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POST-EXPOSURE PROPHYLAXIS FOR NON-OCCUPATIONAL EXPOSURE TO HIV: EXPERIENCE IN NSW ONE YEAR AFTER THE INTRODUCTION OF THE GUIDELINES

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In December 1998, the NSW Department of Health released guidelines for the use of post-exposure prophylaxis (PEP) in situations of non-occupational exposure to HIV.[1] This report summarises the first year's results of the study that was instituted as part of these guidelines to monitor the use of PEP.

BACKGROUND

Only a few health jurisdictions in the world have published policies recommending PEP in the context of non-occupational exposures, although it is recognised that PEP is used informally in non-occupational settings in a number of countries. The NSW Department of Health is currently the only health jurisdiction in Australia to provide guidelines recommending PEP for non-occupational exposures to HIV (see Box for a description of the stages of HIV reproduction). Some other states and territories have guidelines in development.

Although there is no direct evidence from randomised controlled trials of the efficacy of PEP for HIV, other data suggest that PEP may be effective.[2] A case-controlled study of health care workers reported a 79 per cent reduction in the risk of seroconversion with zidovudine (a nucleoside analogue reverse transcriptase inhibitor) PEP treatment.[3] (See Table 1 for more detail on types of antiviral drugs and their modes of action). Animal studies also suggest that PEP may successfully prevent HIV infection.[4] Randomised controlled trials have demonstrated that antiretroviral treatment

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decreases mother-to-child transmission of HIV,[5] and part of this effect appears to be through post-exposure prophylaxis in the infant. Evidence to support this has come from a recent study, which found that a single dose of nevirapine (a non-nucleoside reverse transcriptase inhibitor) administered intrapartum, followed by a single dose to the newborn infant reduced HIV transmission by almost 50 per cent.[6]

HIV post-exposure prophylaxis has been available for some time for occupational exposures among health care workers in NSW.[7] However, more than 90 per cent of HIV transmission episodes in Australia occur in non-occupational settings through sexual activity, and up to five per cent through injecting drug use.[8] The risks of transmission associated with unprotected intercourse and needle sharing in discordant couples (where one individual is HIV antibody positive and the other is HIV antibody negative) have been estimated to be at least as high as the risks of transmission in occupational exposures.[9]

PEP against HIV infection comprises four weeks of therapy with highly specialised antiretroviral drugs. In the NSW Department of Health guidelines, two nucleoside analogue reverse transcriptase inhibitors are recommended for most situations, with the addition of a protease inhibitor for certain particularly high risk exposures (Table 2). The cost of this treatment to the health care system is approximately $600 for double- and $1000 for triple-combination therapy.

The types of exposures recommended for PEP in the guidelines include percutaneous and mucous membrane exposures, which may occur from sexual and injecting drug use behaviours. In all situations, the possibility that the source is infected with HIV and the nature of the exposure must be weighed up.

For percutaneous exposures, where the source is known to be HIV positive and with a significant blood exposure to high HIV titre, PEP comprising triple-combination therapy (two nucleoside reverse transcriptase inhibitors and a protease inhibitor) is recommended. PEP is also recommended for unprotected receptive or insertive anal or vaginal intercourse, using two nucleoside analogues in most circumstances, with the addition of a protease inhibitor in situations of particularly high risk.

The guidelines suggest that PEP might be considered in other circumstances, such as percutaneous exposures to blood-stained fluid, and significant mucous membrane exposure to blood or blood-stained fluid. It is recommended not to offer PEP for exposures to non blood-stained fluids, exposures to intact skin, or for needle-stick injuries from discarded injecting equipment.

Observational study of non-occupational PEP in NSW

In view of the limited evidence regarding the efficacy of PEP, the December 1998 guidelines from the NSW Department of Health recommended that the use of this treatment be closely monitored. A study was initiated to monitor implementation of the guidelines, which is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR) and the National Centre in HIV Social Research. This study has been approved by the research ethics committee at the University of New South Wales as well as by individual NSW Area Health Services. Coordination of the study was possible through an unrestricted grant from GlaxoWellcome.

METHODS

In NSW, medical practitioners operating outside hospital HIV specialist units may prescribe specialised drugs for the treatment of HIV infection provided they are registered with the HIV Prescribers Project. This project is funded by the NSW Department of Health and

STAGES OF HIV REPRODUCTION

1. HIV enters a CD4+ cell.
2. HIV is a retrovirus, meaning that its genetic information is stored on single-stranded RNA instead of double-stranded DNA found in most organisms. To replicate, HIV uses an enzyme known as reverse transcriptase to convert its RNA into DNA.
3. HIV DNA enters the nucleus of the CD4+ cell and inserts itself into the cell's DNA. HIV DNA then instructs the cell to make many copies of the original virus.
4. New virus particles are assembled and leave the cell ready to infect other CD4+ cells.

TABLE 1

TYPES OF POST-EXPOSURE PROPHYLAXIS AND THEIR MODE OF ACTION.

Class–Drug	Mode of acton
Non-nucleoside reverse transcriptase inhibitors (NNRTIs) • Delavirdine • Efavirenz • Nevirapine	The newest class of antiretroviral agents, NNRTIs stop HIV production by binding directly onto reverse transcriptase and preventing the conversion of RNA to DNA. These drugs are called 'non-nucleoside' inhibitors because even though they work at the same stage as nucleoside analogues, they act in a completely different way.
Nucleoside analogue reverse transcriptase inhibitors • Didanosine (ddI) • Lamivudine (3TC) • Stavudine (d4T) • Zalcitabine (ddC) • Zidovudine (ZDV or AZT) • Abacavir	The first effective class of antiretroviral drugs was the nucleoside analogues. They act by incorporating themselves into the DNA of the virus, thereby stopping the building process. The resulting DNA is incomplete and cannot create a new virus.
Protease inhibitors • Amprenavir • Indinavir • Nelfinavir • Ritonavir • Saquinavir	Protease inhibitors work at the last stage of the virus reproduction cycle. They prevent HIV from being successfully assembled and released from the infected CD4+ cell.

Source: adapted from educational material produced by Boehringer Ingelheim International GmbH.

provides ongoing education for HIV prescribers throughout NSW. It is through this project that data collection forms for the study have been provided to all doctors who are HIV prescribers in NSW. Hospital emergency departments, sexual health clinics and sexual assault clinics have also been provided with enrolment packs in most Area Health Services via the HIV-Sexual Health Coordinators. Patients who consent have been enrolled in the study when they presented for PEP and are followed up for six months using questionnaires to their doctors.

Enrolments to the study commenced in December 1998. The data collected by the doctor includes demographic information (age, sex, postcode), baseline HIV status and details of the exposure involved. The doctor also collects information about the source person from the PEP recipient. Depending on the recipient's knowledge of the source, this could include the source's sex, HIV exposure category, HIV status and treatment with antiretroviral therapy. All individuals who present and are eligible to be prescribed PEP are also eligible to be enrolled on the study, including those who elect not to take PEP. For those who are prescribed PEP, details of the drug treatment used is sought. Follow-up is conducted after four weeks to assess adherence to treatment and side effects, as well as HIV status. Results of all HIV testing are obtained at the final follow-up after six months. Summary statistics of the data collected have been generated.

RESULTS

During the period December 1998 to February 2000, 88 participants have been enrolled in the study. The monthly number of prescriptions for non-occupational PEP tended to increase over the first year (Figure 1). The exposure leading to presentation for PEP was male homosexual contact in 70 per cent, heterosexual contact in 10 per cent, percutaneous in 17 per cent, and other exposures in three per cent. Over 25 per cent of percutaneous exposures resulted from assaults with a used syringe, a further 25 per cent was due to community acquired needle stick injuries, and the remaining 50 per cent being related to re-use of injecting equipment. The median time between exposure and presentation for PEP was 30 hours (range 1 to 171). The majority of PEP prescriptions (73 per cent) have been for triple-combination therapy. Nearly all prescriptions have been for twice-daily dosing regimens. The source person was known to be HIV positive in 47 per cent overall, although among

homosexual exposures this was slightly higher (53 per cent) and lower among percutaneous exposures (22 per cent).

The majority (72 per cent) of participants adhered to and completed the four week course of treatment. Over 75 per cent experienced side-effects which were mostly reported as mild, although there were five cases where side-effects were the reason for discontinuing treatment. Four week follow-up has been completed for 61 subjects. There have been no HIV seroconversions, and four participants have been lost to follow-up. Six month follow-up has been completed for 28 subjects, with no seroconversions, and a further four who have been lost to follow-up.

