	1	14
	Rate	0.1	0.1	0.9	0	0.1	0.4	0	0	0.1	
WSA	No.	1	1	1	0	1	1	1	2	3	11
	Rate	0.2	0.2	0.2	0	0.1	0.1	0.1	0.3	0.4	
SWS	No.	0	1	0	0	2	0	2	2	0	7
	Rate	0	0.1	0	0	0.3	0	0.3	0.2	0	
WEN	No.	0	1	1	2	0	0	1	2	1	8
	Rate	0	0.3	0.3	0.6	0	0	0.3	0.6	0.3	
CCA	No.	0	1	0	0	3	3	1	21	7	36
	Rate	0	0.4	0	0	1	1	0.3	6.9	2.3	
ILL	No.	12	2	7	7	37	15	20	8	4	112
	Rate	3.6	0.6	2.1	2.1	10.7	4.3	5.7	2.3	1.1	
HUN	No.	2	1	4	7	11	12	8	101	20	166
	Rate	0.4	0.2	0.8	1.3	2.1	2.2	1.5	18.5	3.7	
SA	No.	122	4	2	3	27	10	31	9	9	217
	Rate	68.8	2.2	1.1	1.7	14.9	5.5	16.7	4.8	4.8	
GMA	No.	2	1	9	7	16	4	3	3	3	48
	Rate	0.8	0.4	3.5	2.7	6.3	1.6	1.2	1.2	1.2	
NEA	No.	3	5	5	1	7	4	5	7	8	45
	Rate	1.7	2.8	2.8	0.6	4	2.3	2.9	4	4.6	
MWA	No.	0	0	2	0	2	2	1	0	3	10
	Rate	0	0	1.2	0	1.2	1.2	0.6	0	1.8	
FWA	No.	0	3	4	9	2	3	7	5	2	35
	Rate.	0	6	8.1	18.4	4.1	6.3	14.5	10.4	4.2	
MAC	No.	3	1	2	2	1	3	3	2	1	18
	Rate	2.9	1	1.9	1.9	1	2.9	2.9	1.9	0.9	
MNC	No.	71	61	96	50	79	94	216	181	122	970
	Rate	29	24.6	38.2	19.6	30.7	36.1	81.7	67.7	45.1	
NRA	No.	48	87	40	44	58	40	102	45	264	728
	Rate	19.6	35.1	15.9	17.3	22.6	15.4	38.3	16.7	96.4	
Total		**271**	**172**	**184**	**134**	**249**	**195**	**402**	**391**	**451**	**2449**

CSA = Central Sydney Area, SWS = South Western Sydney Area, HUN = Hunter Area, MWA = Mid Western Area, NRA = Northern Rivers Area, NSA = Northern Sydney Area, WEN = Wentworth Area, SA = Southern Area, FWA = Far West Area, SES = South Eastern Sydney Area, CCA = Central Coast Area, GMA= Greater Murray Area, MAC = Macquarie Area, WSA = Western Sydney Area, ILL = Illawarra Area, NEA = New England Area, MNC = North Coast Area

Source: Graphical Online Data Surveillance and Evaluation for Notifiable Diseases (GODSEND). Communicable Diseases Branch, NSW Department of Health. Accessed 15 June 2004.

Area Health Service in 1998 (18.4/100,000), 2001 (14.5/100,000), and 2002 (10.4/100,000).

A seasonal variation is evident from Figure 2, with the most common season of illness being late summer to early autumn.

The age range for BFV disease cases was 2 months to 98 years, with the median age group being 45–49 years. Slightly more male (51 per cent) than female cases were reported (Figure 3).

At the national level, there were 7,518 notifications from 1995 to 2003. Fifty-seven per cent of these notifications were from Queensland, 32 per cent from New South Wales, 5 per cent from Western Australia, 3 per cent from Victoria, and 3 per cent from the Northern Territory (Figure 4).

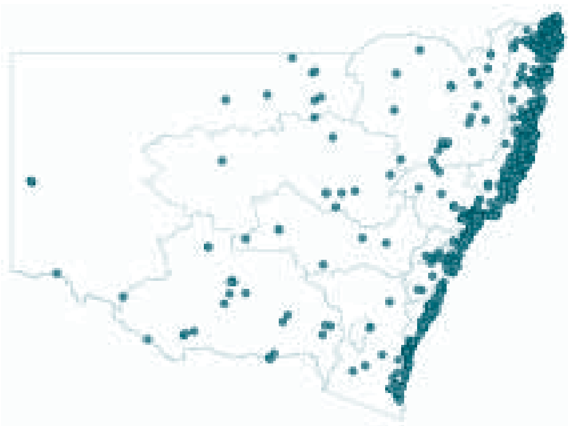
DISCUSSION

Annual notifications of BFV infection in New South Wales have increased from 6 in 1991 to 451 in 2003. The first reported major outbreak of human disease resulting from BFV infection occurred in 1995 on the south coast, with the focus of activity around Batemans Bay. In total, 135 cases were identified from this outbreak. There was little BFV activity reported on the south coast after 1995. Since 2001, the majority of notifications have been in people resident on the north coast. Thus the distribution of notified cases in the state is predominantly coastal, a finding that is supported by serosurveys undertaken in the mid-1980s, which showed that BFV antibodies were highest in residents of coastal areas.[14,15]

While previous reports have described coastal activity, there is some indication of inland rural BFV activity, with

FIGURE 1

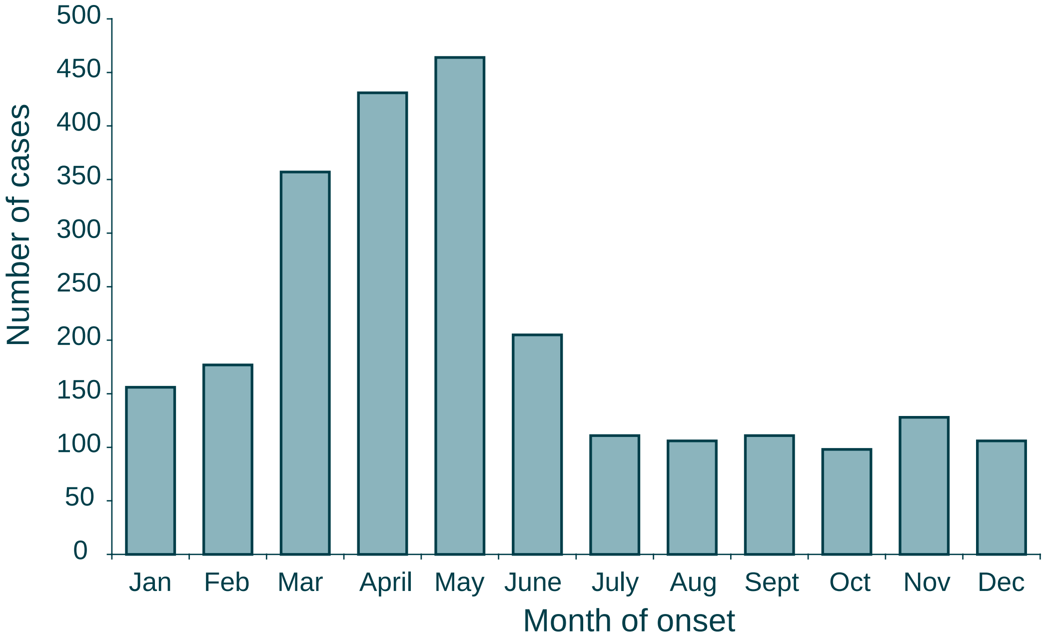
DISTRIBUTION OF CASES OF BARMAH FOREST VIRUS INFECTION, NEW SOUTH WALES, 1991–2003.



Source: Notifiable Diseases Database (HOIST). Communicable Diseases Branch, NSW Department of Health.

FIGURE 2

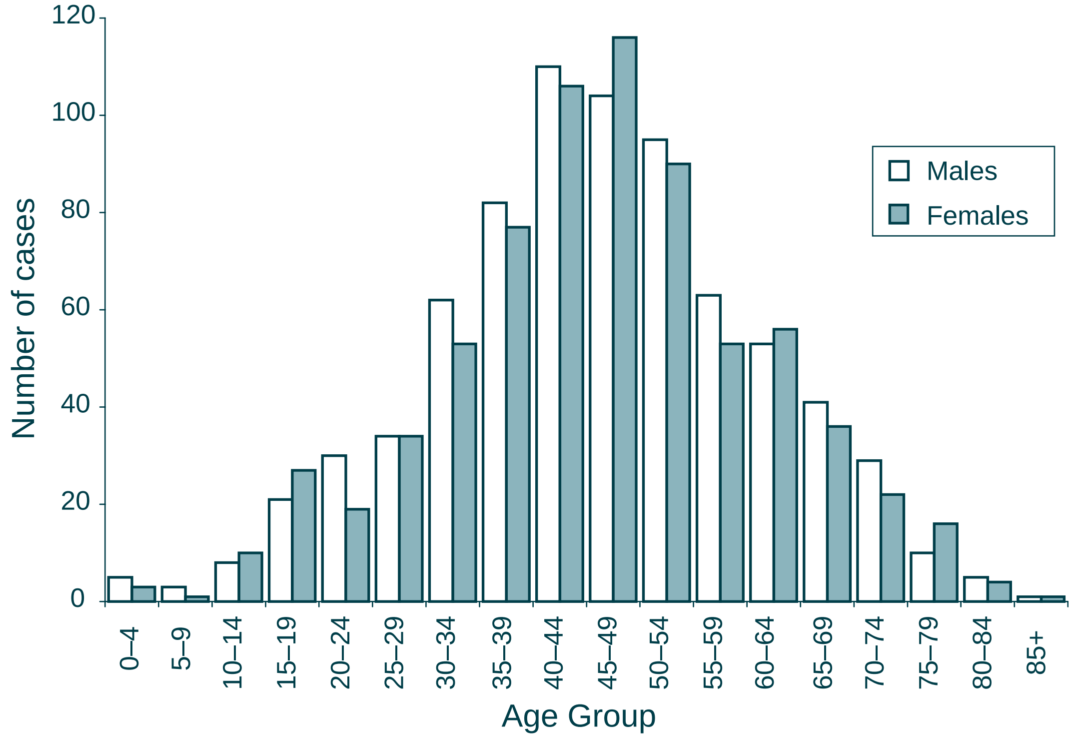
BARMAH FOREST VIRUS INFECTION, ESTIMATED MONTH OF ONSET OF ILLNESS, NEW SOUTH WALES, 1995–2003



Source: Graphical Online Data Surveillance and Evaluation for Notifiable Diseases (GODSEND). Communicable Diseases Branch, NSW Department of Health.

FIGURE 3

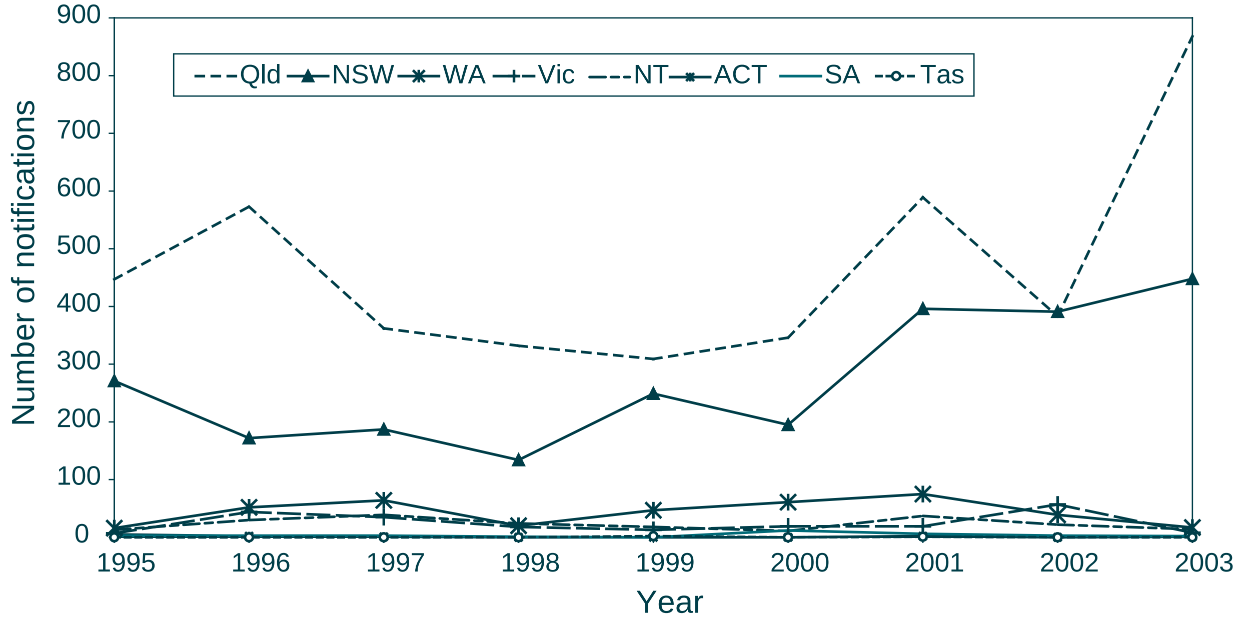
BARMAH FOREST VIRUS INFECTION, NOTIFICATIONS BY SEX AND AGE, NEW SOUTH WALES, 1995–2003



Source: Graphical Online Data Surveillance and Evaluation for Notifiable Diseases (GODSEND). Communicable Diseases Branch, NSW Department of Health.

FIGURE 4

NOTIFICATIONS OF BARMAH FOREST VIRUS INFECTIONS IN AUSTRALIAN STATES AND TERRITORIES 1995–2003



Source: National Notifiable Diseases Surveillance System, Australian Government Department of Health and Ageing.

notifications being recorded in the Far West Area Health Service. While inland residents may have contracted the infection when visiting coastal areas, there is previous evidence of local vector activity in the inland region, notably a report in 1993 of the identification of BFV isolated from a *Culex annulirostris* mosquito trapped in Menindee.[9]

On a national level, notifications also rose between 1995 and 2003. There is no obvious annual pattern in notification numbers between the states. In 1996, there was an increase in notifications in Queensland without a corresponding increase in New South Wales. There was an increase in notifications in 2001 in both states, but this was not sustained in Queensland in 2002.

It is difficult to separate the factors that may have contributed to the increase in notifications. Is it a true reflection of increase in virus activity, or does it reflect increased use of BFV-specific serological assays, or increased recognition of clinical disease by doctors, or increased media interest and public awareness of the disease? As the current case definition of suspected BFV infection is based on a single positive BFV-specific IgM, there may be false positive results from commercial assays or inadequate differentiation from other alphaviruses. Ideally, a single positive BFV-specific IgM should be confirmed by another assay or evidence of BFV-specific IgG seroconversion on a convalescent serum sample. On the other hand, early serological testing of suspected clinical cases may be negative, as BFV seroconversion can be slow.

Unlike Ross River virus, relatively little is known about the natural cycle of BFV. The virus is mosquito-borne, and laboratory studies have shown that the saltmarsh mosquitoes *Ochlerotatus vigilax*, *Oc. camptorhynchus*, *Verrallina funerea* and *Coquillettida linealis* and the freshwater mosquitoes *Oc. notoscriptus*, *Oc. procax* and *Oc. multiplex* are efficient vectors of BFV.[1] *Culex annulirostris* (freshwater) has been shown to be a possible but inefficient vector.[16] Increased numbers of *Oc. vigilax* and *Oc. camptorhynchus* in Western Australia, *Oc. camptorhynchus* in Victoria and *Oc. vigilax* in New South Wales have been associated with outbreaks of human disease. Why the BFV cases remain predominantly coastal in distribution when suitable vector habitats occur inland remains unanswered.