DISCUSSION

The majority of exposures that have resulted in prescription for PEP have fallen within the NSW Department of Health guidelines. The distribution of risk behaviours among participants is broadly similar to that in people with HIV infection in Australia (NCHECR, 1999), with most being related to male homosexual contact. However, there is a higher representation of percutaneous and 'other' exposures in the study which reflects a greater proportion of cases of assault or accidental injury with a used needle. The NSW Department of Health Guidelines do not recommend offering PEP for needle-stick injuries that occur from discarded injecting equipment in the community, as these cases are usually low risk exposures. This is because injecting drug users have a relatively low risk of HIV infection in Australia, and because the exposure is to a small volume of blood. In addition, even in cases where the source was HIV positive, the blood is likely to have very low viral load related to viral decay outside the human body.[10]

Despite the guidelines recommending that two drugs are sufficient for most exposures, nearly three-quarters of the prescriptions for PEP in the study have been for three antiretroviral drugs.

A limitation of this study is that not all people who have been eligible for inclusion might be enrolled on the study. We confirmed that we had enrolled almost 100 per cent of eligible patients at one large public hospital and one large private practice. At other sites, it was not possible to determine participation rates. However, the study has been actively promoted to all prescribing general practitioners as well as in all Area Health Services in NSW.

The results of this study of the first year of non-occupational PEP in NSW raises a number of operational issues, which need to be considered in policy formulation and health service delivery. These include the following:

- How can the prescription of PEP for community acquired accidental needle-stick injury be discouraged given that the likelihood of transmission through discarded injecting equipment is very small?
- Why are doctors and patients exhibiting a preference for three over two drugs?
- Given the evidence that the efficacy of PEP declines with increasing time since exposure, can the time between exposure and prescription be further reduced by the implementation of urgent triaging procedures in public and private settings?
- Should the availability of PEP be promoted among individuals at particular risk? For instance, given that approximately half of the sexual exposures in the

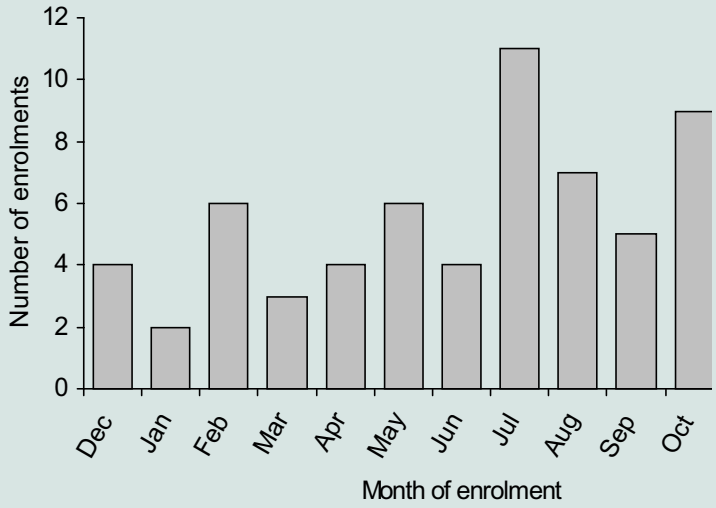
TABLE 2

ANTIRETROVIRAL DRUGS USED FOR HIV POST-EXPOSURE PROPHYLAXIS IN NSW

Antiretroviral combinations	Number of prescriptions
zidovudine–lamivudine–nelfinavir	28
zidovudine–lamivudine	22
zidovudine–lamivudine–nevirapine	8
zidovudine–lamivudine–indinavir	7
stavudine–didanosine–nelfinavir	6
stavudine–lamivudine–nelfinavir	5
other combinations	8
not prescribed PEP	4
Total	88

FIGURE 1

ENROLMENTS TO STUDY OF NON-OCCUPATIONAL PEP IN NEW SOUTH WALES: DECEMBER 1998 TO DECEMBER 1999



study occurred where the source was known to be HIV positive, should serodiscordant couples be targeted in promoting PEP?

- How can appropriate risk reduction counselling be provided in public and private settings?

Addressing these issues will help ensure the most appropriate and effective use of PEP and assure that PEP is properly positioned as the prevention mechanism of last resort within a broader scheme of HIV prevention.

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THE EPIDEMIOLOGY OF RESPIRATORY SYNCYTIAL VIRUS INFECTIONS IN NSW CHILDREN, 1992–1997

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This paper evaluates the data describing respiratory syncytial virus (RSV) infection in infants and children aged less than five years in NSW, and in particular children aged less than one year. This younger age group forms the primary target group for immunisation against RSV and vaccine development is now well advanced. The data outlined in this paper provide a baseline for evaluating the burden of disease before and after the introduction of a vaccine.

BACKGROUND

RSV is the most important cause of viral lower respiratory tract disease in infants and children throughout the world.[1] RSV is estimated to cause up to 80 per cent of hospital admissions for bronchiolitis in infants under one year of age and is characterised by wheezing and hypoxia. RSV is also associated with pneumonia, croup, bronchitis, otitis media and upper respiratory tract infections.[2,3,4] Acute bronchiolitis and bronchitis are the sixth most common causes of hospital admissions in Australian children.[5]

Overseas literature suggests that infants aged 2–6 months are most severely affected by RSV infection, and mortality rates are higher among those with underlying respiratory and cardiac conditions.[2,6] Despite the considerable effect on public health of this disease, there are very little recent epidemiological data available in Australia. Treatment options remain limited but vaccine development is proceeding and clinical trials have commenced.[7]

METHODS

As RSV infection is not a notifiable disease, a number of sources of data were used to build a picture of the age distribution, seasonality and incidence of RSV infection in NSW. Three data sources, each identifying the most severe outcomes of RSV disease (that is, cases resulting in hospitalisation) were used.

The Virology and Serology Laboratory Reporting Scheme (LabVISE)

This is a national sentinel surveillance database reporting a range of virologic and serologic identifications and is co-ordinated by the National Centre for Disease Control.[8] The scheme comprises sentinel laboratories across Australia. However the laboratories included can vary over time, and not all hospitals submit diagnostic specimens to the collection. Data items analysed in our study for RSV were collection date, laboratory code, age, sex and postcode of residence.

NSW Inpatient Statistics Collection

This database provides information on all hospital admissions in private and public hospitals in NSW. Data were accessed via the Public Health Division's HOIST data warehouse. It includes the principal diagnosis responsible for the hospital admission, which is classified as an ICD-9 code.[9] As there was no specific ICD-9 code for RSV infection during the study period, alternative codes were investigated, which we understood would cover most RSV infections in young children. Consequently, patient records with ICD-9 codes 466.1 ('acute bronchiolitis') and 079.89 ('other specified viral infections') as the principal diagnosis for admission (mutually exclusive) were extracted.[9] Records for young children (aged less than one year) coded under 079.89 were called 'presumed RSV' in this study. Data for acute bronchiolitis were available for analysis from 1990 to 1995, and for 'presumed RSV' from June 1994 to December 1995. Data items analysed were age, sex, hospital admission date and Area Health Service of residence.

Australian Bureau of Statistics (ABS) Mortality Data

This collection provides information on all deaths in Australia as collected from registration of deaths provided by the Registrars of Births, Deaths and Marriages in each State and Territory. Data were accessed via the Public Health Division's HOIST data warehouse. The underlying cause of death is classified according to ICD-9 codes and deaths due to acute bronchiolitis (ICD-9 code 466.1) from 1992 to 1996 in NSW were analysed. Data items were age and year of death.

RESULTS

Laboratory Reports

Between January 1993 and December 1997 a total of 4,665 cases of RSV infection were reported to LabVISE for all ages where either postcode of residence or the notifying laboratory was in NSW. Between 770 and 1,131 cases were reported annually, with numbers peaking in 1997. Of all cases, 98.5 per cent were for children aged less than five years, 78 per cent for children less than one year of age, 53 per cent for children less than six months of age and 29 per cent for children less than three months of age.