Similarly, the reservoir of BFV remains unknown. The reservoirs for Ross River virus include macropods,[17] possums,[18] and horses.[17] Limited serological testing has not found evidence of BFV antibodies in possums and horses. There is some evidence that *Macropus giganteus* (kangaroo) and *Phascolarctos cinereus* (koala),[19] waterbirds,[20] and cows,[17] have detectable BFV antibodies and therefore may be potential reservoirs for the virus. Flying foxes have been implicated in the transmission of other viruses in Australia (henipavirus, Australian bat lyssaviruses). Given their coastal distribution they may be implicated in BFV transmission, but this requires further study.

CONCLUSION

Notifications of BFV infection have increased both nationally and in New South Wales, particularly in the coastal regions of northern parts of the State in the last 3 years. Residents and visitors to the northern coastal areas need to be aware of the importance of taking precautions against mosquito bites. Serosurveys of the human population may be indicated, to determine whether the increase in notifications is either a true reflection of increasing incidence of BFV infection, or reflects the increased awareness of and capacity for testing for the virus. Similarly, as relatively little is known of the natural cycle of BFV, serosurveys of potential reservoir hosts may provide valuable insight to other regions of potential outbreaks.

ACKNOWLEDGEMENTS

The authors would like to thank Stephen Doggett and Linda Hueston at the Institute of Clinical Pathology and Medical Research for mosquito and laboratory information, and Alan Willmore from the Centre of Epidemiology and Research, NSW Department of Health, for his invaluable assistance with the MapInfo software.

REFERENCES

1. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000; 2: 1693–1704.
2. Doggett SL, Russell RC, Clancy J, Haniotis J, Cloonan MJ. Barmah Forest virus epidemic on the south coast of New South Wales, Australia, 1994–1995: viruses, vectors, human cases and environmental factors. *J Med Entomol* 1999; 36(6): 861–868.
3. Beard JR, Trent M, Sam GA, Delpech VC. Self reported morbidity of Barmah Forest virus infection on the north coast of New South Wales. *Med J Aust* 1997; 167: 525–528.
4. Lindsay M, Johansen C, Broom AK, Smith DW, Mackenzie JS. Emergence of Barmah Forest virus in Western Australia. *Emerg Infect Dis* 1995; 1(1): 22–26.
5. Passmore J, O'Grady KA, Moran R, Wishart E. An outbreak of Barmah Forest virus disease in Victoria. *Commun Dis Intell* 2002; 26(4): 600–604.
6. Marshall ID, Woodroofe GM, Hirsch S. Viruses recovered from mosquitoes and wildlife serum collected in the Murray Valley of south-eastern Australia February 1974, during an epidemic of encephalitis. *Aust J Exp Biol Med Sci* 1982; 60: 457–470.
7. Boughton CR, Hawkes RA, Naim HM. Illness caused by Barmah Forest like virus in New South Wales. *Med J Aust* 1988; 148: 146–147.
8. Phillips DA, Murray JR, Aaskov JG, Wiemers MA. Clinical and subclinical Barmah Forest virus infection in Queensland. *Med J Aust* 152: 463–466.
9. Flexman JP, Smith DW, Mackenzie JS, Fraser JR, Bass SP, Hueston L, et al. A comparison of the diseases caused by

Ross River virus and Barmah Forest virus. *Med J Aust* 1998; 169: 159–163.

10. Communicable Diseases Surveillance and Control Unit. *Notifiable Disease Manual*. Sydney: NSW Department of Health, 2002.
11. MapInfo Professional Version 7.0. MapInfo Australia.
12. Muscatello D, McAnulty J. Arboviruses in NSW 1991 to 1999. *N S W Public Health Bull* 11(11): 190–192.
13. Australian Government Department of Health and Ageing. National Notifiable Diseases Surveillance System. Available at www.cda.gov.au/surveil (accessed 19 February 2004).
14. Vale TG, Carter I, McPhie, James GS, Cloonan J. Human arbovirus infections along the south coast of New South Wales. *Aust J Exp Biol Med Sci* 1986; 64: 307–309.
15. Hawkes RA, Clement RB, Naim HM, Myrick BA, Ramsay LG. Barmah Forest virus infections in humans in New South Wales. *Med J Aust* 1987; 146(11): 569–573.
16. Boyd AM, Kay BH. Vector competence of *Aedes aegypti*, *Culex sitiens*, *Culex annulirostris* and *Culex quinquefasciatus* for Barmah Forest virus. *J Med Entomol* 1999; 36(4): 508–514.
17. Vale TG, Spratt DM, Cloonan MJ. Serological evidence of arbovirus infection in native and domesticated mammals on the south coast of New South Wales. *Aust J Zool* 1991; 39: 1–7.
18. Boyd AM, Hall RA, Gemmell RT, Kat BH. Experimental infection of Australian brushtail possums, *Trichosurus* (Phalangeridae: Marsupialia), with Ross River and Barmah Forest viruses by use of natural mosquito vector system. *Am J Trop Med Hyg* 2001; 65(6): 777–782.
19. Aldred J, Campbell J, Mitchell G, Davis G, Elliott J. Involvement of wildlife in the natural cycle of Ross River and Barmah Forest viruses. *Proceedings from Wildlife Diseases Association Meeting 1991* (unpublished).
20. Russell R. Arboviruses and their vectors in Australia: an update on the ecology and epidemiology of some mosquito borne arboviruses. *Rev Med Vet Entomol* 1995; 83(4): 141–143.

THE INCREASE IN PRESENTATIONS OF DENGUE FEVER IN NEW SOUTH WALES

Linda Hueston
Centre for Infectious Diseases and Microbiology
Institute of Clinical Pathology and Medical Research, Westmead

BACKGROUND

The earliest known reports of dengue fever, a mosquito-borne disease, are from China in 992 AD.[1] During the 18th and 19th centuries, both the slave trade and increases in shipping and commercial trade saw the disease spread throughout the world via sailing ships.[1,2] This spread was largely due to the water supplies stored on board ships, which provided an effective means of travel for the virus and vector that cause dengue fever.

The earliest record of dengue fever in Australia is 1873, when 8 cases occurred in Sydney, imported from a ship from Mauritius.[3] The last epidemic in New South Wales was between 1942 and 1944, and is attributed to troop movements by steam train.[4] While epidemics of dengue fever have been documented in Queensland, New South Wales, Western Australia and the Northern Territory, it is unlikely that dengue fever has remained endemic between these epidemics.[5] It is more likely that dengue fever was, and continues to be, reintroduced by tourists or residents returning from overseas countries where dengue fever is endemic.[4,6] Since 1944, epidemics have been confined to those areas of Queensland that correspond to the geographic range of the vector mosquito *Aedes aegypti*.[7] This confinement may be due to the introduction of reticulated water supplies and the reduction of breeding sites, the combined effect of which has seen the reduction and eradication of the vector mosquito in some areas.[4] Since 1944, all cases of dengue fever in New South Wales but one have been acquired in Queensland or overseas. The one exception was an infection acquired by a biomedical engineer working with live viruses in the production of diagnostic kits.

In 1991 dengue fever became a notifiable disease in New South Wales. Since then all new laboratory notifications are entered into the NSW Notifiable Diseases Database (NDD), maintained by the Communicable Diseases Branch, NSW Department of Health, and are accessed through the Graphical Online Data Surveillance Evaluation for Notifiable Diseases (GODSEND), maintained by the Centre for Epidemiology and Research, NSW Department of Health. A review of the NDD has shown an increase in the number of notifications of dengue fever over the last 5 years. The Arbovirus and Emerging Diseases Unit, Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research (Westmead), undertakes a large proportion of dengue virus testing for New South Wales. We have noticed an increase in requests for dengue serology and also an increase in the number of positive notifications between 1999 and 2003. This article describes the pattern of requests and the clinical and travel histories of cases notified through our laboratory, and discusses how these findings relate to the apparent increase in notifications of dengue fever in New South Wales.

METHODS

In New South Wales, a case of dengue fever is defined according to national guidelines.[8] The majority of notifications of dengue fever are serologically determined, usually on the basis of a single IgM positive result.

Our laboratory defines a primary case of dengue fever as one in which IgG and IgM are negative on acute phase samples but positive on the convalescent phase sample. Alternatively, a primary case can be defined where IgG is negative, IgM is positive on an acute phase sample and where there is evidence of IgG seroconversion in the convalescent phase sample. We define a secondary case of dengue fever as one where IgG is positive but IgM is negative on an acute sample and which demonstrates a fourfold or greater rise in IgG titre with or without the presence of IgM. In addition to the serology findings, the case must have a consistent clinical and travel history.

Our interest in the reasons for the increase in both requests for serology and dengue fever infections led us to develop a questionnaire to obtain more information on notified cases from the physician they attended for treatment.

RESULTS

Figure 1 shows the increasing trend in notifications of dengue fever reported to NSW Health between 1999 and 2003.[9] This figure does not include secondary infections without IgM or unspecified flavivirus infections (most of which would be dengue, based on travel and clinical history) reported to the NDD. The data may therefore represent an underestimate of case numbers.

Our laboratory has noted a 30 per cent increase in the number of requests for dengue serology over the last 5 years (Table 1).

In 2003, we diagnosed 111 cases of primary dengue fever. In 1999 and 2000 only primary infections were diagnosed. In 2001 we began to see cases of secondary infection (1 case), in 2002 there were 2 cases of secondary infection, and in 2003 there were 9 cases.

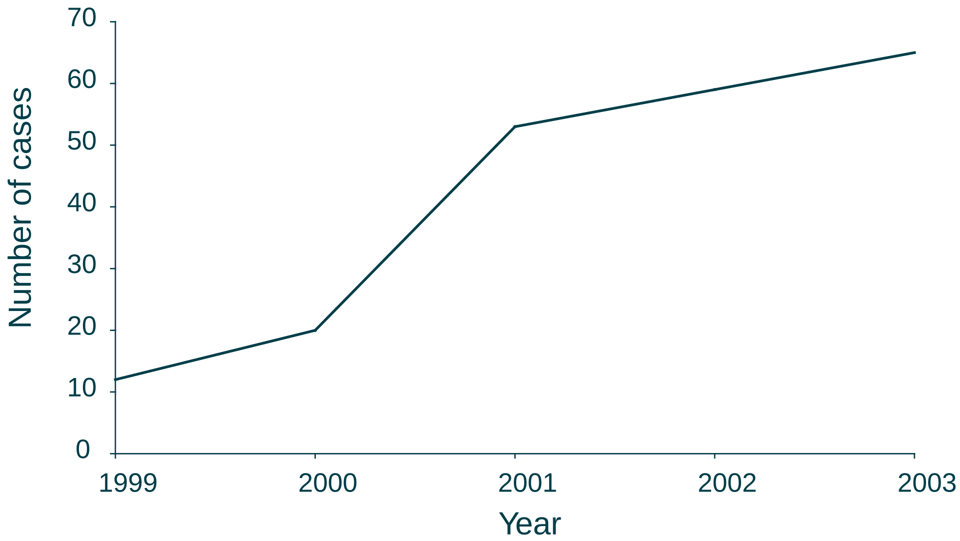
In a follow-up of 100 serology requests that originated in New South Wales, we used the questionnaire for attending physicians shown in Table 2, from which the following information was obtained.

The clinical presentation of dengue fever was broad, ranging from mild flu-like illness through to haemorrhagic symptoms and moderate liver involvement. Most patients presented within 5–7 days of onset. All cases had histories of overseas travel. Destinations included Thailand, Malaysia, Indonesia, Korea, India, Sri Lanka, Timor, the Solomon Islands, Fiji, Vanuatu, Samoa, Tahiti, Noumea and New Caledonia. Only 5 cases were tourists visiting New South Wales from Asia (3 cases from Malaysia) and the Pacific (2 cases from Samoa). The remainder were residents of New South Wales.

General practitioners saw the majority of primary infections. Emergency departments were the next most

FIGURE 1

NOTIFICATIONS OF DENGUE FEVER, NEW SOUTH WALES, 1999–2003



Source: Notifiable Diseases Database, Communicable Diseases Branch, NSW Department of Health, accessed via the Graphical Online Data Surveillance Evaluation for Notifiable Diseases (GODSEND), Centre for Epidemiology and Research, NSW Department of Health.

TABLE 1

NUMBER OF SEROLOGY REQUESTS AND CASES OF PRIMARY AND SECONDARY INFECTION, DENGUE FEVER, NEW SOUTH WALES, 1999–2003

Year	Serology requests	Positive serology	Primary infection	Secondary infection
1999	700	67	67	0
2000	800	70	70	0
2001	850	81	80	1
2002	920	90	88	2
2003	1000	120	111	9

Source: Dengue Statistics Database, Arbovirus and Emerging Diseases Unit, Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research (Westmead).

TABLE 2

FOLLOW-UP QUESTIONNAIRE FOR PRACTITIONERS, NOTIFICATIONS OF DENGUE FEVER, NEW SOUTH WALES, 1999–2003

What was the clinical picture?
What was the onset date of symptoms?
Has there been any recent travel?
If yes specify places visited and travel dates.
Was the patient seen at an emergency room, medical clinic, or general practice?
Did the patient require hospitalisation? If yes what was the length of stay? Was intensive care required?
Is the patient resident in NSW?
Country of birth. If not Australia what was the patient's age on arrival in Australia?
Is the patient an overseas visitor?
What was the reason for testing?
Is follow-up testing possible?
Before this case how aware were you of dengue fever as a cause of infection?
Would you like to receive training material on dengue fever diagnosis?

Source: Arbovirus and Emerging Diseases Unit, Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research (Westmead).

common detection point for primary infections, particularly on weekends and holidays. The secondary infections were seen through emergency departments. Six secondary infections and 3 primary infections spent time as hospital inpatients. The duration of inpatient stay was 2–13 days. All secondary cases were residents who, before moving to Australia, had been born and lived for several years in countries where dengue fever was endemic.

Thirty per cent of laboratory requests included some clinical history, 2 per cent mentioned travel in the history but did not specify the travel destination, and none mentioned the date of onset. Twenty per cent of patients had received information during their overseas travel advising that dengue fever was active in the areas visited and that they should seek medical assistance if on returning home they developed symptoms compatible with dengue fever. The majority of patients were tested because they were clinically ill, although some patients requested testing because their travel companions had been diagnosed overseas. However, one common feature was that dengue fever was not rated highly in the differential diagnosis. Generally, dengue fever was considered after other possibilities were excluded, thereby delaying a diagnosis for up to 5 days.

Malaria was the most commonly suspected cause, followed by influenza, glandular fever and hepatitis. The majority of practitioners and patients were happy to send follow-up blood samples, particularly if it improved the chances of obtaining a definitive diagnosis. It was not always possible to obtain follow-up blood samples on tourists, as they had frequently moved on to their next destination. Seventy per cent of practitioners felt they would benefit from receiving information on overseas areas where dengue fever is considered a problem and information pertaining to diagnosis and treatment.

DISCUSSION

Dengue fever has become one of the most significant emerging diseases in tropical countries. Worldwide, more than 2.5 billion people are at risk of infection and each year 50–100 million cases of dengue fever are believed to occur.[1] There are many reasons for this global increase, including: complacency in mosquito control measures; increased population growth and subsequent unplanned urbanisation, leading to increases in breeding sites for the vector mosquito; and susceptible populations for the virus. Increased international trade has provided a rapid means of transport for the vector mosquito to new areas, and has facilitated its reintroduction to areas where it had previously been eradicated. The increase in air travel has provided an ideal mechanism for transporting the virus to new areas via travellers.[1,10]

Data from the Bureau of Tourism Research shows that 47 per cent of tourists enter Australia through Sydney. The number of Australians travelling abroad has also shown a steady increase in the last 5 years. The majority of tourists arrive via Asia and the Pacific, areas that have significant problems with dengue fever. These regions are also among the most popular destinations for Australians.[11] As our study suggests, it is reasonable to expect that the increase in cases in dengue fever in New South Wales is a result of increasing travel to endemic areas.

Although the numbers in our study are small, it would seem that secondary dengue fever infections are also increasing. This is not surprising because, as the number of primary infections rise, the stage is set for subsequent infections with additional travel. Before moving to

Australia, many New South Wales residents were born and lived in overseas areas where dengue fever is endemic and may have been previously infected. This immunologic 'priming' increases the risk of the more serious haemorrhagic dengue fever if previously infected people travel to endemic areas in the future.

Dengue fever is a disease that many New South Wales general practitioners and casualty department staff will see in their careers. Our study has shown that among this group there is a low index of suspicion and therefore dengue fever is not always considered in the differential diagnosis. If laboratories are to accurately diagnose infections of dengue fever, the provision of clinical and travel histories is essential to ensure that the correct viral test panels are undertaken and that interpretation of the results is appropriate. This is particularly important in New South Wales where several flaviviruses known to infect humans circulate.

It is important to ensure that cases are followed up to determine that infections were acquired overseas and not locally. Certainly the Queensland experience has shown that diagnostic training for general practitioners and emergency department physicians is an important surveillance tool for dengue fever, in addition to follow-up by public health authorities. This would provide a window of opportunity for public health practitioners in New South Wales to take a leading role in the provision of training and educational opportunities to the relevant clinical groups. Importantly, our study has also shown an interest in obtaining such information by health care providers.

In recent years, we have seen *Aedes aegypti* reintroduced into Queensland. There is ample evidence of dengue fever epidemics in Queensland beginning with one traveller 'seeding' the vector mosquito population and subsequently causing locally acquired cases.[5,7,10] It has long been assumed that *Aedes aegypti* had been eradicated from the remainder of Australia.[12] However, in February this year *Aedes aegypti* was found in significant numbers in Tennant Creek in the Northern Territory. The vector mosquito status of New South Wales may change in the future, as it has in Queensland and the Northern Territory. Whether this happens or not, dengue fever remains the most common cause of flaviviral disease in New South Wales, and case numbers are increasing. We cannot stop people travelling, but we can improve the index of disease suspicion and diagnosis of disease.

Dengue fever and its vector mosquito have adapted and evolved in such a way as to maximise their opportunities to increase their geographic range. In such a climate, the importance of rapidly and accurately diagnosing imported cases of dengue in tourists or returning residents, and thereby preventing onward transmission, is an important public health role shared by general practitioners, public health officers, and diagnostic and reference laboratories throughout Australia.

REFERENCES

1. Gubler DJ. Dengue and dengue hemorrhagic fever: Its history and resurgence as a global health problem. Gubler DJ, Kuno G (editors). *Dengue and dengue hemorrhagic fever*. Fort Collins, Colorado: CAB International; 1997: 1–22.
2. Gubler DJ. Epidemic dengue–dengue hemorrhagic fever as a public health, social and economic problem in the 21st century. *Trends Microbiol* 2002; 10: 100–3.
3. Lumley GF, Taylor FH. *Dengue: service publications.* Sydney: School of Public Health and Tropical Medicine, University of Sydney, 1943; 1–171.
4. Mackenzie JS, LaBrooy JT, Hueston L, Cunningham AL. Dengue in Australia. *J Med Microbiol* 1996; 45: 159–61.
5. Kay BH, Barker-Hudson P, Stallman ND, Wiemers MA, Marks EN, Holt PJ, et al. Dengue fever: Reappearance in Northern Queensland after 26 years. *Med J Aust* 1984; 140: 264–8.
6. Mackenzie JS, Lindsay MD, Coelen RJ, Broom AK, Hall RA, Smith DW. Arboviruses causing human disease in the Australasian zoogeographic region. *Arch Virol* 1994; 136: 447–67.
7. Sinclair DP. The distribution of *Aedes aegypti* in Queensland, 1990 to 30 June 1992. *Commun Dis Intell* 1992; 16: 400–3.
8. Australian National Notifiable Diseases List and Case Definitions. Available at www.health.gov.au/internet/wcms/publishing.nsf/Content/cda_nndss_dislist.htm.
9. Graphical Online Data Surveillance Evaluation for Notifiable Diseases (GODSEND). Sydney: Centre for Epidemiology and Research, NSW Department of Health, 2004.
10. Hanna J, Ritchie S, Merritt A, van den Hurk A, Phillips DA, Serafin IL, et al. Two contiguous outbreaks of dengue type 2 in North Queensland. *Med J Aust* 1998; 168: 221–5.
11. Tourism Snapshot: International 1997–2002. Available at www.btr.gov.au.
12. Mackenzie JS, Broom AK, Hall RA, Johansen CA, Lindsay MD, Phillips DA, et al. Arboviruses in the Australian region, 1990 to 1998. *Commun Dis Intell* 1998; 22: 93–100.

NITBUSTERS: HEADLICE IN SCHOOLS PROGRAM

The Nitbusters program is a NSW Health initiative to reduce the prevalence of headlice in the community. The project, developed in consultation with the NSW Federation of Parents and Citizens Associations and the NSW Department of Education and Training, educates school children and parents about headlice and how to screen for and treat them as a community.

The program is aimed not at eradicating headlice but at identifying and managing infestations. Nitbusters tries to educate communities through schools about the most effective ways to reduce populations of headlice by encouraging school 'Nitbuster days'. These days are coordinated by parent volunteers, who use a fine-toothed nit comb and white hair conditioner to both screen for and treat headlice.

As most parents realise, eliminating headlice completely is probably—for the moment at least—not realistic. However, learning a safe and effective and simple method of removing headlice can make the management of infestations a little easier. Nitbusters recommends that all families regularly practise this method of treatment. Keep a good quality nit comb in the shower and train children to use it every time they wash their hair, even if their heads are not itchy.

The Nitbusters program has held demonstration training days in a number of primary schools across New South Wales. Neighbouring schools were invited to attend these days and learn how to coordinate their own Nitbuster day.

Data is available from some of those demonstration schools. Over 3,000 primary school children have been screened. Of those screened, more than 24 per cent had infestations of headlice. This is similar to both Victoria and Queensland, where more than 20 per cent of primary school children have been reported to have headlice.

Information on headlice, and the Nitbusters program, including how to run a Nitbuster day, is available at **www.health.nsw.gov.au/headlice**.

OVERVIEW OF THE PUBLIC HEALTH IMPLICATIONS OF COCKROACHES AND THEIR MANAGEMENT

Peter Miller and Bryce Peters
Department of Health Sciences
University of Technology, Sydney

BACKGROUND

There are approximately 4,000 species of cockroaches worldwide and 428 species in Australia.[1] The majority of these species are not pests but live in the wild, feeding on decaying vegetation or other organic matter, and they are important in recycling this material. A number of cockroaches have become pests and live in or around homes where they are omnivorous scavengers. The 2 most significant pest cockroaches worldwide are the German cockroach *Blattella germanica* (Linnaeus) and the American cockroach *Periplaneta americana* (Linnaeus).

There are health implications from these pests, as they move freely from areas that may harbour pathogenic organisms: for example, from sewers to food or food preparation surfaces. Cockroach allergens can also be responsible for asthma. This article describes the public health implications of cockroaches, and their management, including consequences for the management of other pests.

THE COCKROACH SPECIES

The German cockroach *Blattella germanica*

The German cockroach is the most common cockroach in houses and apartments in Australia.[2] Adults are about 15 mm long and first instar nymphs (that is, the first nymphal stage) are about 3 mm long. They are able to live and breed in the numerous cracks and crevices and hiding places present in most kitchens, bathrooms and living areas. Their small size means that they are initially tolerated by human occupants, many of whom do not recognise early nymphal stages as cockroaches. Their rapid reproduction rate enables a few individuals to become a pest problem over one season, as each female produces a number of egg cases containing numerous eggs (Table 1). The egg cases are carried until just before the eggs hatch. This helps protect the egg cases and the eggs and is another factor in their success as pests.

Like other pest cockroaches, German cockroaches are nocturnal and forage for food and water at night when

TABLE 1

LIFE HISTORY OF THE GERMAN AND AMERICAN COCKROACHES

Cockroach	No. of eggs per egg case	Duration of nymphal development	Adult lifespan	No. of egg cases per female
German cockroach *Blattella germanica*	30–40	6–12 weeks	4–6 months	5–8
American cockroach *Periplaneta americana*	12–16	6–12 months	6–12 months	10–50

Source: Summarised from Hadlington and Gerozisis.[2]

they are less likely to be seen. In the daytime, they hide in cracks and crevices in cupboards and kitchen appliances and so are easily overlooked. The German cockroach is the most difficult pest cockroach species to control.

The American cockroach *Periplaneta americana*

This is the largest of the pest species, growing to around 40 mm in length.[2] It is red-brown, with fully developed wings that cover the abdomen, and it will fly in warm conditions. The species produces fewer generations per year than does the German cockroach and infestations therefore build up more slowly (Table 1). Because of the large size of both adults and nymphs, people are less tolerant of this species of cockroach in their homes or businesses, and the cockroaches also find fewer places inside to hide in the daytime. When established in homes they are normally found in wall voids or behind cupboards, in underfloor areas or in roof spaces. If sanitation is poor they can establish and breed inside homes but normally they enter living rooms, kitchens and bathrooms when they are foraging for food and water. In commercial premises, they are found in similar places and also in basement areas, service ducts and grease traps.

American cockroaches are often called peridomestic cockroaches because they are most associated with the areas around homes or buildings. Common areas where they are found include gardens, around garbage, inside drains and in outhouses such as sheds or garages. They can be common in sewers and sewer manholes. Because of their large size and relatively fast movement, a few American cockroaches inside the home means that people often initiate pest control measures more quickly than if they see a few German cockroaches.

Other pest cockroaches

There are other pest species of *Periplaneta* in Australia and throughout the world, and in some areas these may be as common as the German and American species. The smoky brown cockroach *Periplaneta fuliginosa* (Serville) is found in and around Sydney, and the Australian cockroach *Periplaneta australasiae* (Fabricius) is found commonly in tropical and subtropical areas of Australia. Both are large peridomestic cockroaches (around 35 mm long) that feed mainly on garden organic matter but they will forage inside buildings and establish themselves in garages and outbuildings, under floor areas, and in wall voids. These species are not usually found in sewers, unlike the American cockroach. The smoky brown cockroach is dark brown and the Australian cockroach is red-brown with distinctive yellow edges on the protective forewings.

The brown-banded cockroach *Supella longipalpa* (Serville) is about the same size as the German cockroach and has distinctive light bands running across the wings and abdomen. These cockroaches are often found dispersed through the house behind picture frames and in light switches and furniture. They are found in the warmer northern areas of Australia.

Finally, the Oriental cockroach *Blatta orientalis* (Linnaeus) can be encountered in cooler areas of Australia. It is about 30 mm long and has small functionless wings. Oriental cockroaches are dark brown or black and may be found under floors, in sewers and drains, and around garden rubbish.

Cockroaches as vectors of pathogens

The habits of cockroaches mean that they have the potential to be vectors of organisms that cause disease. A number of species live in sewers from which they can escape via poorly fitting manholes, vent pipes or drains. (Cockroaches are able to pass through the water in the S-bends of plumbing fixtures.) Cockroaches may feed on sewage, garbage and rotting food, which all support pathogens, and then transfer to food or food preparation surfaces and utensils. Roth and Willis published an extensive review of the biotic associations of cockroaches in which they cite numerous pathogens harmful to humans being found in or on cockroaches or in the faeces.[3] Brenner summarised the organisms pathogenic to humans that have been isolated from cockroaches.[4] There were 32 species of bacteria (including *Salmonella* and *Shigella* species), 15 species of fungi and moulds, 7 helminths (intestinal parasites), 2 protozoans, and 1 virus. Ash and Greenburg reviewed the vector potential of the German cockroach in spreading *Salmonella* enteritidis (Gaertner).[5] They pointed out that there was ample evidence that cockroaches could occur in large numbers in homes, restaurants and other institutions, and that they lived in close association with people, thus satisfying the requirement for synanthropy (a preference for living in human settlements). They found that bacteria rarely multiplied in the gut of the German cockroach but that the cockroach was capable of giving an inoculative dose to food. After experimental feeding of cockroaches with *S.* enteritidis, their faeces contained the organism for between 3 and 20 days.

Klowden and Greenburg came to a similar conclusion for the American cockroach as a disease vector.[6] Mackerras and Mackerras studied gastroenteritis in Queensland in 1946–1947 when there was an epidemic caused by *S.* bovismorbificans (Basenau).[7] They examined the cockroach population in hospitals and surrounding suburbs and concluded that the prevalence of contaminated cockroaches in hospitals could be regarded as a reflection of the opportunities they had to acquire and disseminate infections in the wards. Mackerras and Pope found that infected cockroaches could become carriers of *Samonella* for 2–7 weeks.[8]

Cockroaches were also implicated in the spread of infective hepatitis in California,[9] as evidenced by the decrease in the disease after cockroach control and the increase when control ceased.

COCKROACH ALLERGIES AND ASTHMA

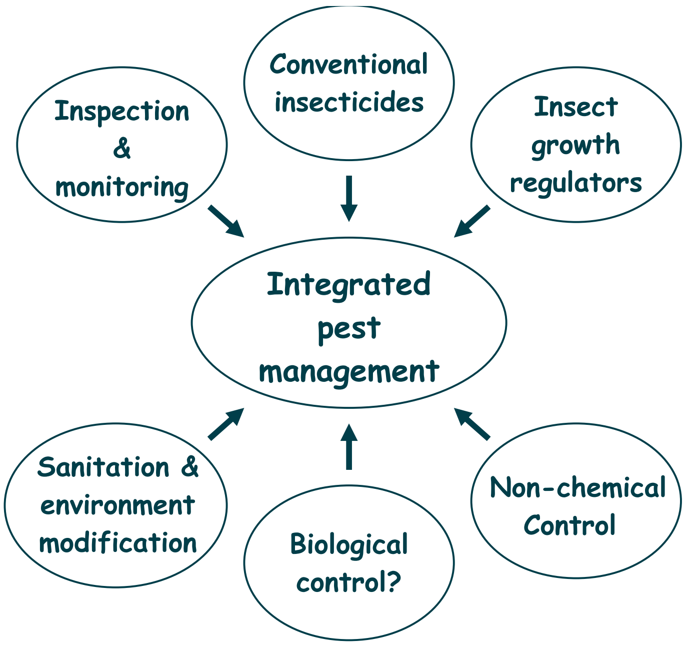
Bernton and Brown first demonstrated in 1964 that people could become allergic to cockroaches and their faeces.[10] This allergic reaction is a worldwide phenomenon, with sensitivity to cockroaches ranging from 23 to 60 per cent of the population tested.[11] Cockroach allergens are present mostly in settled dust, rather than air, as the particles are large and do not remain airborne unless disturbed. There seems to be a particular association between cockroach allergens and asthma but they also can cause rhinitis and dermatitis. The allergens are potent sensitisers of children and exposure to cockroach allergens early in life has been found to be a predictor for the development of asthma.[12]

Brenner cites the German cockroach as the principal cockroach causing allergies,[4] which is to be expected because of the close association between German cockroaches and people. Cockroach infestations in bedrooms are particularly associated with asthma, presumably because of the extended close association between the person and the cockroach allergens.