There were more reports of RSV infection in males (male:female ratio = 1.4:1) for both all ages and children aged less than one year. A distinct seasonal pattern was found for the period 1993 to 1997, with

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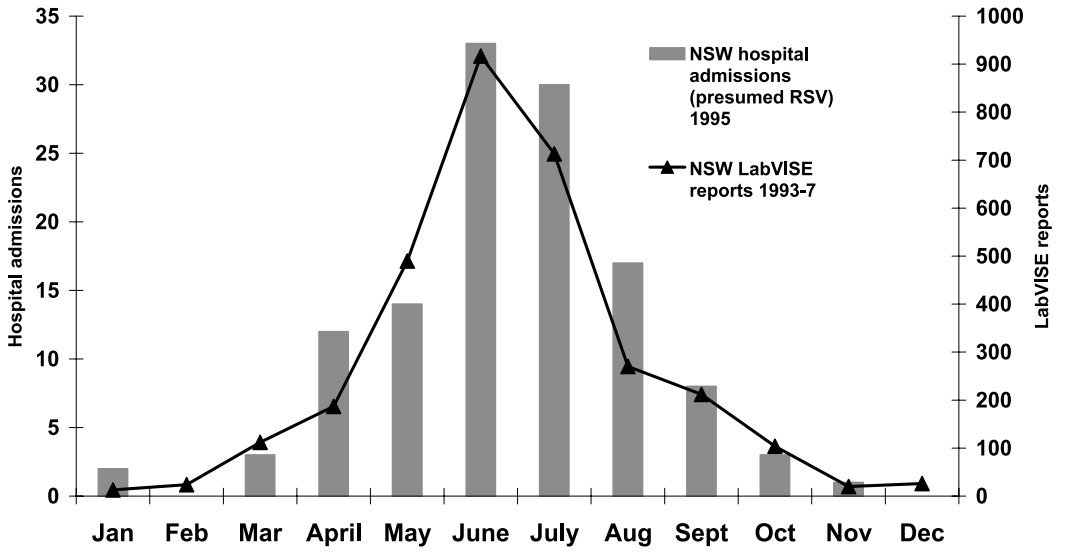
FIGURE 2

COMPARISON OF HOSPITAL ADMISSIONS FOR 'PRESUMED RSV' INFECTION AND LABVISE REPORTS BY MONTH FOR CHILDREN AGED LESS THAN ONE YEAR, NSW



reports increasing in May and peaking in June each year with little annual variability. Most cases (84 per cent) in children aged less than one year of age were reported between May and September (see Figure 2).

NSW Inpatient Statistics Collection (ISC)

Between 1990 and 1995 there were 22,969 admissions to hospital in NSW for all ages with acute bronchiolitis as the principal diagnosis. Of all cases, most (86 per cent) were children less than one year of age and a further 12 per cent were aged between 1–5 years. There were 375 admissions for other specified viral infections between June 1994 and December 1995. Of these, 66 per cent were children aged less than one year and a further 17 per cent were aged between 1–5 years.

Three-quarters (75.5 per cent) of admissions for acute bronchiolitis, and 78 per cent of those for 'presumed RSV' infection, were children aged six months or less. Admissions for acute bronchiolitis and 'presumed RSV' infection were similar in pattern to the LabVISE reports and peaked at between one and two months of age (see Figures 3 and 4 respectively).

A seasonal pattern was found, with a peak in June for 'presumed RSV' infection, and July for acute bronchiolitis. Both are compared with LabVISE reports in Figures 2 and 5.

Age-specific rates for acute bronchiolitis showed an overall increase in the six-year period from 1990 to 1995 with some variability on a year-to-year basis. Comparable data for 'presumed RSV' infection over this time frame were not available for analysis.

The annual incidence of hospitalisation for children less than one year of age for both conditions was significantly higher in rural than metropolitan (all health regions in Sydney, the Illawarra and Hunter districts) NSW. Rates for acute bronchiolitis in children less than one year of age were 56 per 1,000 population for rural NSW compared to 41 per 1,000 for metropolitan NSW ($P<0.0001$). For 'presumed RSV' infection the rates were three per 1,000 population for rural NSW compared to 1.5 per 1000 for metropolitan NSW ($p<0.0001$). Rates of hospital admission for all causes in this age group in 1995 were marginally higher in rural infants (1,372 per 1000) than metropolitan (1,340 per 1,000).

Mortality

From 1992 through 1996, seven children aged less than one year from NSW were reported to the ABS Cause of Death register with a diagnosis of acute bronchiolitis. Five of these deaths were in infants aged three months or less. The other two deaths were children aged between one and two years. One or two deaths occurred each year from 1992 to 1996, with boys more likely to die of acute bronchiolitis (male:female ratio = 1.5:1) in children aged one year or less. Children in this age group represented 56 per cent of deaths for all ages due to acute bronchiolitis in NSW.

DISCUSSION

Large numbers of children are admitted each winter to NSW hospitals with acute bronchiolitis, most of which is caused by RSV infection. Hospital data on RSV infection have been incomplete until recently. However, in July 1998 an ICD-10 code for RSV infection was introduced, so specificity of diagnoses should improve, subject to coding practices. LabVISE reports for RSV infection are likely to represent hospitalised cases, as laboratory tests