A number of studies have examined threshold levels of cockroach exposure above which susceptible individuals may be at risk of developing symptoms of asthma. For example, Arruda et al. found that levels of greater than 8 micrograms of 1 allergen in children's bedrooms led to increased hospital admissions for asthma.[13] Regular cockroach control will reduce the incidence of cockroaches and hence reduce the build-up of allergens.[14] However, even after cockroach control, allergens persist. Cleaning to reduce cockroach allergen may be possible to lower the risk of sensitisation or cockroach-induced asthma. Eggleston et al. used abamectin baits to control German cockroach populations and coupled this with

FIGURE 1

INTEGRATED MANAGEMENT OF PEST COCKROACHES



Source: Department of Health Sciences, University of Technology, Sydney.

cleaning.[15] The amount of allergens was reduced, but generally they were still at a level to cause clinical effects.

If control and cleaning techniques are to be successful in reducing allergens, the control must occur in all rooms and be completed with thorough cleaning to reduce allergen reservoirs to acceptable levels.[13] There should also be measures to maintain control and prevent reinfestation.

CONTROL OF COCKROACHES

Insect pest management, which involves integrating a number of procedures to gain control of cockroaches, can be instigated (Figure 1).[16] Insecticides remain the most common control method and these are usually applied as sprays to the cockroaches' hiding places and breeding areas. For German cockroaches, this would involve crack and crevice treatments, particularly to kitchens and bathrooms but often to bedrooms and living rooms. For American and other peridomestic cockroaches it would also involve treatment to under-floor and wall and ceiling spaces, drains and some garden areas.

Gels are newer formulations that are being used with good results. Gels are a combination of insecticide, food, attractants and water, which rely on the natural foraging and feeding behaviour of the cockroach. They involve less insecticide use and disruption to the human occupants. They are applied by means of a gel gun and they appear to control an infestation as effectively as or better than sprays.[17] The changeover to gels means that other pests, which may have been killed by sprays, are not controlled as they are not attracted to the gel. This may explain the increased problems from other pests such as ants and bedbugs.

CONCLUSION

Cockroaches live and feed in unhygienic places such as sewers and drains, or feed on garbage that may be contaminated. They certainly have the opportunity to transfer pathogens physically to humans or to their food and living areas, but whether they are competent vectors of the organisms they carry is still under debate. However, the general conclusion is that they should be controlled, particularly in sensitive areas such as medical facilities or food preparation areas, to limit their potential for physical transfer of pathogens. Cockroach allergens are potent sensitisers of children to asthma and are triggers for asthma attacks. Control of cockroaches should be instigated to limit potential adverse health effects from their presence. The newer gel formulations are effective and reduce insecticide use but their close targeting of cockroaches means that other pests, such as bedbugs, are no longer controlled during cockroach control programs.

REFERENCES

1. Naumann ID, editor. *The insects of Australia*. Vol 1. Melbourne: Melbourne University Press, 1991.
2. Hadlington P, Gerozisis J. *Urban pest control in Australia*. Sydney: New South Wales University Press, 1985.
3. Roth LM, Willis ER. The biotic associations of cockroaches. *Smith Inst Misc Coll* 1960; 141: 1–470.
4. Brenner RJ. Economics and medical importance of German cockroaches. Rust MK, Owens JM, Reierson DA (editors). *Understanding and controlling the German cockroach*. Oxford: Oxford University Press, 1995; 77–92.
5. Ash N, Greenburg B. Vector potential of the German cockroach (Dictyoptera: Blattellidae) in dissemination of *Salmonella enteritidis* serotype *typhimurium*. *J Med Entomol* 1980; 17: 417–23.
6. Klowden MJ, Greenburg B. *Salmonella* in the American cockroach: Evaluation of vector potential through dosed feeding experiments. *J Hyg* 1976; 77: 105–11.
7. Mackerras IM, Mackerras MJ. An epidemic of infantile gastroenteritis in Queensland caused by *Salmonella bovis-morbicans* (Basenau). *J Hyg* 1949; 47: 166–81.
8. Mackerras IM, Pope P. Experimental *Salmonella* infections in Australian cockroaches. *Exp J Biol Med Sci Aust* 1948; 26: 465–70.
9. Tarshis IB. The cockroach: a new suspect in the spread of infectious hepatitis. *Am J Trop Med Hyg* 1962; 11: 705–11.
10. Bernton H, Brown H. Insect allergy: Preliminary studies of the cockroach. *J Allergy* 1964; 35: 506–13.
11. Arlian LG. Arthropod allergens and human health. *Annu Rev Entomol* 2002; 47: 395.
12. Litonjua AA, Cary VJ, Burge HA, Weiss ST, Gold DR. Exposure to cockroach allergen in the home is associated with incident doctor-diagnosed asthma and recurrent wheezing. *J Allergy Clin Immunol* 2001; 107: 41–7.
13. Arruda LK, Vailes LD, Ferriani VPL, Santos ABR, Pomes A, Chapman MD. Cockroach allergens and asthma. *J Allergy Clin Immunol* 2001; 107: 419–28.
14. Gergen PJ, Mortimer KM, Eggleston PA, Rosenstreich D, Mitchell H, Ownby D, et al. Results of the National Cooperative Inner-City Asthma Study (NCICAS) environmental intervention to reduce cockroach allergen exposure in inner-city homes. *J Allergy Clin Immunol* 1999; 103: 501–6.
15. Eggleston PA, Wood RA, Rand C, Nixon WJ, Chen PH, Lukk P. Removal of cockroach allergen from inner-city homes. *J Allergy Clin Immunol* 1999; 104: 842–6.
16. Rust MK, Owens JM, Reierson DA. *Understanding and controlling the German cockroach*. Oxford: Oxford University Press, 1995; 77–92.
17. Miller PF, Peters BA. Performance of Goliath cockroach gel against German cockroach (Blattodea: Blattellidae) and a mixed population of American cockroach and Australian cockroach (Blattodea: Blattidae) in the field. Robinson WH, Rettich F, and Rambo GW (editors). *Proceedings of the 3rd International Conference on Urban Pests*. Prague: Graficke zavodny Hronov, 1999; 153–159.

REVIEW OF PUBLIC HEALTH ADVICE ABOUT TICKS

Maria Torres
NSW Public Health Officer Training Program
NSW Department of Health

Victor Carey
Northern Sydney Public Health Unit

To make decisions about how to deal with ticks, the public, clinicians and public health professionals require evidence-based, unambiguous and practical information. This article provides an introduction to ticks and a brief description of the review of NSW Health's public health advice about ticks.

BACKGROUND

Ticks are arthropods: that is, animals with an external skeleton and jointed legs. Within this phylum they are arachnids, in the subclass acari, closely related to mites. There are 2 main tick families: Ixodidae, or hard ticks, with over 700 species worldwide; and Argasidae, or soft ticks, with up to 185 species worldwide.[1]

In Australia there are approximately 70 species of ticks, most of which are hard ticks. The majority of these ticks are native, but some introduced species are widely distributed. In New South Wales, from a public health perspective, the most important is *Ixodes holocyclus*, a native species also known as 'paralysis tick'. Like all ticks, *I. holocyclus* is sensitive to desiccation (dehydration) and so a temperate climate with relatively high levels of humidity is the best for tick survival.[2] *I. holocyclus* is found from Queensland to Victoria, mainly in humid bushland areas on the eastern seaboard along a coastal band that, in parts, extends up to 70 km inland. Encounters between *I. holocyclus* and humans are relatively common, due to the fact that a large proportion of the human population lives within the coastal band, and urban development is increasingly encroaching into bushland.

Ticks are ectoparasites, which means they live and feed on the outside of their hosts. The main hosts for *I. holocyclus* are bandicoots, but the tick also attaches itself to other animals including humans. The life cycle of the tick includes 4 stages of development: egg, larva, nymph and adult. During their lifecycle, most species of hard ticks feed on the blood of 3 different hosts, 1 each for the larva, nymph and adult stages. Larvae and nymphs feed for several days and then drop off the host to the ground where they moult into the next stage. Adult females feed to obtain nutrients to develop eggs; after feeding for several days they drop off the host and lay thousands of eggs on the ground before dying. Adult male ticks feed on hosts and on engorging adult female ticks. *I. holocyclus* takes approximately 1 year to complete its lifecycle. Larvae are most common in the autumn months, nymphs are most common in winter, and adults are most common in spring, but tick stages can overlap across the seasons.

Figure 1 includes a graphic representation of the life cycle of *I. holocyclus*.

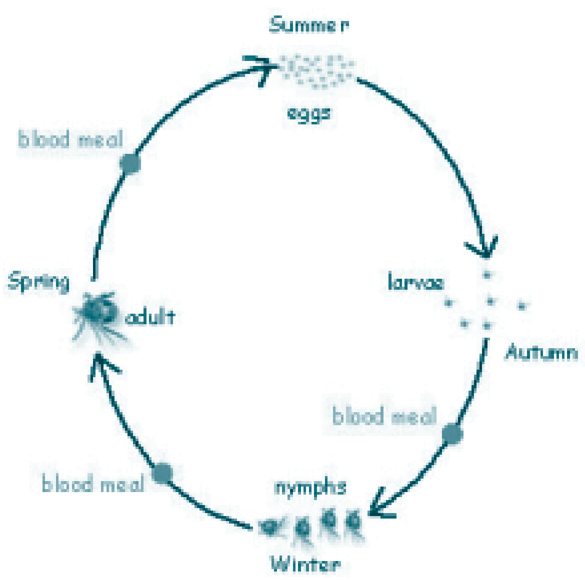
From the ground, ticks climb to grasses or low bushes and 'quest' for a passing host. Once on the host they move upward until they find a suitable place to attach. In humans this is often a place where they will not be easily found such as skin folds. Ticks use their conical lower lip (hypostome) to penetrate the skin of the host. They then secrete a mixture of substances, such as anticoagulants and prostaglandins, to inhibit haemostasis, augment local blood flow and suppress the inflammatory and immune response of the host, and thus secure both attachment to and meals from the host.[2] In addition, some ticks secrete a cement to further secure attachment. *I. holocyclus* does not secrete cement but penetrates the skin deeper than some of the other species of tick.

Clinical presentation and public health importance

In addition to being itchy and sometimes painful, the bites of *I. holocyclus* may be associated with other health problems such as allergic reactions, tick paralysis and the transmission of organisms that can cause infectious diseases. Further, scratching at the site of the bite can lead to secondary infection, and a foreign body granuloma

FIGURE 1

LIFE CYCLE OF *IXODES HOLOCYCLUS*



Note: The sizes of unfed ticks (larvae: pin-point size; nymphs: pin-head size; unfed adult: match-head size) may not be accurately represented in the picture.

Source: Dr Norbert Fischer, Crown Street Veterinary Hospital, Wollongong, New South Wales.

may develop when parts of the tick's mouth are left in the host after incomplete removal of the tick.[3]

Allergic reactions to tick bites

Allergic reactions to tick bites are caused by allergens contained in the saliva of *I. holocyclus*. These allergens, studied extensively by Gauci et al.,[4] are introduced into the host from the time of the tick's attachment. It has been reported that all biting stages of *I. holocyclus* can sensitise a host, which can later precipitate an allergic reaction.[2,5] Anecdotal evidence suggests that most allergic reactions follow bites by female adult ticks.

Allergic reactions range from mild local reactions to generalised and sometimes severe reactions including anaphylaxis.[5,6] Local reactions are the most common. They may last for weeks and, depending on their severity, may require medical treatment. Even though severe allergic reactions are rare, it is important to be aware that they may occur shortly after a tick bite.[7] Usually a history of worsening reactions to previous tick bites precedes a severe systemic reaction,[2] and adrenaline and resuscitation facilities may be needed to treat these systemic reactions.[2,5] Individuals who have experienced severe allergic reactions to tick bites should have access to injectable adrenaline at all times.[2]

Tick paralysis

Tick paralysis in humans is a rare but potentially fatal condition; young children are the most commonly affected.[8,9] Tick paralysis is caused by neurotoxins contained in the saliva of engorging female adult ticks. Symptoms start several days after attachment of the tick, when the tick reaches a rapid feeding phase accompanied by intense salivation, which coincides with high production of toxins.[8] Initial symptoms of tick paralysis include unsteady gait, weakness of limbs, and lethargy; an ascending, flaccid and symmetrical paralysis progresses over hours. In severe cases ventilatory failure may occur. Tick paralysis, particularly in a child, should be treated in intensive care where supportive management is usually sufficient. In severe cases the use of an antitoxin may be necessary,[10] but antitoxins should be used cautiously as they may cause allergic reactions.[11] Removal of the tick is an important step in the treatment of tick paralysis. However, an important characteristic of paralysis caused by *I. holocyclus* is that the condition may continue to deteriorate even after the tick has been removed.[3,8,10] Recovery is often slow.

Tick-borne infectious diseases

After a few days of attachment, a tick infected with a pathogen (whether a virus, bacteria or protozoa) may transmit the pathogen to the host with its saliva and cause an infectious disease.

Spotted fevers are the main tick-borne infectious disease in Australia. Even though they are not thought to be common diseases, the real incidence of these and other tick-borne infectious diseases in New South Wales is not known as the conditions are not notifiable.

I. holocyclus is the main vector for human transmission of *Rickettsia australis*, the bacterium that causes one of the spotted fevers (Queensland tick typhus). The geographical distribution of Queensland tick typhus is the same as that of *I. holocyclus*. Nonspecific symptoms develop between 1 and 11 days after the tick bite and include fever, chills, myalgia, arthralgia, headache and regional lymphadenopathy. In up to 70 per cent of cases, a characteristic eschar (dry scab) with a black necrotic centre and red areola is present at the site of the bite.[11] A generalised maculopapular rash (a rash that usually covers a large area, is red and has small confluent bumps) may appear a few days after the onset of the nonspecific symptoms. Clinical diagnosis is confirmed by serology. Queensland tick typhus can be treated with doxycycline, an antibiotic belonging to the class called tetracyclines. Serious illness is rare.[3] If untreated, the fever usually resolves in 1–2 weeks, but other symptoms may persist for several months.[3]

Flinders Island spotted fever has a similar presentation to Queensland tick typhus. It is caused by *Rickettsia honei* and the main vector is the tick *Ixodes cornuatus*. Most reported cases are from Flinders Island, mainland Tasmania and Victoria.[3]

Lyme disease is caused by the bacterium *Borrelia burgdorferi*, which is transmitted to humans by certain species of *Ixodes* ticks. Symptoms of Lyme disease appear within days, weeks or months of a tick bite and include early nonspecific symptoms such as fever, headache, arthralgia and myalgia, which may be accompanied by erythema migrans, a characteristic 'bull's-eye' rash around the site of the tick bite. The nervous, cardiac and musculoskeletal systems may be affected at later stages of Lyme disease. Cases of patients with symptoms resembling Lyme disease have been reported from eastern Australia since 1982,[12] but these cases have not been confirmed with serology.[3] Hudson et al. postulate that the cause of the disease in Australia is a spirochaete (a spirally-coiled rodlike bacterium) related to *B. burgdorferi*.[13] However, a study that examined over 12,000 ticks collected in coastal areas of New South Wales failed to detect *B. burgdorferi* or any other spirochaete.[14] *I. holocyclus*, the logical candidate vector of the pathogen in Australia, has been shown to be incapable of maintaining or transmitting *B. burgdorferi* to humans.[15] The existence of Lyme disease in Australia continues to be debated.

I. holocyclus is also a vector for *Coxiella burnetti*, the agent responsible for Q fever. However, this disease is mainly acquired through contact with infected farm and domestic animals.

Other infectious diseases such as tick-borne arboviral infections, babesiosis and ehrlichiosis are a burden in other parts of the world because of their effect on both human and animal health. The Australian Quarantine and Inspection Service ensures that the species of ticks that are vectors for these diseases are not introduced into Australia.

REVIEW OF PUBLIC HEALTH ADVICE ABOUT TICKS, IN PARTICULAR ABOUT *I. HOLOCYCLUS*

In 2002, in response to public concern about ticks, the NSW Department of Health published the brochure *Tick Alert*. A review of the brochure, which is mainly about *I. holocyclus*, was completed in March 2004. The review involved an initial revision of the existing brochure by the Northern Sydney Public Health Unit followed by consultation with relevant stakeholders. These included infectious disease physicians, emergency medicine clinicians, dermatologists, clinical toxicologists, immunologists, entomologists, toxicologists, consumers, veterinarians, health departments and other providers of information about ticks to the public such as the NSW Poisons Information Centre and St John Ambulance Australia.

There was general agreement that in addition to providing information about health problems that may follow a tick bite, one of the main messages of the brochure should be how to prevent tick bites—just as prevention advice is provided about other vectors of disease. To ground this advice, the brochure includes information about the ecology of ticks, in particular *I. holocyclus*, their lifecycle and habitat.

During the first round of consultation, many of the comments received were about methods of tick removal. Most stakeholders proposed 1 of 2 methods: mechanical removal of ticks or killing the tick in situ prior to removal. One of the difficulties faced at this point was that there is no clear evidence to support or refute either method for the removal of *I. holocyclus*. An evaluation of 5 methods commonly advocated for tick removal concluded that mechanical removal by grasping the tick's mouth-parts close to the skin and pulling it off should be used for all ticks unless research on a particular species suggested a different approach.[16] This method is recommended in many publications that refer to tick removal. However, proponents of the method of killing the tick in situ pointed out that certain characteristics of *I. holocyclus* (such as its small size and its method of attachment by deep penetration of the skin without deposit of cement) may require a different method of removal. These proponents refer to advice provided by Stone,[17] who postulated that the mechanical removal of *I. holocyclus* may induce anaphylaxis as a result of rapid dispersal of toxins and allergens away from the bite site. Stone has suggested that *I. holocyclus* should be killed in situ using an insect or tick repellent containing pyrethrins or synthetic pyrethroids.[17]

A meeting of stakeholders was held in February 2004. Some stakeholders provided comments in writing before the meeting and these were used to inform the discussion. Consensus was reached at the meeting to advise the public to remove ticks as soon as they are found, using fine forceps (not ordinary tweezers) or surgical scissors. There was agreement that there was not enough evidence to suggest that killing the tick in situ reduced an individual's exposure to allergens. In addition, participants discussed the danger of providing advice to the public that recommended killing attached ticks with repellents, as this advice may lead to the use of inappropriate products on the skin.

There are several products and methods commonly used to treat tick bites, including applying petroleum jelly, methylated spirits or nail polish, and burning the tick with a hot match. These methods were evaluated by Needham, who found that they failed to cause ticks to detach.[16] Anecdotal accounts suggest that sodium bicarbonate may be useful to calm the itchiness associated with tick bites, but there is no evidence to support this practice.

CONCLUSION

Even though the burden of disease attributable to tick-related illness is perceived to be small, the incidence of tick-related illnesses is unknown. Evidence is also lacking in relation to methods of tick removal. Studies to answer these questions would be useful.

The public health advice about ticks in New South Wales should be reviewed regularly, particularly if new evidence relevant to this advice becomes available.

> The revised public health information brochure *Tick Alert* can be downloaded from the NSW Department of Health website at **www.health.nsw.gov.au**.

REFERENCES

1. Barker SC, Murrell A. Systematics and evaluation of ticks with a list of valid genus and species names. Brisbane: University of Queensland (unpublished).
2. Sutherland SK. Ticks. *Australian animal toxins*. Sutherland SK, Tibballs J, (editors). Melbourne: Oxford University Press, 2001; 467–88.
3. Playford G, Whitby M. Tick-borne diseases in Australia. *Aust Fam Physician* 1996; 25(12): 1841–5.
4. Gauci M, Stone BF, Thong YH. Isolation and immunological characterisation of allergens from salivary glands of the Australian paralysis tick *Ixodes holocyclus*. *Int Arch Allergy Immunol* 1988; 87(2): 208–12.

5. Brown AFT, Hamilton DL. Tick bite anaphylaxis in Australia. *J Accid Emerg Med* 1998; 15(2): 111–3.
6. Gauci M, Loh RKS, Stone BF, Thong YH. Allergic reactions to the Australian paralysis tick, *Ixodes holocyclus*: diagnostic evaluation by skin test and radioimmunoassay. *Clin Exp Allergy* 1989; 19: 279–83.
7. Kemp A. Tick bites. *Med J Aust* 1986; 144: 615.
8. Grattan-Smith PJ, Morris JG, Johnston HM, Yiannikas C, Malik R, Russell R, et al. Clinical and neurophysiological features of tick paralysis. *Brain* 1997; 120: 102–13.
9. Barber PA, Chambers ST, Parkin PJ. Australian paralysis tick bite. *N Z Med J* 1994; 107(980): 252–3.
10. Pearn J. The clinical features of tick bite. *Med J Aust* 1977; 2: 313–8.
11. Storer E, Sheridan A, Warren L, Wayte J. Ticks in Australia. *Australas J Dermatol* 2003; 44(2): 83–9.
12. Stewart A, Glass J, Patel A, Watt G, Cripps A, Clancy R. Lyme arthritis in the Hunter Valley. *Med J Aust* 1982; 1(3): 139.
13. Hudson BJ, Barry RD, Shafren DR, Wills MC, Caves S, Lennox VA. Does Lyme borreliosis exist in Australia? *J Spirochetal Tick-Borne Dis* 1994; 1(2): 46–51.
14. Russell RC, Doggett SL, Munro R, Ellis J, Avery D, Hunt C, et al. Lyme disease: a search for a causative agent in ticks in south-eastern Australia. *Epidemiol Infect* 1994; 112(2): 375–84.
15. Piesman J, Stone BF. Vector competence of the Australian paralysis tick, *Ixodes holocyclus*, for the Lyme disease spirochete *Borrelia burgdorferi*. *Int J Parasitol* 1991; 21(1): 109–11.
16. Needham GR. Evaluation of five popular methods for tick removal. *Pediatrics* 1985; 75(6): 997–1002.
17. Stone BF, Binnington KC, Gauci M, Aylward JH. Tick–host interactions for *Ixodes holocyclus*: role, effects, biosynthesis and nature of its toxic and allergenic oral secretions. *Exp Appl Acarol* 1989; 7: 59–69.

A SURVEY OF BEDBUGS IN SHORT-STAY LODGES

Nathan Ryan
Environmental Health Unit
City of Sydney Council

Bryce Peters and Peter Miller
Department of Health Sciences
University of Technology, Sydney

An increase in the number of anecdotal reports of bedbug infestations in short-stay lodge-type accommodation used by backpackers and other budget travellers in the City of Sydney prompted a pilot survey to determine the extent of the problem. The aim of the survey was obtain the perspective of the lodge managers on bedbug problems and how they handled them. This article describes the survey results and assesses the effectiveness of the methods of control reported.

BACKGROUND

Bedbugs have long been associated with humans and have been a significant problem for accommodation providers for centuries. The bug uses its piercing proboscis to feed on human blood at night and lays eggs in bedding and furniture. Bedbug bites can cause significant irritation, and some individuals are particularly sensitive. While posing a potential problem, bedbugs are not known to be vectors of disease.[1,2] Only the common bedbug *Cimex lectularius* has been found in Sydney to date, although the tropical bedbug *Cimex hemipterus* may be a recent import, given the number of travellers arriving from northern Australia and Asia, where it is endemic, and given that this species was recently recognised in Queensland.[3]

Infestations of bedbugs have traditionally been associated with poor sanitation, but the dramatic resurgence of bedbug activity in Australia and overseas may be attributed to a number of different causes.[4] The introduction of residual insecticides and improved standards of domestic hygiene have significantly reduced the bedbug problem but a number of recent studies indicate a reappearance of bedbugs overseas and in Australia.[5–8] Increasing complaints of bedbugs by short-stay guests, including reports to the City of Sydney's Environmental Health Unit, the South Eastern Sydney Public Health Unit, and local doctors, prompted us to conduct a survey of the situation in short-stay lodges in Sydney.

Short-stay guests are an important component of Australian tourism, contributing around 20 per cent of the total number of tourists. Following recent council amalgamations, the enlarged City of Sydney now has about 6,000 short-stay beds in around 65 lodges, some of which have been in continuous operation for more than 15 years. Guests stay an average of 3–4 days. Many of these guests have spent time in lodges throughout Europe, Asia and Australia, where they may have been exposed to bedbugs before arriving in Sydney, although there is no evidence of transmission of bedbugs from other countries to Australia or from other states to New South Wales.

SURVEY REVEALS EXTENT OF THE PROBLEM

All 52 short-stay lodges on the City of Sydney City register before the council amalgamation were asked to participate in a pilot survey to provide a complete picture of the bedbug problem in the industry. Between October and December 2003, face-to-face interviews were conducted at 47 of the 52 short-stay lodges chosen for the survey (5 lodges declined to participate). Face-to-face surveys were conducted in preference to mail-return surveys to ensure a high response rate and to allow the authors to view the premises at close hand. Infestations reported by guests were initially confirmed by the managerial or cleaning staff of the lodges. A complaint from 1 guest, and our visual inspections of 4 of the premises with active infestations during the survey, confirmed the presence of bedbugs. The species of bedbug was not identified.

We asked the lodge operators about their history of infestations over the past year and whether the problem recurred after their preferred treatment, which included both chemical and non-chemical approaches. A high proportion of premises (79 per cent, or 37/47 lodges) reported some bedbug activity in the preceding 12 months. Of these, more than one-half reported 3 or more outbreaks; that is, reports of guests being bitten or confirmed infestations. Most lodge operators reported that the problem was increasing, with long-term operators regarding the last 2 years as their worst ever for bedbugs. A follow-up report from 1 participant operator indicated that up to 35 per cent of their beds were infected. This raises the issue that the closure of any beds, or whole rooms, for treatment of bedbugs has a significant economic effect, both in terms of the cost of professional treatment and lost bed rentals.

The number of reported infestations peaked in the summer months, coinciding with increased numbers of tourists returning from northern Australia. The infestations were not related to the age, construction material, bedding material or apparent level of hygiene of the lodges. Two-thirds of the short-stay lodges had some kind of regular pest control program in place, often supplied by pest control operators and usually intended for control of cockroaches, but these were not generally effective against bedbugs. One-third of lodges had no regular pest control program.

REPORTED CONTROL METHODS WERE INEFFECTIVE

After an initial bedbug pesticide treatment, either by the lodge operator or by a pest control operator, 57 per cent of responding lodges suffered a repeat infestation in the room treated, mostly within 1 month. That is, the lodge operators either found live bedbugs and/or received reports of guests being bitten by bedbugs. This showed that the current treatment methods and/or insecticides were ineffective. Less than half of the lodge owners followed up initial treatment with a second application of insecticide. Insecticidal agents applied by the lodge operators included various flea bombs, sprays, cockroach baits and ant dusts, and bleach or borax powder. Many lodge operators claimed that currently available pesticides were ineffective, but this was probably more due to poor choice of agent or application than any chemical resistance. For example, cockroach baits and borax are not designed to kill bedbugs.

Non-chemical means of controlling bedbug infestations had equally poor results. Most lodge operators routinely banned the use of sleeping bags in dormitories. When an infestation was reported, many hostel operators insisted that backpackers washed their clothes and linen in hot water, but few operators confirmed the usefulness of these measures. Also, the backpacks may not have been included in the cleaning and washing process and may have acted as a source of reinfestation.

A number of unorthodox measures were employed to control infestations or rid an infected room of bedbugs. One lodge operator reported sprinkling bleach powder around the bed and crumpled camphor into skirting boards. A number of operators reported dipping the ends of wooden slats from bunk beds into boiling water or bleach. Similarly, hot water was poured between floor cracks and skirting boards to kill eggs. Some operators just sprayed mattresses with domestic insecticides. One operator believed that smoking tobacco in a room would prevent bedbugs from biting the occupants. A number of operators used tea-tree (melaleuca) or eucalyptus oils in the belief that these would repel the bedbugs.

It was evident from the responses that many of the lodge operators lacked sufficient knowledge about effective bedbug treatments. Information that is freely available on the internet or from pest control operators was not commonly accessed. One worrying belief among lodge operators was that professional pest control treatments did not work any better than those domestic treatments described above, suggesting that lodge operators needed advice about the value of professional treatment.

RECOMMENDATIONS FOR THOROUGH TREATMENT

For successful control of bedbugs, one must kill adult, nymph and egg forms of the pest, preferably using a residual insecticide and follow-up treatments. Mechanical means of removing bugs (burning, squashing and scrubbing) are highly labour-intensive and are effective only in the areas they reach: too many places in a room that harbour bugs may be overlooked for these methods to be reliable on their own. Good housekeeping, such as regular vacuuming around skirting boards, is often recommended as a preventative strategy. Steam can also be very effective in controlling bedbugs; one study from the United States showed that steam cleaning of mattresses gave more effective long-term control than did chemicals.[9]

Chemical control is more effective (if applied correctly) as it eliminates current infestations and protects against subsequent infestations, although this depends on the insecticides used.[10] Bedbugs have shown resistance to most major insecticide groups but the most appropriate insecticide is still permethrin.[11,12] A residual formulation is recommended to control emerging nymphs and also those nymphs and adults not directly treated. In an ideal environment, adult bedbugs may live for up to 6 or even 12 months without blood feeding, waiting in stored furniture and bedding for their next opportunity to feed. A thorough insecticide treatment regime is vital, including reapplication of insecticides after 1–2 weeks to allow for new eggs to hatch.