FIGURE 3

HOSPITAL ADMISSIONS FOR ACUTE BRONCHIOLITIS AND LABVISE (RSV) REPORTS IN CHILDREN AGED LESS THAN ONE YEAR, NSW

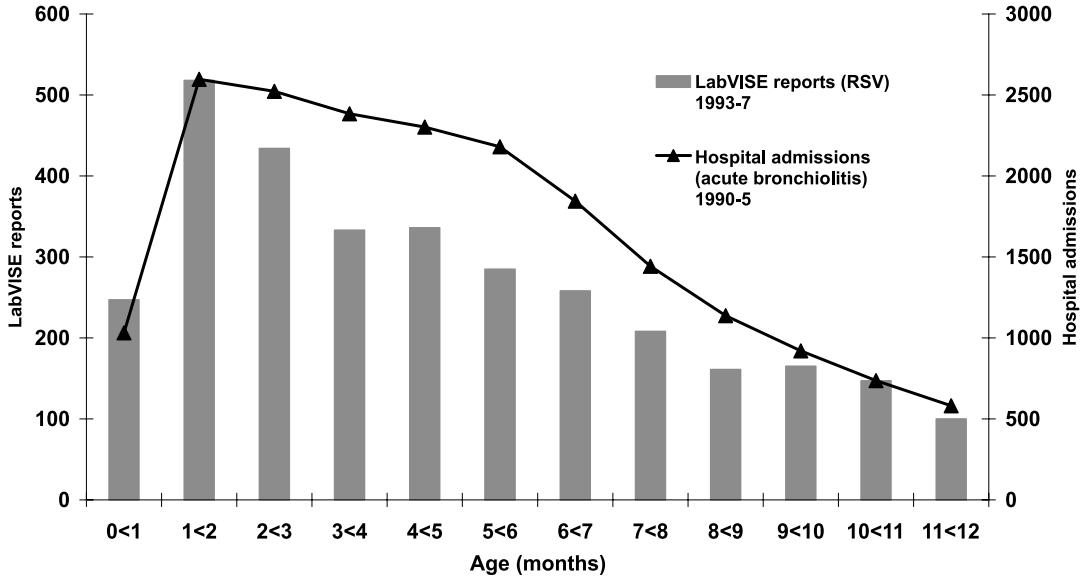


FIGURE 4

HOSPITAL ADMISSIONS FOR 'PRESUMED RSV' INFECTION AND LABVISE (RSV) REPORTS IN CHILDREN AGED LESS THAN ONE YEAR, NSW

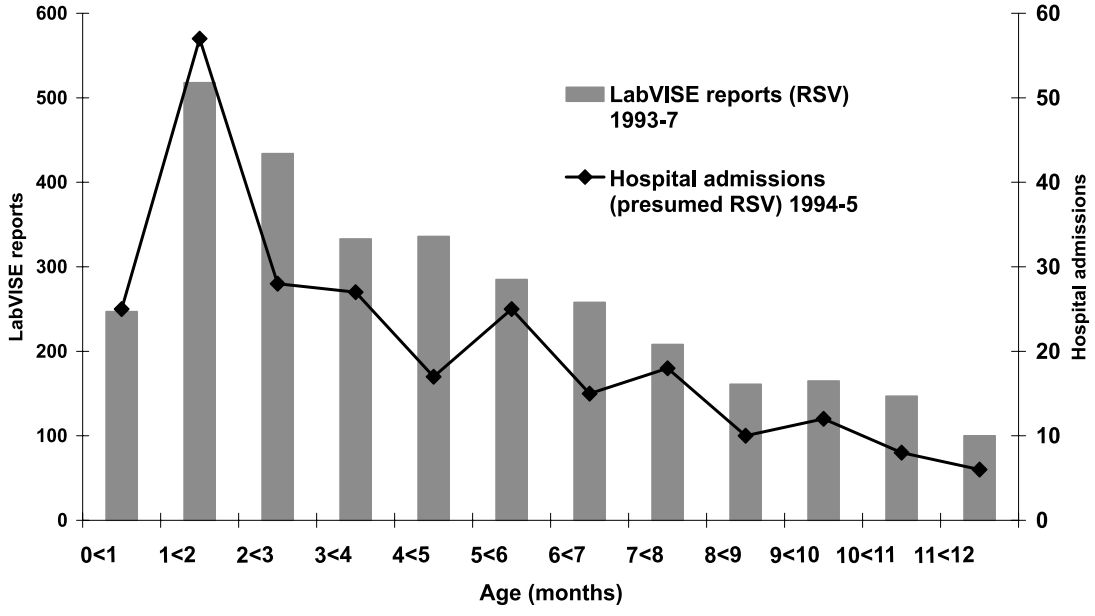
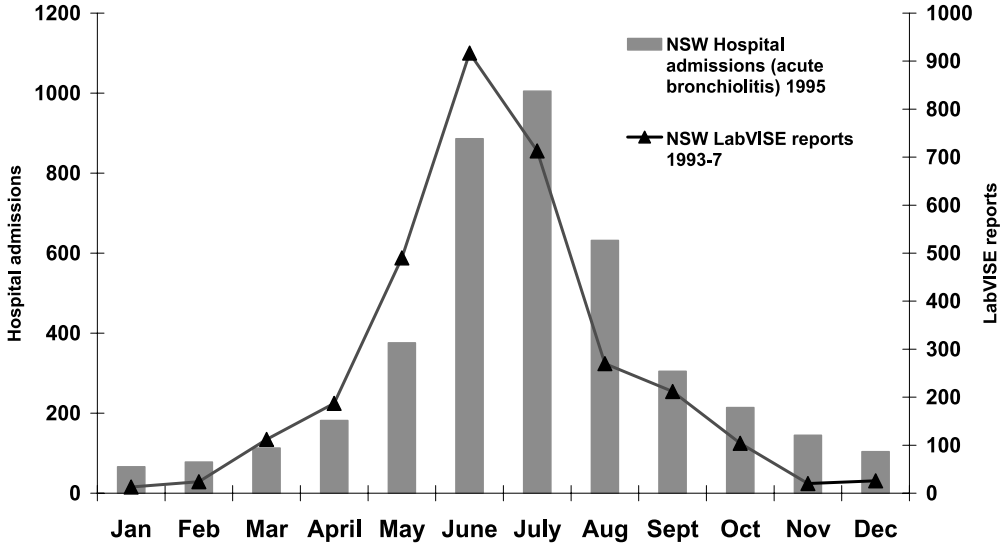


FIGURE 5

COMPARISON OF HOSPITAL ADMISSIONS FOR ACUTE BRONCHIOLITIS WITH LABVISE (RSV) REPORTS BY MONTH OF YEAR FOR CHILDREN AGED LESS THAN ONE YEAR, NSW



are almost exclusively done in these cases, particularly for young children. The data reported here thus reflect the pattern of hospitalised cases rather than RSV infection in the community.[10]

The increased reports of RSV from 1993–97 in LabVISE are likely to be due to increases both in testing and in the number of participating laboratories rather than a real increase in overall incidence of disease.[10] Hospital admission data also show a marked increase in 1995, reflecting year-to-year variation in RSV activity in NSW.

Admissions to hospital for 'presumed RSV' infection show a distinct seasonal pattern very similar to the pattern of reports from LabVISE, with the peak for admissions in June and most occurring between May and September (see Figures 2 and 5). Hospital admissions by age for both ICD-9 codes also show a very similar pattern to the LabVISE reports (see Figures 3 and 4). This close correspondence increases confidence that the two data sets are capturing similar populations. The analyses undertaken in this study suggest a slightly closer correspondence between 'presumed RSV' infection and LabVISE than for acute bronchiolitis, indicating that ICD-9 code 079.89 may be more specific for RSV infection in children aged less than one year.

Our study shows that children in rural areas have higher admission rates for acute bronchiolitis and RSV infection than urban children, but as rates of admission in young children for all causes are marginally higher in rural NSW, differences in medical and coding practices could partially explain this.

Hospitalisation in NSW occurred mainly in infants aged six months or less, particularly those aged one to two months. This is consistent with overseas and interstate findings.[1, 6] Overall, NSW hospitalisation rates for acute bronchiolitis in children less than one year of age are higher than those reported from Western Australia,[11] the USA, [12] and Denmark, [13] but these differences could be explained by annual variation in RSV occurrence and hospitalisation practices.

Mortality due to acute bronchiolitis shows a similar pattern to hospital admission and LabVISE data, with infants aged three months or less most severely affected. Again, these data are consistent with findings from overseas.[2,6]

Worldwide, RSV infection is a major cause of morbidity and an important priority for vaccine development. With up to two-thirds of infants infected with RSV by one year of age of whom 2.5 per cent are hospitalised, prevention of severe disease-causing hospitalisation in this age group is the primary objective of childhood immunisation.[1, 7] In NSW, prevention of RSV-related hospitalisation would result in a significant decrease in the absolute number of hospitalised infants, with potentially large cost savings. These data provide a baseline for assessment of the effect of RSV in NSW and for following annual trends to evaluate the burden of disease before and after the introduction of vaccine to prevent RSV infection.

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NSW PUBLIC HEALTH BULLETIN

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

Submission of articles

Articles, news and comments should be 1000 words or less in length and include a summary of the key points to be made in the first paragraph. References should be set out in the Vancouver style, described in the *New England Journal of Medicine*, 1997; 336: 309–315. Send submitted articles on paper and in electronic form, either on disc (Word for Windows is preferred), or by email. The article must be accompanied by a letter signed by all authors. Full instructions for authors are available on request from the editor.

Editorial correspondence

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Back issues can be obtained from the Public Health Training and Development Unit, Locked Mail Bag 961, North Sydney NSW 2059.

A CAPACITY BUILDING WEB SITE

The Health Promotion Branch, NSW Department of Health, has developed an interactive Capacity Building Web site to provide information and improve the dissemination of capacity building research and tools. The site is located on the NSW Health Web site at **www.health.nsw.gov.au/public-health/health-promotion**.

Key items on the site include:

- key questions answered
- capacity building colloquium
- grants scheme
- framework of strategies
- indicators
- references
- resources and contacts.

Also available on the Web site is information describing the *Capacity Building: Mastering the Art of the Invisible* colloquium. The colloquium, held at Sydney University on the 6th of March this year, brought together over 100 researchers, practitioners and policy makers from across Australia. The key presenters included Professor Stephen Leeder, Dr Penny Hawe, Dr Robert Bush, Associate Professor Hal Swerissen, and Robert Fitzgerald, NSW Community Services Commissioner.

The major outcomes from the day were:

- a shared understanding of the various ways of thinking about capacity building
- a clear direction for further work in capacity building.

Transcriptions from the presentations, discussions and questions to the panel are all available on the Web site.

For further information about the Capacity Building West site contact Shelley Bowen, Health Promotion Strategies & Settings Unit, NSW Department of Health, by telephone on 9391.9540, or by email at sbowe@doh.health.nsw.gov.au.

A DRINKING WATER INVESTIGATION

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Hunter Public Health Unit

The aim of this investigation was to ascertain whether untreated water drawn from rivers, springs and rainwater tanks, and which is intended for drinking purposes in rural accommodation establishments (such as caravan parks, wilderness resorts, 'country retreat' and bed and breakfast style accommodation), complied with the *Australian Drinking Water Guidelines*. For a description of the guidelines and how they were applied to this investigation see page 126. The investigation revealed that the majority of premises did not have water treatment systems installed, and where they were installed they were inadequately maintained. At the time of the investigation there was no legislation through which such premises could be directed to treat drinking water. This article describes the potential public health implications of guests consuming untreated water from these establishments.

BACKGROUND

In November 1996, the Hunter Public Health Unit began a program to sample the drinking water provided by a number of rural accommodation establishments in the Williams, Allyn and Chichester River Catchments. The program followed a reported outbreak of diarrhoea and vomiting among guests of a rural accommodation establishment, where one guest reportedly attended hospital. Following this notification, the premises were inspected by Food Surveillance Officers and Environmental Health Officers from the Hunter Public Health Unit. Food hygiene practices at the establishment were reported by the Food Surveillance Officers as being of a poor standard. However, due to previous problems

TABLE 3

LEVELS OF FAECAL COLIFORMS, *E. COLI* AND TOTAL COLIFORMS IN WATER SAMPLES: WILLIAMS, ALLYN AND CHICHESTER RIVER CATCHMENTS, NOVEMBER 1996

Site	Location	Faecal coliforms	*E. coli*	Total coliforms	Water treated
1a	External Tap	<1	<1	<2	yes
1b	Kitchen Tap	<1	<1	<2	yes
1c	Draw off	120	120	150	no
2a	Kitchen Tap	<1	<1	<2	no
2b	Draw off	94	94	460	no
3a	Restaurant	31	16	62	no
3b	Guest Lodge	<1	<1	<2	no
3c	Staff tap	2	2	4	no
4a	External Tap	20	20	47	no
4b	Draw off	140	140	540	no
5a	Lodge Tap	<1	<1	<2	no
5b	Staff tap	2	2	10	no
6a	External tap	<1	<1	<2	partially
7a	Kitchen Tap	<1	<1	<2	partially
8a	Staff tap	8	8	120	no

NOTE: Australian Drinking Water Guidelines for faecal coliforms, *E. coli* and total coliforms are zero per 100mL sample.

TABLE 4

LEVELS OF FAECAL COLIFORMS, *E. COLI* AND TOTAL COLIFORMS IN WATER SAMPLES: WILLIAMS, ALLYN AND CHICHESTER RIVER CATCHMENTS, DECEMBER 1996 (FOLLOW-UP STUDY)

Site	Location	Faecal coliforms	*E. coli*	Total coliforms	Water treated
la	Kitchen Tap	<1	<1	<1	yes
lb	Draw off	58	58	800	no
2a	Kitchen Tap	<1	<1	<1	no
2b	Draw off	15	15	1200	no
3a	Restaurant	1	1	290	no
3b	Guest Lodge	<1	<1	30	no
3c	Owners res.	1	1	340	no
4a	Draw off	120	120	430	no
5a	Lodge Tap	2	2	2	no
5b	Owners res.	11	*5*	16	no
6a	Outside Tap	<1	<1	<1	partially
7a	Kitchen Tap	<1	<1	10	partially
8a	Owners res.	<1	<1	38	no

with drinking water in this particular catchment area, it was considered important that water samples were also taken.

Examination of the water sample from the initial investigation indicated that the river water, water storage tanks and reticulated supply to the kitchen and guest rooms all failed to meet the standards set out by the *Australian Drinking Water Guidelines*. This untreated water was used for food preparation in the main kitchen, to make water-based drinks, and was reticulated to all guest rooms. Fifty-four questionnaires were issued to guests of the accommodation establishment who were present at the time of the outbreak. Forty questionnaires were returned, with twenty-two respondents reporting diarrhoea and vomiting. The questionnaire responses indicated that the highest attack rate (91 per cent) was among guests who consumed water or ice. No faecal specimens were obtained because the guests were dispersed over long distances and there was delay in notification.

METHODS

On the 4th and 5th of November 1996, 15 water samples were collected from eight separate rural accommodation establishments, and sent to the Division of Analytical Laboratories (DAL) in Lidcombe to be analysed for the presence of faecal coliforms, total coliforms and *Escherichia coli* (*E. coli*). The samples were stored under refrigeration until they were transferred to DAL in an insulated container. All samples from taps were taken using a standard procedure to ensure that they were not contaminated by bacteria living in or around the taps. This procedure involved heating the taps under a naked flame for about 30 seconds and allowing water to run through taps for 15 seconds so that a representative sample of water flowing through the system was obtained, rather than sampling water that was stored in or close to the tap.

Water from the Williams, Allyn and Chichester catchments was included in the study and samples were taken from raw water (untreated) draw off, domestic and drinking sources at the accommodation establishment (Table 3). The sources of the water samples were: one from a natural spring, two were from the Allyn River, two from the Chichester Dam, five from rainwater tanks and five from the Williams River. Two samples were taken from a source at the main water supply from Chichester Dam, which received only partial treatment (low-level chlorination, not filtered). Two samples were taken from a source which treated the water by filtration, chlorination and ultra-violet disinfection.

RESULTS

All four water samples taken from treated supply sources met the bacteriological standards for drinking water as recommended by the *Australian Drinking Water Guidelines*. Of the remaining 11 samples taken from untreated supplies, only three samples met the bacteriological standard of the guidelines. Therefore, 73 per cent of the raw water supplies failed to meet the bacteriological standards for drinking water.

FOLLOW-UP STUDY

In December 1996, a follow-up round of 13 samples was taken. Again four samples taken from treated water supplies met the bacteriological standard recommended by the *Australian Drinking Water Guidelines*. Of the remaining nine samples, seven failed to meet the bacteriological standard for drinking water. This represents a 78 per cent failure rate for raw (untreated) water (Table 4).

DISCUSSION

The *Australian Drinking Water Guidelines* state that where drinking water supplies are not protected and effectively treated, outbreaks of infectious disease, particularly diseases of the intestine, may occur. This potential public health risk has been supported by this investigation, with a high percentage of samples from untreated water supplies not meeting the bacter-iological criteria for drinking water.

Notifications to the Hunter Public Health Unit over the past five years indicate that tourists living in this type of accommodation regularly report gastrointestinal illnesses. It is likely that the cause of these illnesses relates to drinking untreated water. While rural accommodation establishments continue to provide untreated drinking water, the health of guests will be at risk.

Since the conclusion of this investigation the Public Health Act, 1991 has been amended. While at the time of the investigation general powers existed that allowed for the closure of a water supply, the recent amendment strengthens the powers of the Chief Health Officer in relation to drinking water. In addition to this all councils in NSW have been requested to supply details of commercial premises that have an untreated drinking water supply.

ACKNOWLEDGEMENT

The author would like to acknowledge the assistance in the investigation of Christopher David Williams, Environmental Health Officer, Hunter Public Health Unit.

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NATIONAL WATER QUALITY GUIDELINES AND HOW THEY WERE APPLIED TO THIS INVESTIGATION

The *Australian Drinking Water Guidelines* describe drinking water as 'water intended primarily for human consumption which also has other domestic uses'. It may be consumed directly from the tap, or indirectly in beverages or foods prepared with water. Drinking water should be safe to use and aesthetically pleasing, clear, colourless, well aerated, with no unpalatable taste or odour and should contain no suspended matter, harmful chemical substances or pathogenic organisms. Appearance and taste are usually the characteristics by which the public judges water quality; however water which is cloudy or coloured or has an objectionable taste may not be unsafe to drink. The safety of water in public health terms is determined by its microbiological, physical, chemical and radiological quality. Of these parameters, the *Australian Drinking Water Guidelines* state that microbiological quality is usually the most important.[1]

The guidelines state that no drinking water sample should contain faecal coliforms or *Escherichia coli* (*E. coli*). Performance is satisfactory if over a 12 month period at least 98 per cent of scheduled samples contain no thermotolerant coliforms, and at least 95 per cent of samples contain no coliforms. A higher level of contamination may be tolerated in a particular area under certain conditions. These conditions are:

- the water system meets the guideline value for thermotolerant coliforms
- the water authority can satisfy the appropriate health authority that the coliforms are unlikely to be of faecal origin
- there is a level of monitoring sufficient to detect any change in the pattern of coliform occurrence
- there is direct monitoring of the occurrence of pathogenic micro-organisms as the health authority may select to ensure the coliform level does not represent a risk to public health
- agreed levels of service for total coliforms are negotiated with the appropriate authority and the consumers.[1]

These conditions apply to the quality of water at the point of use (for example, kitchen tap or shower) and apply to reticulated water at the consumers' tap, at a rainwater tank tap, and to source water if water is to be used without prior treatment. It should be emphasised that these conditions define water which, based on current knowledge, is safe to drink over a lifetime and therefore constitutes no significant risk to public health.[1]

These conditions are the minimum requirement for drinking water from a public health viewpoint. As the microbiological quality of water is considered to be the most important factor in determining the safety of water supplies, the chemical and radiological quality of the water was not analysed in this investigation.

In a situation where a small supply is serving an isolated establishment such as the accommodation establishments sampled in this study, absolute implementation of these requirements may be unrealistic. For these situations, it is recommended that as a minimum, microbiological characteristics should be monitored. However, if this is not possible, the public should be advised to boil water before it is consumed, or to use bottled water.

NSW HEALTH ESTABLISHES THE CENTRE FOR PUBLIC HEALTH NUTRITION

Jane Moxon, Edwina Macoun and Philip Vita
Health Promotion Branch
NSW Department of Health

The establishment of the Centre for Public Health Nutrition represents a major new investment in public health nutrition by the NSW Department of Health.

The Centre for Public Health Nutrition aims to:

- build capacity to monitor the food and nutrition situation in NSW
- assist workforce development
- support best practice in public health nutrition.

WHY ESTABLISH A CENTRE FOR PUBLIC HEALTH NUTRITION IN NSW?

Improved nutrition is second only to tobacco use as the most important preventable health measure. Nutrition is a key element of chronic disease prevention as diet-related disease costs Australia at least $2.5 billion per year in health-care costs and lost earnings.