Lodge guests must accept that they contribute to the problem by spreading bedbugs in their luggage. Not all insect bites are due to bedbugs, so when an infestation is suspected this should be confirmed by looking for bedbugs as they bite, in the mattress or in luggage and clothing. While many people show no immediate signs or pain from bites, delayed reactions up to 9 days later have been reported.[13] About one-quarter of people are very sensitive and may show severe reactions that are best treated with antihistamines and topical steroids.[14]

SOME POSITIVE OUTCOMES

A major benefit of this survey is that the operators of short-stay lodges are now openly discussing the bedbug issue and effective ways to deal with it. This more open attitude will assist further training of lodge operators. Upcoming initiatives of the City of Sydney Council include the production of information packs for lodge operators and guests, and the promotion of better control methods as up-to-date information becomes available to local government, industry associations and pest control operators. Training environmental health inspectors to identify these problems may also assist lodge operators with how to manage a bedbug infestation in their premises and may assist complainants with how to manage a bedbug infestation on their person.

CONCLUSION

The extent of bedbug infestations is a consequence of various factors such as: the insects' 'hitching a ride' on travellers and their luggage; changes in pest management; appropriate training in the pest management industry; and a reluctance of the accommodation industry to report the problem.[4] The City of Sydney has the largest short-stay lodge industry of any local government area in New South Wales, which is often both the first and the last port of call for short-stay guests. Their high mobility may facilitate the transfer of bedbugs around Australia and overseas.

Most short-stay lodge operators asked for clear information regarding simple treatment programs, and for more information for travellers on how to minimise their risk. A more open approach to the problem of bedbugs in Sydney, including awareness of the issue and education about effective control steps, will go some way to reducing the number of infestations.

ACKNOWLEDGEMENTS

The authors would like to thank the following for assistance with this project: Jim Mann and Ali Khalife, University of Technology, Sydney, for assisting with interviewing; Dr Harry Wilson, University of Technology, Sydney, for preliminary editing of the manuscript; Toni Cains, South Eastern Sydney Public Health Unit, for reports on bedbug infestations; Merilyn Geary, Department of Medical Entomology, Institute of Medical Pathology and Clinical Research, Westmead, for information on the identity of bedbug species in Sydney; Stephen Doggett, Department of Medical Entomology, Institute of Medical Pathology and Clinical Research, Westmead, for his generous comments and suggestions on the final version of the article; and the lodges that participated in the project.

REFERENCES

1. Goddard J. Do bed bugs carry human diseases? A controversy. *Pest Control Technol* 2003; 31: 38–40.
2. Blow JA, Turell MJ, Silverman AL, Walker ED. Stercorarial shedding and transtadial transmission of hepatitis B virus by common bed bugs (Hemiptera: Cimicidae). *J Med Entomol* 2001; 38: 694–700.
3. Doggett SL, Geary MJ, Crowe WJ, Wilson P, Russell RC. Has the tropical bed bug *Cimex hemipterus* (Hemiptera: Cimicidae), invaded Australia? *Environ Health* 2003; 3(4): 80–83.
4. Doggett SL, Geary MJ, Lamond P, Russell RC. Between the sheets with bed bugs: their ecology and control. *Prof Pest Manage* 2004; April–May: 25–27.
5. Krueger L. Don't get bitten by the resurgence of bedbugs. *Pest Control* 2000; 68: 58–64.
6. Koehler P, Harlan H. Bedbugs: infestations on the rise. *Health Med Weekly* 2001; August 27: 6–7.
7. Paul J, Bates J. Is infestation with the common bed bug increasing? *BMJ* 2000; 320: 1141.
8. Institute of Medical Pathology and Clinical Research: *Bed bugs: Fact sheet*. Sydney: Institute of Medical Pathology and Clinical Research, Westmead Hospital, October 2003.
9. Meeks F. Bed bugs bite back. *PCTOnline* 2003; July 44–52. Available online at www.pctonline.com.
10. Fletcher MG, Axtell RC. Susceptibility of the bedbug, *Cimex lectularius*, to selected insecticide and various treated surfaces. *Med Vet Entomol* 1993; 7: 69–72.
11. Myamba J, Maxwell CA, Asidi A, Curtis CF. Pyrethroid resistance in tropical bedbugs, *Cimex hemipterus*, associated with the use of treated bednets. *Med Vet Entomol* 2002; 16: 448–51.
12. Feroz M. Biochemistry of malathion resistance in a strain of *Cimex lectularius* resistant to organophosphorus compounds. *Bull World Health Organ* 1971; 45: 795–804.
13. Sansom JE, Reynolds NJ, Peachey RDG. Delayed reaction to bed bug bites. *Arch Dermatol* 1992; 128: 272–3.
14. Cleary CJ, Buchanan D. Diagnosis and management of bedbugs. *Nurse Prac* 2004, 29: 47–8.

FLY LARVAE FOR WOUND MANAGEMENT: A MAGGOT MAKEOVER

Merilyn Geary and Richard Russell
Department of Medical Entomology
Institute of Clinical Pathology and Medical Research, Westmead

Maggots certainly have an image problem, but this humble insect—the immature stage of a housefly or blowfly—has much to offer the field of medicine in the 21st century. Maggots of certain fly species feed on necrotic flesh, and through this debriding activity can assist the healing of chronic soft-tissue wounds (such as pressure and venous stasis ulcers, diabetic foot infections, and postoperative wounds) that are resistant to surgical or antibiotic intervention.[1,6] This article describes Maggot Debridement Therapy (MDT), an old remedy that has been revised and is now proven to be valuable for treating wounds unresponsive to conventional wound management.

BACKGROUND

For centuries, maggots were known to have beneficial effects on wounds. Ambroise Paré is credited to be the first to note his observations in the 1500s. Throughout military history, many other positive comments have been recorded by military surgeons in regard to maggot-infested wounds. However, it was not until the 1920s that therapeutic experimentation with maggots was instigated by William Baer, a clinical professor in orthopaedic surgery at the Johns Hopkins University in Baltimore, Maryland, whose unorthodox methods were successful in the treatment of osteomyelitis and pyogenic wounds.[2] His method of MDT was adopted and routinely used in over 300 hospitals in the United States throughout the 1930s and early 1940s, but MDT was replaced with the introduction of penicillin and modern surgical procedures.[3–6]

The 1980s brought an increase of bacterial resistance to antibiotics, and MDT was revisited as a procedure to assist in the treatment of nonhealing wounds that were resistant to antibiotics, or when surgical intervention was not an option. In recent years, simple procedures have been developed for the culture of disinfected maggots, and their placement within restrictive dressings into nonhealing wounds, to provide for cleansing of necrotic tissue and initiation of the healing process.[7,8] Dr Ronald Sherman and associates from the Veterans Affairs Medical Center, California, pioneered the reintroduction of MDT. His clinical trials indicated that MDT was several times more efficient at debriding infected and gangrenous wounds (and in healing them more rapidly) than other modern nonsurgical treatments.[1,6,9]

While a range of reports describe good outcomes from MDT on different types of wounds,[2,3,8,9] mystery still surrounds the unique way that maggots 'nurse' wounds as they actively consume dead tissue and fluids. In feeding, maggots secrete proteolytic enzymes that turn necrotic tissues into a semi-liquid form, which they ingest along with microorganisms that cause wound infections. As they cleanse the wound site, they exude an antibacterial agent that has a wide spectrum of activity against many resistant pathogens, while their proteolytic enzymes are also capable of digesting bacteria. The mechanical feeding of the maggots and the reduction of necrotic tissue changes the wound's environment from an acid to a more alkaline pH, which assists in stimulating healthy granulated tissue.[10,11]

In the last 15 years, many thousands of patients with bedsores, leg ulcers, diabetic foot wounds, and postsurgical infections have been successfully treated by MDT. At present, health care facilities in the United Kingdom, Europe, and the United States now produce thousands of medicinal maggots per week for therapists. In the United Kingdom, MDT is now recognised as a procedure that can be officially prescribed and claimed on health care benefits.[12]

MAGGOT DEBRIDEMENT THERAPY IN AUSTRALIA

Persistent requests from throughout Australia for sterile maggots encouraged the Department of Medical Entomology, Institute of Clinical Pathology and Medical Research (Westmead Hospital), to establish a colony of the sheep blowflies *Lucilia cuprina* and *Lucilia sericata* (the latter species being that most widely used for MDT), and to develop disinfection, transport and application procedures. Our techniques were based on Ronald Sherman's proven methodology, and we now supply disinfected *Lucilia sericata* maggots for local, interstate, and overseas patients. We are the sole supplier in Australia. Most of the requests we have serviced have involved patients where a final effort was being made to save limbs or to heal massive wounds, but the maggots have also been used in more routine wound management cases, including for burns.

The increasing incidence of soft-tissue wounds in nursing homes and hospitals, and the increasing prevalence of diabetes mellitus in the general community, suggest that antibiotic-resistant skin infections need to be addressed both at the level of population health and clinical practice.[13]

Although not all patients are suited to MDT therapy, it is an efficient, low-cost alternative method to cleanse and promote the healing of chronic soft tissue wounds before they progress to a stage where amputation is the only alternative. Although use of MDT is not yet widespread in Australia, perhaps a new image of maggots will emerge and they may become more widely recognised and accepted as an effective and economic means of treating wounds and saving limbs for patients in our health system.

For further information on Maggot Debridement Therapy visit **www.medent.usyd.edu.au/projects/maggott.htm** (Institute of Clinical Pathology and Medical Research, Westmead, New South Wales, Australia); **www.ucihs.uci.edu/com/pathology/sherman/home_pg.htm** (Veterans Affairs Medical Center, Los Angeles, California, United States): and **www.larve.com** (Princess of Wales Hospital, Bridgend, Wales, United Kingdom).

REFERENCES

1. Sherman RA. Maggot debridement in modern medicine. *Infect Med* 1998; Sept: 651–6.
2. Baer WS. The treatment of chronic osteomyelitis with the maggot (larva of the blow fly). *J Bone Joint Surg Am* 1931; 13: 438–75.
3. Child FS, Roberts EF. The treatment of chronic osteomyelitis with live maggots. *New York State J Med* 1931; 31: 937–43.
4. Teich S, Myers RAM. Maggot therapy for severe skin infections. *South Med J* 1986; 79: 1153–5.
5. Church JCT. Larva therapy in modern wound care: A review. *Primary Intention* 1999; May: 63–8.
6. Sherman RA, Hall MJR, Thomas S. Medicinal maggots: An ancient remedy for some contemporary afflictions. *Ann Rev Entomol* 2000; 45: 55–81.
7. Sherman RA. A new dressing design for use with maggot therapy. *Plast Reconstr Surg* 1997; 100: 451–6.
8. Thomas S, McCubbin P. Use of maggots in the care of wounds. *Hosp Pharmacist* 2002; 9: 267–71.
9. Sherman RA. Maggot therapy for treating diabetic foot ulcers unresponsive to conventional therapy. *Diabetes Care* 2003; 26: 446–51.
10. Vistnes LM, Lee R, Ksander GA. Proteolytic activity of blowfly larvae secretions in experimental burns. *Surgery* 1981; November: 835–41.
11. Erdmann GR. Antibacterial action of myiasis-causing flies. *Parasitol Today* 1987; 3(7): 214–6.
12. LarvE Biosurgical Research Unit. Maggots on prescription [press release]. Available at www.larve.com (accessed 3 January 2005).
13. Bowler PG, Duerden BI, Armstrong DG. Wound microbiology and associated approaches to wound management. *Clin Microbiol Rev* 2001; 14: 244–69.

COMMUNICABLE DISEASES REPORT, NSW, FOR SEPTEMBER–OCTOBER 2004

For updated information, visit the website **www.health.nsw.gov.au** and click on the link to Infectious Diseases.

TRENDS

The NSW Department of Health's infectious diseases webpage has recently been enhanced to include graphs and tables showing the number of cases of various infectious diseases notified by doctors, hospitals, and laboratories. Data are usually updated on a daily basis, and show aggregate cases by the age and sex over time. To check reports for a particular disease, click on **www.health.nsw.gov.au/living/infect.html**, scroll down the table to the disease of interest, and click on the column headed 'NSW data'.

Tables 2 and 3 and Figure 1 show reports of communicable diseases received through to September and October 2004 in NSW. These data show a upswing in reports of **pertussis** from March through to September 2004, and a subsequent decline (see www.health.nsw.gov.au/data/diseases/pertussis.html). Over the last year, the group with the most notifications has been children aged 10–14 years. To help control ongoing outbreaks in this group, The NSW Department of Health has offered Australian Government-funded vaccination to high school children against pertussis, diphtheria and tetanus since mid-2004 (see **www.health.nsw.gov.au/living/immunisation/school_prog/index.html for details**). Pertussis can cause a serious illness that is characterised by a long lasting cough that can occur in bouts. Coughing bouts are sometimes followed by vomiting, or a gasping for air (or 'whoop'). In older children and adults, the ongoing bouts of coughing may be the only complaint. People with these symptoms should seek advice from their doctor, who can make a diagnosis (often with the assistance of laboratory tests of blood or respiratory sample tests) and if necessary, prescribe specific antibiotics to help prevent the further spread of the disease. Close contacts of patients with pertussis should watch out for symptoms, and those at high risk may need to take specific antibiotics to prevent infection. For more information, see **www.health.nsw.gov.au/pubs/2004/pert_cdfs.html**.

The expected seasonal increase in reports of **meningococcal disease** occurred in spring, although the winter–spring peak in 2004 was less than that seen in 2003 (see **www.health.nsw.gov.au/data/diseases/meningococcal.html**).

INFLUENZA OUTBREAKS IN RESIDENTIAL FACILITIES

In September 2004, 13 outbreaks of influenza-like illness were reported from residential institutions in 6 of the 17 area health service in NSW, including 12 aged care facilities and 1 correctional centre (Table 1). While institutions are not required to report influenza outbreaks under the *NSW Public Health Act*, reporting to public health units is encouraged to facilitate the prompt implementation of control measures.

Intervention

In response to these outbreaks, public health unit staff provided advice to facility managers on control measures.

TABLE1

REPORTS OF INFLUENZA-LIKE OUTBREAKS IN INSTITUTIONS, NEW SOUTH WALES, SEPTEMBER 2004

Facility	Area health service	Influenza-like illness in residents		Influenza-like illness in staff		Influenza confirmed in residents	Deaths in residents	
		N	%	*N*	%		*N*	%
ACF A	Hunter	38/50	76	20/59	34	Influenza A (3)	10	20
ACF B	Hunter	18/40	45	*		Influenza A (5)	*	
ACF C	Hunter	42/68	62	19/88	22	No	8	12
ACF D	Hunter	5/85	6	7/109	6	Influenza A (1)	*	
ACF E	Hunter	20/100	20	9/90	10	Influenza A (1)	*	
ACF F	Hunter	3/100	3	0/100	0	Influenza A (1)	*	
ACF G	Western Sydney	24/49	49	5/49	10	Influenza A (4)	2	4
ACF H	Wentworth	24/68	35	16/100	16	No	*	
ACF I	South Eastern Sydney	32/98	33	3/90	3	Influenza A (5) Influenza B (5)	2	2
ACF J	Northern Sydney	11/48	23	0/60	0	Influenza A (1)	*	
ACF K	Northern Sydney	20/57	35	25/60	42	Influenza A (1)	8	14
ACF L	Central Sydney	29/42	69	10/50	20	Influenza A (4)	4	10
Correctional Centre		23 (inmates)		1		Influenza A (6)	*	

ACF = aged care facility; * = none reported

The NSW Department of Health developed guidelines to assist managers of aged care facilities to minimise the spread of influenza within their institutions. The guidelines—*Controlling influenza outbreaks in aged care facilities*—was distributed by fax to ACFs throughout NSW, and included advice on:

- confirming the cause of respiratory outbreaks;
- forming an outbreak control team;
- isolating ill residents and restricting staff and visitors;
- performing regular hand hygiene;
- wearing masks when caring for ill residents;
- wearing gloves if contacting contaminated materials;
- wearing impervious gowns to protect clothing;
- enhancing cleaning;
- considering the use of anti-influenza medications for prophylaxis;
- immunising all staff and residents annually.