Nutrition is a highly complex issue and is determined by individual choice, the social environment, and the food supply. If the nutritional status of NSW residents is to be improved, action is required by the health system at several levels. This includes:

- continuing to improve the research base for linking dietary factors with health outcomes;
- providing evidence-based public health nutrition programs to influence food choice;
- ensuring the quality and safety of the food supply;
- providing an adequate food supply at reasonable cost to consumers;
- ensuring access to and availability of appropriate foods, particularly to those nutritionally vulnerable groups.

Effective change in the nutritional health of populations requires a range of action including:

- the development of comprehensive programs of action that use a variety of methods and approaches;
- policy development;
- development of best practice guidelines;
- workforce development within the field of nutrition;
- updating current legislation;
- effective communication strategies and community development.

In establishing the Centre for Public Health Nutrition, NSW Health recognised the need to increase its capacity for planning, developing and monitoring public health nutrition initiatives. A systematic planning process involving the development of the State nutrition monitoring report and consultations with key stakeholders highlighted this need.

THE GOAL OF THE CENTRE

The goal of the Centre is to enable NSW Health to establish and maintain a State-level food and nutrition monitoring system that supports workers needs for information, and complements national monitoring efforts. The Centre will also assist with strategy development and quality improvement support in delivering public health nutrition programs within the NSW health system. The Centre will:

- improve access for decision-makers to quality information available about the food and nutrition situation in NSW, relevant to statewide and Area Health Service priorities;
- provide an integrated strategic approach to nutrition at State and Area health service levels that is coordinated with, and complementary to, national policies and strategies;
- support public health workforce development in NSW to improve the use of information for decision-making, policy formulation and practice relating to public health nutrition;
- identify priorities for applied research and program evaluation that will contribute to 'best practice' in community–public health nutrition.

These outcomes are expected to lead to effective, sustainable public health nutrition interventions at both local and State levels, in addition to raising the profile of public health nutrition among health service leaders in NSW.

A CENTRE OF EXCELLENCE

The Nutrition Research Foundation of the University of Sydney won the tender to establish and manage the Centre. The Centre, to be located at the University of Sydney, will be co-directed by Dr Karen Webb and Professor Ian Caterson. A team of experts with skills in nutrition epidemiology and monitoring, program planning and evaluation, and management and analysis of dietary data will staff the Centre.

The Centre will enable excellence in public health nutrition planning, strategy development and monitoring in NSW.

For more information about the NSW Centre for Public Health Nutrition contact Philip Vita, Acting Manager Sun Exposure, Nutrition and Physical Activity Policy Unit, NSW Department of Health, by telephone 9391 9661, or by email on pvita@health.nsw.gov.au.

MENINGOCOCCAL DISEASE IN NSW 1991–1999

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Meningococcal disease is characterised by a rapid onset and, on rare occasions, death. Because these deaths are often sudden and usually affect young people they are often publicised and cause alarm within the community. Here we review the epidemiology of meningococcal disease cases notified in NSW since 1991.

BACKGROUND

Meningococcal disease is caused by infection with *Neisseria meningitidis* bacteria. The bacteria has several different serogroups. The identification of serogroups can help distinguish related cases and describe the epidemiology of the disease.

Around five to 10 per cent of people in some communities may carry the bacteria in their nasopharynx, but disease is rare. When it does occur the illness usually progresses rapidly. The symptoms vary according to main site of infection, and may include:

- high fever
- headache
- nausea and vomiting
- neck stiffness
- drowsiness
- coma
- a characteristic rash.

Specific treatment involves intravenous antibiotics and supportive care. Those at increased risk of disease include:

- persons in close contact with a case
- persons without a functioning spleen
- persons with rare congenital deficiencies of complement or properdin
- travellers to regions where the disease is endemic (for example, sub-Saharan Africa)
- small children
- adolescents.[1]

Cases of meningococcal disease should be notified to public health units (PHUs), whose staff investigate risk factors and provide education and chemoprophylactic therapy to close contacts.

METHODS

Under the NSW Public Health Act 1991, all laboratories and hospitals must notify suspected cases of meningococcal disease to their local PHU. PHU staff record the details on a confidential statewide database.

The characteristics of cases of meningococcal disease notified to PHUs between 1991 and 1999 were analysed. Incidence rates were calculated using the estimated 1997 mid-year population for NSW.

The NSW Department of Health's Inpatients Statistics Collection (ISC) was used to identify hospital separations of NSW residents with an ICD-9 principal diagnosis code of 036, meningococcal disease. Data were only available for complete calender years to 1997. The total number of cases for 1991, 1992 and half of 1993 were estimates based on a weighted sample as a census of all hospital separations was included in the ISC only after June 1993. To better estimate case counts of admitted patients, obvious

TABLE 5

PATIENT NOTIFICATION 1991–1999, HOSPITALISATION 1991–1997, AND DEATHS FROM MENINGOCOCCAL DISEASE, NSW

Case characteristics	*Notified cases*	*Hospital admissions*[1]	*Notified deaths (% of cases)*
Year of onset			
1991	130	118	3 (2)
1992	122	113	7 (6)
1993	153	127	11 (7)
1994	142	132	15 (11)
1995	113	104	6 (5)
1996	161	149	7 (4)
1997	219	218	7 (3)
1998	184	Not available	16 (9)
1999	220	Not available	14 (6)

1. (excludes multiple admissions)

TABLE 6

CHARACTERISTICS OF PATIENTS NOTIFIED WITH MENINGOCOCAL DISEASE, NSW, 1991–1999

Case characteristics	Cases (% total)	Average annual rate per 100,000	Deaths (% of cases)
Residence			
Sydney area	754 (52)	2.3	50 (7)
Other NSW	668 (46)	2.8	35 (5)
Overseas/unknown	22 (2)	-	1 (5)
Sex			
Male	775 (54)	2.8	53 (7)
Female	668 (46)	2.6	33 (5)
Age group			
<1	254 (18)	32.5	17 (7)
1	187 (13)	23.9	8 (4)
2	90 (6)	11.4	4 (4)
3	56 (4)	7.1	3 (5)
4	55 (4)	6.9	2 (4)
Total <5	***642 (44)***	***16.2***	***34 (5)***
5–9	103 (7)	2.6	8 (8)
10–14	87 (6)	2.2	0 (0)
15–19	224 (16)	6.0	14 (6)
20–24	106 (7)	2.5	4 (4)
25–44	136 (9)	0.8	9 (7)
45–64	79 (5)	0.7	10 (13)
65+	67 (5)	0.9	7 (10)
Syndrome			
Meningitis	769 (53)	1.4	25 (3)
Septicaemia	396 (27)	0.7	49 (12)
Unspecified	279 (19)	0.5	12 (4)
Laboratory confirmed	1011 (70)	1.8	67 (7)
Total	**1444 (100)**	**2.6**	**86 (6)**

multiple re-admissions of the same case (based on age, sex, place of residence and admission–separation dates) were removed.

RESULTS

Case reports

During the nine-year period, 1444 cases of meningococcal disease were reported in NSW, an average of 160 per year. The least number of reports of the disease were received in 1995 (*n*=113), and the most in 1999 (*n*=220) (see Table 5). The average annual incidence for this period was 2.6 per 100,000 persons. The incidence of meningococcal disease was clearly seasonal, with prominent peaks occurring in late winter and early spring (see Figure 6).

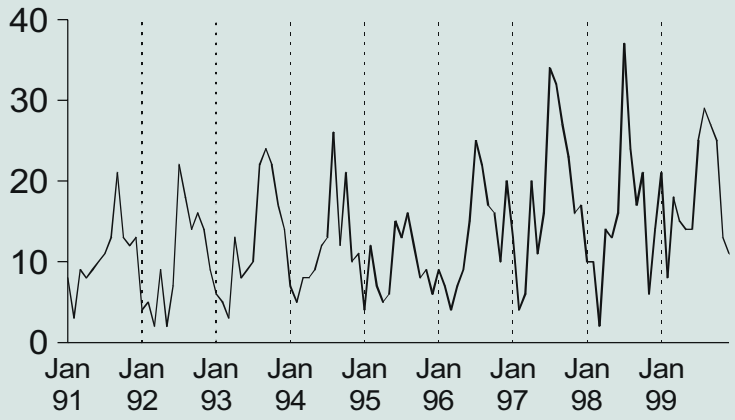
Incidence varied widely with age: it was highest among children less than one year old (32.5/100,000), and gradually declined with increasing age in early childhood. Children under five years of age had an incidence of 16.2/100,000, and accounted for 44 per cent of all notified cases. The next highest incidence was among adolescents aged 15–19 years (6.0/100,000). Incidence was broadly similar among other demographic variables, including sex and place of residence. Approximately half the cases presented with meningitis, a quarter with septicaemia and the mode of presentation of the remainder was not reported (see Table 6).

Seventy percent of cases (*n*=1011) were reported to be confirmed by a laboratory. In 1991 this proportion was only 40 per cent, but in all subsequent years was around 70 per cent. In 1999, 72 per cent (*n*=159) were laboratory confirmed.

For the nine-year period, information about the meningococcus serogroup was available for only 47 per cent (*n*=476) of laboratory-confirmed cases. Of these, 51 per cent were serogroup B, 45 per cent were serogroup C, two per cent were serogroup W135, and two per cent were serogroup Y. The proportion of laboratory-confirmed cases on whom serogrouping was reported has steadily increased from zero per cent in 1991. In 1999, this information was available on 80 per cent (*n*=128) of laboratory-confirmed cases, of which 58 per cent were serogroup B, 40 per cent were

FIGURE 6

NOTIFICATIONS OF MENINGOCOCCAL DISEASE BY MONTH OF ONSET, NSW, 1991 TO 1999



serogroup C, three per cent were serogroup W135, and less than one per cent were serogroup Y.

Hospitalisations

During the period 1991–1997 there were 961 admissions recorded with a primary diagnosis of meningococcal disease in Australia among NSW residents. For the same period 1,040 cases of meningococcal disease were notified in NSW. The extent of overlap between notified and hospitalised cases was not able to be assessed, however, the total hospitalised cases represented 92 per cent of notified cases for this period (see Table 5). The distributions of hospitalised cases and notified cases were broadly similar by sex and age group.

Deaths

During the period 1991–1999, there were 86 deaths from meningococcal disease notified to public health units (six per cent of all cases). By year, the deaths varied from two per cent (*n*=3) in 1991 to 15 per cent (*n*=11) in 1994, and in 1999, six per cent (*n*=14) of cases were reported to have died. Case fatality rates were similar by place of residence, sex, or serogroup, however, it was higher in people aged 45 years and older (12 per cent) than in younger people (five per cent) (relative risk [RR]=2.2, 95 per cent confidence intervals [CI] = 1.3 to 3.6). The case fatality rate was almost four times higher among those presenting with septicaemia (12 per cent) than with meningitis (three per cent), RR=3.8, 95 per cent CI= 2.4 to 6.1) (see Table 6).

DISCUSSION

These data indicate that in NSW, meningococcal disease is rare, but fatal in about one in 17 cases. The disease is more common in small children and adolescents, and the case-fatality rate tends to be high in older adults, and in people presenting with septicaemia.

Surveillance of meningococcal disease can provide basic information on the burden, case fatality rates, and demographic risk factors for disease. Effective surveillance of infectious diseases is often hampered by under-reporting. However, since we can expect that almost all diagnosed cases of meningococcal disease require hospital monitoring and treatment, the comparison of cases identified by the hospital ISC suggests that under-reporting is minimal in NSW. This comparison is limited in that it was not possible to link individuals in the ISC data with those notified, and by the possibility of misclassification errors in the ISC. A substantial number of case reports lack specific information on the meningococcus serogroup that caused the disease, limiting our ability to assess shifts in the distribution of serotypes over time. Available data, however, suggest fairly stable serogroup patterns during the study period.

Rapid identification and reporting of cases allows public health workers to assess whether other people are at increased risk for disease, and thereby help control the further spread. NSW Health will continue efforts to improve the surveillance of meningococcal disease through more complete reporting of serogroup data.

ACKNOWLEDGEMENT

Thanks are due to public health unit staff, doctors, and hospital and laboratory staff for providing their data.

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FACT*SHEET*

ROSS RIVER VIRUS INFECTION

WHAT IS ROSS RIVER VIRUS?

- Ross River virus is a germ that infects people, particularly in rural areas, sometimes causing a flu-like illness with joint pains.
- Ross River virus is not fatal.

HOW IS ROSS RIVER VIRUS SPREAD?

- The virus is spread by certain types of female mosquitoes.
- Female mosquitoes feed on animals and people. If they feed on the blood of an infected animal, the mosquito may become infected. The virus then multiplies within the mosquito and is passed to other animals or people when the mosquito feeds again.
- Infections tend to peak in the summer and autumn months.
- The virus is not spread directly from one person to another.

WHAT ARE THE SYMPTOMS OF ROSS RIVER VIRUS INFECTION?

- Many people who are infected with the virus will never develop symptoms.
- Some people will have flu-like symptoms that include fever, chills, headache and aches and pains in the muscles and joints.
- Some joints can become swollen, and joint stiffness may be particularly noticeable in the morning.
- Sometimes a rash occurs on the body, arms or legs. The rash usually disappears after seven to 10 days.
- A general feeling of being unwell, tired or weak may also occur at times during the illness. This may affect work performance.

HOW SOON DO SYMPTOMS DEVELOP AFTER BEING BITTEN BY AN INFECTED MOSQUITO?

- Symptoms usually show between seven to nine days after being bitten by an infected mosquito. This interval can vary but is generally between five to 21 days.

HOW LONG DOES THE ILLNESS LAST?

- Many people will recover completely within a few weeks. Others have symptoms lasting more than three months, and in rare cases for more than a year.
- The symptoms can re-occur on and off over this period of time. Usually the symptoms become less severe each time they recur.
- A full recovery can be expected.

WHAT IS THE TREATMENT FOR ROSS RIVER VIRUS INFECTION?

- There is no specific treatment for Ross River virus infection.
- Your doctor will be able to advise you on medications that will help ease the discomfort of the symptoms.
- Plenty of rest, along with moderate exercise and healthy eating, may help in your recovery.

HOW DO I KNOW IF I HAVE ROSS RIVER VIRUS INFECTION?

- If you have symptoms, see your doctor, who can order a blood test to diagnose Ross River virus infection.

CAN ROSS RIVER VIRUS DISEASE BE PREVENTED?

- Yes! Avoid being bitten by mosquitoes, especially in the summer and autumn months when infections peak.
- Various species of mosquitoes bite at different times. Avoid being outside in the late afternoon and dusk. Mosquitoes are usually most active up to one to three hours after sunset and again around dawn.
- When outside wear loose fitting, light coloured clothing that covers your arms and legs, and use an insect repellent that contains the chemical diethyl toluamide (DEET).
- Fit fly screens to all windows, doors and chimneys and keep them in good repair.
- Use a knockdown insecticide in bedrooms half an hour before going to bed. Use insecticides according to instructions.

For more information please contact your local public health unit, local government, community health centre, pharmacist, doctor or www.arbovirus.health.nsw.gov.au

A CLUSTER OF LOCALLY-ACQUIRED ROSS RIVER VIRUS INFECTION IN OUTER WESTERN SYDNEY

Tim Brokenshire, Doug Symonds and Roderick Reynolds
Wentworth Population Health Unit

Stephen Doggett, Merilyn Geary and Richard Russell
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This article reports on the investigation of a cluster of Ross River virus (RRV) infections in the outer western suburbs of metropolitan Sydney. Ross River virus is an arbovirus transmitted by mosquitoes, and its symptoms include arthralgia, myalgia, arthritis, fatigue, fever, and headache.[1] A rash, usually maculopapular and found on the trunk and limbs, accompanies these symptoms in 40–60 per cent of patients.[2] Duration and severity of symptoms can vary, with full recovery taking from several months to a number of years.[3]

Ross River virus is most often considered a disease of rural Australia.[4,5] However, an outbreak of RRV infection identified among people living in the north-western outskirts of Sydney, and outbreaks in Brisbane, Perth and south-western Australia, reveal the potential for RRV activity to spread to metropolitan areas.[6,7,8,9]

BACKGROUND

In early 1999, the Western Sector Public Health Unit (WSPHU) began receiving an increased number of notifications of RRV virus. Follow-up was conducted by the WSPHU, and a team from the Department of Medical Entomology at the Institute of Clinical Pathology and Medical Research (ICPMR) conducted an intense short-term mosquito trapping program in the Werrrington area to identify possible local vectors associated with a cluster of RRV infection. The Werrington area is a residential area within the boundaries of the Wentworth Area Health Service, split by a creek and a park. At the south end are the large open grasslands of the University of Western Sydney (Nepean). At the northern end is a large natural woodlands and wildlife area with an abundant macropod population.

METHODS

Infectious Disease Team

For the purpose of this investigation a confirmed case of RRV infection was defined as being any person:

- residing in the Wentworth Health Area, comprising the local government areas of Penrith, Blue Mountains and Hawkesbury;
- notified between January 1 and May 31 1999 by a laboratory or doctor, with a clinical onset after 31 December 1998;
- with two blood tests, collected at least two weeks apart, demonstrating a four-fold or greater increase in IgG antibody titre to RRV.