Comment

These outbreaks highlight the high attack rates (up to 76 per cent of residents and 42 per cent of staff) and death rates (up to 20 per cent in residents) associated with influenza outbreaks in aged care facilities, where residents at high risk for severe disease (because of their older age or concurrent illness) are clustered together.

The WHO Collaborating Centre for Influenza in Melbourne, reports that A/Fujian/411/2002-like (90 per cent) and B/Shanghai/361/2002-like (9 per cent) have been the most commonly identified influenza virus types identified in Australia through 2004. Data from NSW Health's Influenza Surveillance Report (see **www.health.nsw.gov.au/living/flureport.html**) suggest that influenza activity in the wider NSW community—as measured by attendance for influenza-like illness to selected general practitioners and emergency departments, as well as influenza diagnoses by major laboratories—began to increase in September in 2004, which is later than usual. However, influenza activity to date in 2004 appears lower than in 2002 and 2003 (when the Fujian strain of influenza A first appeared).

In previous years, NSW Health has not actively solicited reports of influenza outbreaks from institutions, or systematically collated information on reported outbreaks. The reasons for the apparent large number (13) reported in NSW in September 2004, and the large proportion of these reported from the Hunter Area are unclear, but one explanation could be improved reporting in 2004 following the release of the guidelines *Controlling influenza outbreaks in aged care facilities*. In addition, the first outbreak in the Hunter Area (in aged care facility A) was accompanied by substantial media interest that may in turn have led to improved reporting by other aged care facilities.

NSW Health provides Australian Government-funded influenza vaccine annually to residents of aged care facilities. However, in investigating many of these outbreaks, public health units found that residents' immunisation records did not provide clear evidence of vaccination, perhaps because the turnover of residents in aged care facilities was sometimes high and the immunisation status of new residents was not always assessed on admission.

The annual immunisation of both residents and staff before winter (when the influenza activity usually begins) is vital for limiting the extent of such outbreaks, even though vaccine efficacy declines in older people.[1] Managers of aged care facilities should ensure that record systems are in place to document the vaccination status of residents and staff, and flag the records of new residents and staff to ensure that they are offered immunisation. With growing evidence that anti-influenza medicines are effective in containing outbreaks,[1] managers of aged care facilities and clinicians should strongly consider their use to limit the spread of the infection in residential facilities.

Reference

1. Centers for Disease Control and Prevention. Prevention and control of influenza: Recommendations of the Advisory Committee on Immunization Practices. *MMWR* 2003; 52(RR-8) available online at www.cdc.gov/mmwr/PDF/rr/rr5208.pdf.

UPDATE ON AVIAN INFLUENZA

There has been a recent increase in the reported number of outbreaks of highly pathogenic avian influenza (HPAI) among poultry flocks in Asia. Since the beginning of the epidemic in January 2004, (as of 5 October) 16 human cases (including 11 deaths) of laboratory-confirmed HPAI have been reported in Thailand, and 27 (including 20 deaths) have been reported in Vietnam. Most cases in humans are thought to have been acquired from direct or indirect exposures to infected poultry.

However, a probable case of person-to-person transmission of HPAI was reported in Thailand in September 2004. This transmission resulted from sustained, close personal contact between a mother and her sick child, and not because the virus had changed into a form that would facilitate the ready transmission from one person to another. Such transmission remains highly unusual. Disease control experts are concerned that while the HPAI epidemic in poultry smolders on, the propensity of the influenza virus to mutate will lead to a form that is more easily transmitted from person-to-person. If this were to occur, there is a possibility that an influenza pandemic could ensue.

Disease control activities in Asia to date have centred on active surveillance, culling of infected flocks, import–export restrictions, and movement restrictions around infected farms. Large-scale vaccination of poultry has been advocated by some. Work to develop a human vaccine for the current strain in birds is ongoing. For updated information see the World Health Organization's website **www.who.int/csr/disease/avian_influenza/en**.

SALMONELLOSIS CLUSTER

In September, the NSW Department of Health was notified by the NSW Food Authority of gastrointestinal illness in children attending a birthday party. To identify the likely source of the illness, the Communicable Diseases Branch conducted a cohort study of those attending the party. Each person was asked for details of illness and the foods they ate at the party.

Four adults and 13 children aged from 12 to 15 years attended the party. Eleven of the children reported gastrointestinal illness following the party with symptoms including diarrhoea, fever and abdominal pain. None of the adults reported illness. *Salmonella* Typhimurium phage type 126 was isolated in stool specimens taken from 2 children.

Foods served at the party included commercially prepared pizza, sausage rolls, various chips and chocolates, and home-made tiramisu (an Italian dessert made with sponge, mascarpone cheese and raw egg). The association between each food eaten at the party and illness was calculated. The tiramisu was strongly associated with illness. Eleven of the 12 people who reported eating the tiramisu became ill (an attack rate of 92 per cent), while none of 6 who did not eat the tiramisu reported illness. There was no association between consuming other foods and reported illness. The 1 person who ate tiramisu and did not become ill was an adult who reported eating only a spoonful of the desert.

There are a number of ways food can be contaminated in the home. Cross-contamination from raw meat products to ready-to-eat foods, and undercooking contaminated foods are common causes of foodborne outbreaks. An environmental investigation is underway to better define the source of contamination of the tiramisu in collaboration with the NSW Food Authority.

UNUSUAL *SALMONELLA* SEROVAR AND EXPOSURE TO CATTLE

Peter Massey and Kylie Taylor

New England Public Health Unit

During April and September 2004, the New England Public Health Unit received 2 notifications of unusual serovars of *Salmonella*: *S.* Meleagridis and *S.* Stanley.

Salmonellosis can be a severe illness, characterised by sudden onset of headache, fever, abdominal pain, diarrhoea, nausea and sometimes vomiting. Dehydration can be severe, especially in the elderly or in infants. Complications such as septicaemia or localised infections can also occur. Death from salmonellosis is uncommon but morbidity associated with the infection can be substantial.[1]

There are over 1,800 known *Salmonella* serovars that current classification considers to be separate species. *S.* Typhimurium and *S.* Enteritidis serovars cause the large majority of human infections.

In NSW, a diagnosis of salmonellosis by laboratories is notifiable to public health units. To determine the cause of illness, the New England Public Health Unit investigated each case and their likely source of infection.

Investigation

Case 1

The notification in April 2004 was of a 3-month old child with *S.*Meleagridis infection. The child was reported by a general practitioner to have a fever and diarrhoea. The child was fully breastfed. The investigation identified that the child was washed in the shower while being held by its mother. The child ingested water by sucking it from its mother's arms while in the shower. The water supply to the house came directly from a creek and is untreated. Cattle cross the creek upstream from where the water is sourced.

Case 2

In September 2004, a 26-year old person with *S.*Stanley infection was notified to the New England Public Health Unit. The person was reported to have had a diarrhoeal illness for approximately 6 months. The case was interviewed for possible exposures. The most likely source of infection was frequent exposure on the hands and face to water used for cleaning at an abattoir.

Discussion

The National Enteric Pathogen Surveillance Scheme (NEPSS) reports that no human cases of *S.*Meleagridis have been recorded since 2001. The serovar has been found in some foods during 2002, in chicken litter and equine intestine,[2] in tree nuts and meat–bone meal.[3] Testing of the water into the house of the case with *S.* Meleagridis did not reveal any *Salmonella* bacteria. As no other possible exposure could be found, it is hypothesised that the child was infected from the water source via the shower.

*S.*Stanley is reported to be more common than *S.*Meleagridis. The NEPSS report that in NSW there were 12 cases reported to the end of October 2004, 11 cases in 2003, and 15 cases in 2002. The serovar has been found in some food sources, mainly porcine in recent years, but also in macadamia nuts and imported Chinese peanuts in 2001.[4] The serovar has also been reported from faecal samples of various animals.

The investigation into these unusual serovars highlight some issues about the surveillance and control of *Salmonella*:

- unusual serovars can indicate unusual types of exposures;
- direct or indirect contact with cattle may result in infection with *Salmonella*;
- untreated water may provide a vehicle for spreading the bacteria.

References

1. Heymann DL (editor). *Control of Communicable Diseases Manual: 18th Edition*. Washington, DC: American Public Health Association, 2004.
2. Davos D (editor). Australian *Salmonella* Reference Centre 2002 Annual Report. Adelaide: Institute of Medical and Veterinary Science, 2002.
3. Davos D (editor). Australian *Salmonella* Reference Centre 2003 Annual Report. Adelaide: Institute of Medical and Veterinary Science, 2003.
4. Davos D (editor). Australian *Salmonella* Reference Centre 2001 Annual Report. Adelaide: Institute of Medical and Veterinary Science, 2001.

Q FEVER CLUSTER IN A SHEARING TEAM

Peter Massey and Kylie Taylor
New England Public Health Unit

During August 2004, the New England Public Health Unit received 10 notifications of Q fever, representing a monthly rate (64.8/100000 population), well above the rate for all of 2003 (34.0/100000).

Q fever is sometimes a severe illness characterised by sudden onset of fever, chills, headache, weakness and sweats. Pneumonitis, hepatitis, endocarditis and neurological complications may follow. It is caused by infection with *Coxiella burnetii*, a rickettsia, which is commonly carried by a range of farm and wild animals. The infection is transferred to humans when they inhale droplets that are contaminated with the bacteria and which become aerosolised during the slaughter of an infected animal or through the discharge of products (urine, faeces, milk, and birth by-products) of an infected animal.[1] Q fever is mainly an occupationally-acquired disease in workers in the livestock, agriculture, and meat industries.

The abattoir industry in NSW has had an immunisation program in place for a number of years and subsequently the proportion of Q fever notifications in abattoir workers has decreased.[2] Cases are now predominately people working on the land who are associated with the livestock industry.[3]

In NSW, a diagnosis of Q fever by laboratories is notifiable to public health units. To determine the cause of the increase in notifications in August, the New England Public Health Unit investigated each case and their likely source of infection.

Investigation

The notifications in August 2004 for New England were of 9 males and 1 female. The age of these cases was 35–55 years. Each case had presented to their general practitioner. Reported symptoms included fever, chills, headache, myalgia, arthralgia, nausea and lethargy. One case required hospitalisation for pneumonia. All the cases reported that their illnesses lasted between weeks and months, and prevented them from working.

Five cases reported common risk factors: all worked on the same shearing team and had onset of their illness during a 3 week period in June–July 2004. The shearing team comprised 10 workers who had been shearing in the same shed at a property for some time. Two other members of the team were also reported to have signs and symptoms consistent with Q fever but did not seek medical advice. One case was the replacement shearer for another member who was off work with the illness. The attack rate in the team was 64 per cent (7/11).

Only 1 person in the shearing team reported having been immunised against Q fever, even though the Q fever immunisation had been available free as part of the national program. Two of the cases reported that some of the sheep in contact with the shearers at this property were lambing during the period of exposure.

Comment

Q fever remains a problem in rural NSW. In 2004 through to September, Macquarie (62.4/100000), New England (29.07/100000), and Far West (26.48/100000 areas all reported significantly higher notifications rates for Q fever than for NSW as a whole. Identification of clusters requires either the patient to be alert to the possibility, clinician reporting links among cases, or a public health investigation. In this cluster, because each case presented to a different doctor, it is unlikely that it would have been identified without a public health investigation.

Both direct and indirect contact with sheep has been shown to be a risk factor in outbreaks of Q fever mainly through airborne transmission.[4,5] High concentrations of *C. burnetii* are found in the birth by-products of infected animals.[6] Shearing brings people into very close contact with sheep and the confines of a shearing shed may also have contributed to the exposure.

We were unable to find data describing the immunisation rate among shearers, but this cluster indicates that there are agricultural workers who remain susceptible to this potentially debilitating disease.

This cluster highlights a number of issues about Q fever:

- the high attack rate in the 1 shed suggests that members of the shearing team may have had an exposure from contact with the infected sheep and the environment of the shed may have increased this exposure;

- Q fever can cause an illness that is severe enough to interfere with shearing work and thus have economic consequences for rural families and communities;
- investigation of Q fever can detect clusters and contribute to our understanding of the disease and its risks;
- Q fever immunisation research into the barriers to the uptake of the immunisation by this group should be identified.

References

1. Centre for Epidemiology and Research, NSW Department of Health. *N S W Public Health Bull* 2002; 13(8): 191.
2. New England Public Health Unit (unpublished notifiable diseases data).
3. Boland PJ, Parker NR. Q fever in south west Queensland. *Med J Aust* 1999; 171(8): 446.
4. Manfredi et al. Investigation of a Q-fever outbreak in northern Italy. *Eur J Epi* 1996; 12(4): 403–8.
5. Lyytikainen et al. An outbreak of sheep associated Q fever in a rural community in Germany. *Eur J Epi* 1998; 14(2): 193–9.
6. Chin J (editor). *Control of Communicable Diseases Manual: 17th Edition*. Washington, DC: American Public Health Association, 2000; 407–411.

FIGURE 1

REPORTS OF SELECTED COMMUNICABLE DISEASES, NSW, JAN 1999 TO OCT 2004, BY MONTH OF ONSET

Preliminary data: case counts in recent months may increase because of reporting delays.

Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis

BFV = Barmah Forest virus infections, RRV = Ross River virus infections

lab+ = laboratory confirmed

Men Gp C and Gp B = meningococcal disease due to serogroup C and serogroup B infection, other/unk = other or unknown serogroups.

NB: multiple series in graphs are stacked, except gastroenteritis outbreaks.

NB. Outbreaks are more likely to be reported by nursing homes & hospitals than from other institutions

NSW population	
Male	50%
<5	7%
5–24	28%
25–64	52%
65+	13%
Rural*	42%

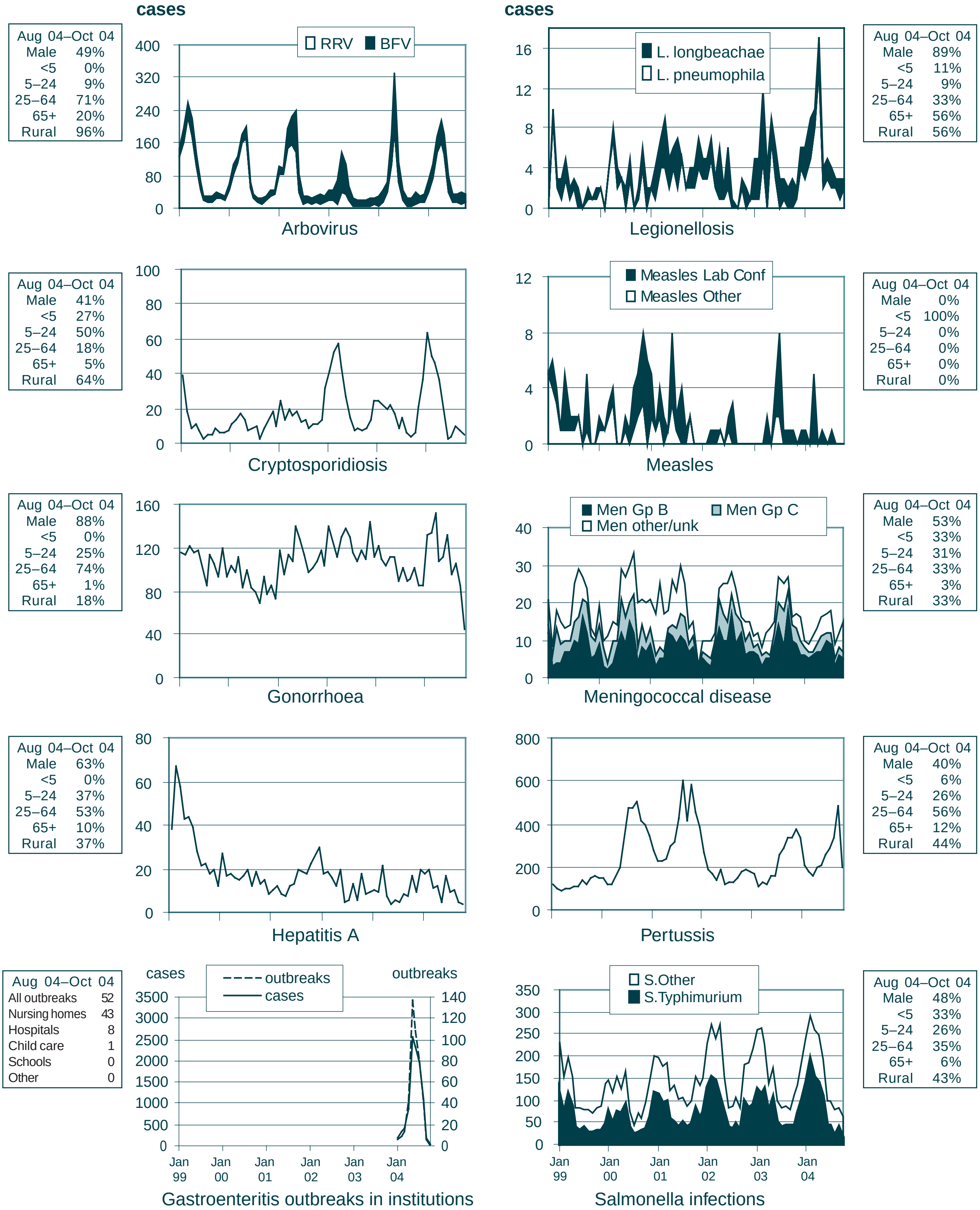


TABLE 2 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN SEPTEMBER 2004 BY AREA HEALTH SERVICES

Condition	Area Health Service																		Total	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	CHS	for Sept†	To date†
Blood-borne and sexually transmitted																				
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	2	46	36	18	69	25	70	15	159	46	35	104	21	23	13	22	14	1	731	7272
Gonorrhoea*	9	5	3	2	3	1	5	1	44	3	2	12	1	-	1	-	-	-	93	999
Hepatitis B—acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	37
Hepatitis B—other*	18	31	61	5	96	7	1	5	41	3	3	-	1	4	2	1	2	2	285	2968
Hepatitis C—acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	15
Hepatitis C—other*	54	19	52	30	85	39	29	25	84	25	38	9	10	12	2	11	7	9	546	5203
Hepatitis D—unspecified*	-	-	1	-	1	-	-	1	-	-	-	-	-	-	-	-	-	1	4	11
Syphilis	20	1	8	2	30	4	4	2	43	1	1	2	-	2	5	2	-	-	127	917
Vector-borne																				
Barmah Forest virus*	-	-	-	-	-	1	3	-	-	7	16	1	-	-	-	-	-	-	28	305
Ross River virus*	-	1	-	-	-	2	2	-	-	-	6	-	-	-	1	-	-	-	12	627
Arboviral infection (Other)*	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	1	31
Malaria*	-	1	3	-	1	-	-	-	3	-	-	1	-	-	-	-	-	-	9	68
Zoonoses																				
Anthrax*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis*	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	5
Leptospirosis*	-	-	-	-	-	-	1	-	-	-	-	2	1	-	-	-	-	-	4	30
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis*	-	-	-	-	1	-	3	-	-	1	-	-	-	1	-	2	-	-	8	55
Q fever*	-	-	2	-	-	1	2	-	1	2	-	3	9	-	2	-	1	-	23	164
Respiratory and other																				
Blood lead level*	1	-	-	1	5	1	-	1	-	1	2	1	4	-	2	-	-	-	19	220
Influenza*	7	27	41	7	38	3	16	6	91	8	-	-	3	3	-	1	1	2	254	515
Invasive pneumococcal infection*	5	8	22	2	15	4	11	7	19	3	4	-	-	2	1	1	-	-	104	724
Legionella longbeachae infection*	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1	20
Legionella pneumophila infection*	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	39
Legionnaires- disease (Other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4
Meningococcal infection (invasive)*	1	2	1	-	-	1	-	1	3	1	-	-	-	-	1	-	1	-	12	117
Tuberculosis	6	3	4	-	2	1	2	-	6	-	1	-	-	-	-	-	1	1	28	281
Vaccine-preventable																				
Adverse event after immunisation**	1	-	-	-	2	1	-	1	4	-	-	1	-	1	-	3	1	-	15	148
H. *Influenzae b* infection (invasive)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3
Measles	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	9
Mumps*	-	1	-	-	-	-	-	1	-	-	-	-	1	-	-	-	-	-	3	40
Pertussis	42	39	50	25	52	14	51	16	78	18	10	11	75	10	3	18	7	-	519	2365
Rubella*	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	15
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Enteric																				
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Cryptosporidiosis*	-	-	1	-	-	-	-	-	-	-	1	1	1	2	-	-	-	-	6	257
Giardiasis*	4	20	15	7	8	1	5	4	7	1	4	4	1	4	2	1	5	-	93	971
Haemolytic uraemic syndrome	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5
Hepatitis A*	1	2	3	-	-	2	-	1	1	-	1	-	-	-	-	-	-	-	11	123
Hepatitis E*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	6
Listeriosis*	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	23
Salmonellosis*	6	7	10	5	7	1	4	4	8	8	7	4	2	1	3	1	1	-	79	1658
Shigellosis*	-	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	65
Typhoid and paratyphoid*	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	26
Verotoxin producing *E. coli**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Miscellaneous																				
Creutzfeldt-Jakob disease	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4

* lab-confirmed cases only + includes cases with unknown postcode * * HIV and AIDS data are reported separately in the *NSW Public Health Bulletin* each quarter

** AEFIs notified by the school vaccination teams during the National Meningococcal C Program are not included in these figures. These notifications are reviewed regularly by a panel of experts and the results will be published quarterly in the *NSW Public Health Bulletin* in 2004

CSA = Central Sydney Area
NSA = Northern Sydney Area
WSA = Western Sydney Area
WEN = Wentworth Area
SWS = South Western Sydney Area
CCA = Central Coast Area
HUN = Hunter Area
ILL = Illawarra Area
SES = South Eastern Sydney Area
NRA = Northern Rivers Area
MNC = North Coast Area
NEA = New England Area
MAC = Macquarie Area
MWA = Mid Western Area
FWA = Far West Area
GMA = Greater Murray Area
SA = Southern Area
CHS = Corrections Health Service

TABLE 3 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN OCTOBER 2004 BY AREA HEALTH SERVICES

Condition	Area Health Service																		Total	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	CHS	for Oct†	To date†
Blood-borne and sexually transmitted																				
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	72	95	67	18	50	25	84	37	185	22	21	27	11	16	2	29	18	-	787	8219
Gonorrhoea*	13	7	10	-	3	3	4	4	164	2	2	-	-	-	-	-	-	-	213	1228
Hepatitis B—acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	37
Hepatitis B—other*	2	37	46	4	78	4	10	4	34	2	1	1	1	1	2	1	2	2	233	3257
Hepatitis C—acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	14
Hepatitis C—other*	67	23	41	20	79	32	36	21	61	17	20	8	7	19	1	12	16	7	464	5768
Hepatitis D—unspecified*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	11
Syphilis	15	11	12	2	14	-	2	3	31	1	-	1	-	-	2	-	-	-	94	986
Vector-borne																				
Barmah Forest virus*	-	1	-	-	-	-	4	1	-	4	14	-	-	-	-	-	-	-	24	329
Ross River virus*	-	-	-	-	-	-	4	1	-	1	6	2	2	-	-	-	-	-	16	643
Arboviral infection (Other)*	-	-	-	-	-	-	-	-	2	-	1	-	-	-	-	-	1	-	4	35
Malaria*	-	-	2	-	2	1	1	1	1	-	-	-	-	-	-	3	-	-	11	78
Zoonoses																				
Anthrax*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis*	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	1	6
Leptospirosis*	-	-	-	-	-	-	1	-	-	-	-	1	-	-	-	-	-	-	2	32
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis*	-	-	-	1	-	-	2	1	-	1	-	-	1	-	-	1	-	-	7	63
Q fever*	-	1	-	-	-	-	1	1	-	2	-	2	9	-	1	-	1	-	18	182
Respiratory and other																				
Blood lead level*	-	1	1	-	2	-	9	3	-	2	-	-	-	1	-	-	-	-	19	254
Influenza*	12	22	28	3	14	1	43	4	52	10	2	1	2	4	-	1	3	-	202	727
Invasive pneumococcal infection*	4	7	3	5	6	4	8	5	7	-	-	-	1	1	-	3	2	-	56	783
Legionella longbeachae infection*	-	-	1	-	-	-	-	1	1	-	-	-	-	-	-	-	-	-	3	23
Legionella pneumophila infection*	-	-	-	-	-	-	-	1	-	-	-	-	-	1	-	-	-	-	2	42
Legionnaires- disease (Other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4
Meningococcal infection (invasive)*	2	6	2	-	3	-	1	-	2	-	-	-	-	-	-	1	-	-	17	134
Tuberculosis	6	1	-	2	23	-	1	-	10	-	-	-	-	-	-	-	-	-	43	337
Vaccine-preventable																				
Adverse event after immunisation**	-	-	1	-	-	1	3	-	-	1	-	-	-	-	-	1	-	-	7	155
H. *Influenzae b* infection (invasive)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3
Measles	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	9
Mumps*	-	-	-	-	2	-	2	-	1	-	-	-	-	-	-	-	-	-	5	46
Pertussis	28	35	36	30	34	8	56	19	58	6	11	7	44	7	-	36	8	-	423	2797
Rubella*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	16
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Enteric																				
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Cryptosporidiosis*	-	-	2	-	-	-	1	-	2	2	-	-	2	-	-	-	-	-	9	266
Giardiasis*	7	11	13	4	4	6	4	2	13	-	2	3	1	5	-	1	-	-	76	1051
Haemolytic uraemic syndrome	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	1	6
Hepatitis A*	1	-	-	-	2	-	-	-	-	2	-	-	-	-	-	-	-	-	5	126
Hepatitis E*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	6
Listeriosis*	1	1	-	-	-	-	-	-	2	-	-	-	-	-	-	-	-	-	4	27
Salmonellosis*	5	14	8	5	11	1	7	3	10	6	9	5	2	-	-	4	4	-	94	1756
Shigellosis*	4	-	-	-	-	-	-	-	1	1	3	3	-	-	-	-	-	-	12	78
Typhoid and paratyphoid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	24
Verotoxin producing *E. coli**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Miscellaneous																				
Creutzfeldt-Jakob disease	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4

* lab-confirmed cases only + includes cases with unknown postcode * * HIV and AIDS data are reported separately in the *NSW Public Health Bulletin* each quarter

** AEFIs notified by the school vaccination teams during the National Meningococcal C Program are not included in these figures. These notifications are reviewed regularly by a panel of experts and the results will be published quarterly in the *NSW Public Health Bulletin* in 2004

CSA = Central Sydney Area
NSA = Northern Sydney Area
WSA = Western Sydney Area
WEN = Wentworth Area
SWS = South Western Sydney Area
CCA = Central Coast Area
HUN = Hunter Area
ILL = Illawarra Area
SES = South Eastern Sydney Area
NRA = Northern Rivers Area
MNC = North Coast Area
NEA = New England Area
MAC = Macquarie Area
MWA = Mid Western Area
FWA = Far West Area
GMA = Greater Murray Area
SA = Southern Area
CHS = Corrections Health Service

2004 INDEX BY SUBJECT

2003 INDEX BY AUTHOR

T

U

W

NSW PUBLIC HEALTH BULLETIN

The *NSW Public Health Bulletin* is a publication of the NSW Department of Health.
The editor is Dr Lynne Madden, Manager, Public Health Training and Development Branch.
Dr Michael Giffin is the managing editor.

The Bulletin aims to provide its readers with population health data and information to support effective public health action.

The Bulletin is indexed by MEDLINE and *Index Medicus*.

Submission of articles

The preferred length of Bulletin articles is 1500 words. Tables and figures may be additional to that.

News, comments, and other reports should be 500–600 words.

All manuscripts should contain a short introductory abstract that reflects the structure of the manuscript.

References should be set out in the Vancouver style.

Send submitted manuscripts on paper and in electronic form, either on disc (Word for Windows is preferred), or by email.

The manuscript must be accompanied by a letter signed by all authors.

Full instructions for authors are available on request from the managing editor.

Editorial correspondence

Please address all correspondence and potential contributions to The Managing Editor, *NSW Public Health Bulletin,* Locked Mail Bag 961, North Sydney, NSW 2059, Australia or by email to phbulletin@doh.health.nsw.gov.au. Tel: 61 2 9391 9241, Fax: 61 2 9391 9232.

Distribution

To obtain copies of the *NSW Public Health Bulletin* please contact your local public health unit or by telephone at 61 2 9391 9942.

A new subscribers–change of address form is printed in most issues of the Bulletin. There is also an online subscription form available at the Bulletin's website.

The Bulletin can be accessed in both PDF and HTML formats from **www.health.nsw.gov.au/public-health/phb/phb.html**.

All back issues are downloadable from the website. Back issues of the printed version can be obtained from:
Public Health Training and Development Branch
NSW Department of Health
Locked Mail Bag 961
North Sydney, NSW 2059, Australia.

NSW HEALTH
Working as a Team

NEW SUBSCRIBERS–CHANGE OF ADDRESS FORM

Please return to:

NSW Public Health Bulletin
NSW Department of Health
Locked Mail Bag 961, North Sydney NSW 2059, Australia

Fax: 61 2 9391 9232

SUBSCRIBE AND UNSUBSCRIBE

☐ I wish to be placed on the mailing list

☐ Please remove me from the mailing list

Name: ____________________

Organisation: ____________________

Mailing address: ____________________

____

City: __________ State: __________

Postcode: __________ Country: __________

Telephone: __________ Facsimile: __________

Email: ____________________

CHANGE OF ADDRESS

☐ I wish to change my mailing details, as follows:

FROM: ____________________

____

____

____

TO: ____________________

____

____

____

SUBSCRIBE TO THE WEB VERSION

The *Bulletin* can be accessed via the Internet from our Web site at **www.health.nsw.gov.au/public-health/phb/phb.html**. If you would like to be informed by email when new issues of the *Bulletin* become available, please subscribe to the Internet mailing list when you next visit the site.