On notification of the first result, WSPHU staff contacted the referring doctor to ascertain the cases' clinical presentation, possible onset date and a travel history (if known). An exposure period was considered to be in the range of 3–28 days before onset of symptoms. This wider range than the 3–21 day incubation period was used to help account for any inaccuracies in the recollection of the case's activities and mosquito exposures.[10] The case was then contacted and a questionnaire administered to confirm symptoms, onset and travel history. Information was also recorded on where the disease may have been acquired. Both the case and referring doctor were advised of the need for convalescent phase sera to be collected at least two weeks after the initial serology.

Environmental Health Team

Adult mosquito trapping

Weekly mosquito collections were conducted over a three-week period from late March to mid-April 1999. Dry-ice baited light traps were set at 10 sampling sites within the Werrington area,[11] located in the Penrith Local Government Area. Trapping sites were located 3–5 km in a north-easterly direction from the Penrith train station in the approximate area from which the human infections were reported, or near local mosquito habitats.

Arbovirus isolation

The mosquito collections were transported live to the Department of Medical Entomology at ICPMR, where they were identified and processed for virus isolation and identification of alpha- and flaviviruses.[12]

RESULTS

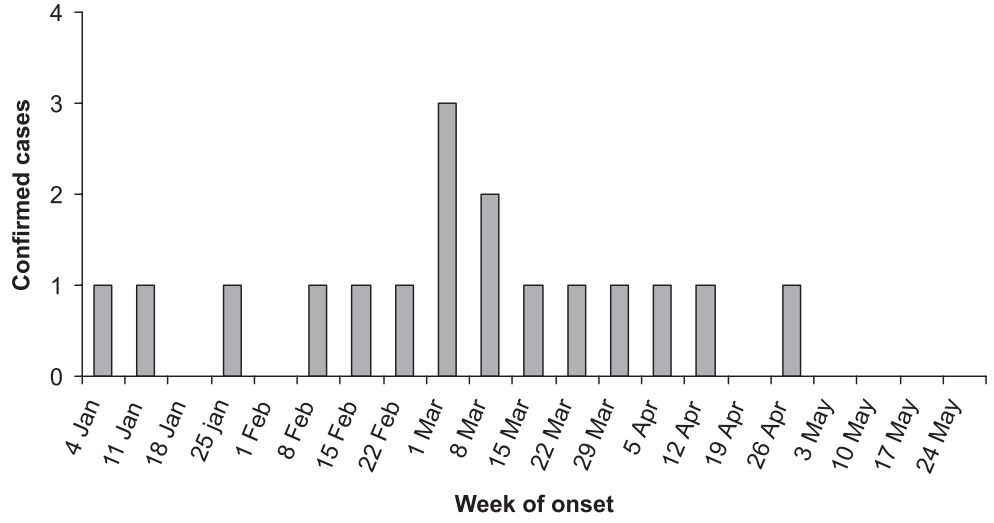
Infectious Disease Team

Between January and May 1999, 37 notifications for RRV virus were received by WSPHU. Twenty-six cases had repeat serology collected. Twenty-five cases were confirmed using the case definition. One case showed no rise in antibody titre after the repeat serology and wasn't classified as a confirmed case.

Histories were obtained for each confirmed case. Of the confirmed cases, 17 cases had not travelled beyond the boundaries of the Wentworth Area Health Service while eight cases had travelled to other areas. Of the 17 who had not travelled outside the area, 15 reported being bitten by mosquitoes in the exposure period prior to the onset of symptoms. Of those who had travelled out of the area, six reported contact with mosquitoes while two could not recall being bitten by mosquitoes.

FIGURE 7

SYMPTOM ONSET OF ROSS RIVER VIRUS, WERRINGTON NSW, JAN–MAY 1999.



Results of the case histories identified three cases who lived in the Werrington area, and who recalled being bitten in the local area. A further case was identified as a man who worked in the Werrington area. The onset of symptoms rose during the early months of 1999 and peaked in March with numbers declining in the following months (Figure 7).

Environmental health team

Adult mosquito trapping

Seventeen species of mosquito were collected. *Aedes notoscripus* and *Culex annulirostris* were the most common species, and both have been associated with RRV. *Culex annulirostris* is the major vector of RRV in inland NSW; *Aedes notoscripus* is a competent laboratory vector, has been found naturally infected, and is suspected as a vector for urban areas. Other species recorded appeared infrequently or rarely, and were less significant as potential vectors in the circumstances.

Arbovirus isolation

One hundred and ninety-eight mosquito pools were processed. RRV was not isolated from any of the specimens examined. However, a Stratford virus was isolated from a mosquito trapped on 8–9 April 1999.

DISCUSSION

The investigation of these notifications identified four cases of RRV infection that were acquired in Werrington, between January and March, 1999. While Amin et al. identified a cluster of cases in a rural setting in the Sydney basin,[6] we believe that this is the first report of a cluster of RRV cases acquired in metropolitan Sydney.

The number of confirmed cases may have been limited by the need to obtain paired sera in order to rule out false positives and past infection. While each case and their doctor was made aware of the need for a second serology, this was not always obtained. Reliance on the cases to recall onset date, place of acquisition and travel history, may have also had an effect on the result of the investigation.

The trapping program undertaken at Werrington occurred when mosquito abundances were on the decline due to cooler autumn weather. Thus, the number and species collected may not be a true reflection of the activity during the warmer summer months, when vector populations would normally peak. However, several mosquito species that are considered important vectors of pathogens were trapped: *Aedes notoscripus*, *Culex annulirostris* and *Culex annulirostris*.

Despite the intense trapping program, there were no isolates of RRV. Ross River virus activity may have ceased, however, as trapping occurred 2–5 weeks after the likely date of the majority of human infection acquisitions. Other viruses may have been active during this period, as evidenced by the isolation of Stratford virus in early April. It appears that the common domestic breeding mosquito, *Aedes notoscriptus* may have a role in the transmission of this virus which could become more common in NSW, with three other Stratford virus isolates collected from the Sydney region in 1999 from *Aedes notoscriptus* (Doggett and Russell, unpublished data). Clinical infections with Stratford virus have been recorded, with symptoms including fever, lethargy and arthritis.[13]

The close proximity of some outer western Sydney suburbs to natural woodlands, abundant macropod hosts and local vector breeding suggests that local residents may be at increased risk of RRV infection. General Practitioners in the area should consider the diagnosis of RRV infection in patients with consistent symptoms, even if there is no history of travel to endemic areas, and should encourage the collection of convalescent serology. Enhanced surveillance of human RRV infections and enhanced mosquito trapping activities in conjunction with local councils is being undertaken for the year 2000 season. Currently four traps are located within the Penrith region as part of the NSW Arbovirus Monitoring Program. The Werrington area is now included in the Program for the 1999–2000 season.

From a public health perspective there are a number of implications arising from these findings. Firstly, there is a greater realisation of the potential for RRV activity to spread to metropolitan areas. There is an increased need for collaboration between a number of agencies and professionals to provide enhanced disease surveillance and identification of mosquito vectors. Secondly, there is a health promotion component, which indicates a greater need for accessible community information regarding personal protection methods in areas not previously known to be endemic for RRV.

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INFECTIOUS DISEASES, NSW: JULY 2000

TRENDS

Laboratory-confirmed cases of **mumps** have increased recently, with 24 notified in the three months to the end of May 2000. Twenty-five cases were notified in 1999, 39 in 1998, 29 in 1997 and 27 in 1996. Of the 24 cases notified in the last three months, 95 per cent were from Sydney, 71 per cent were aged between 5–24 years, and 62 per cent were males. Laboratory-confirmed cases of mumps are likely to represent only a small fraction of all infections occurring in the community.

Reports of **Ross River virus** infection rose in May 2000, with 295 cases notified from a number of rural Areas. Notifications of **legionnaires disease** increased with seven cases reported in April. Investigation of these seven cases showed no common causal links. No cases of **measles** were notified in May (see Table 7, Figure 8).

UPDATED INFECTIOUS DISEASE NOTIFICATION FORMS

Doctors, hospital and laboratory staff will be pleased to learn that forms for the confidential notification of scheduled medical conditions (that is, conditions reported in the Bulletin), including special forms for the notification of AIDS and death following HIV, have been updated and are available on the Internet. All forms are provided as Acrobat PDF files and are available from **www.health.nsw.gov.au/public-health/forms**. Notifications of scheduled conditions should be made by telephone or mail to the local public health unit, and cannot be made via the Internet.

FIGURE 8

REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, JANUARY 1995 TO MAY 2000, BY MONTH OF ONSET

These are preliminary data: case counts in recent months may increase because of reporting delays

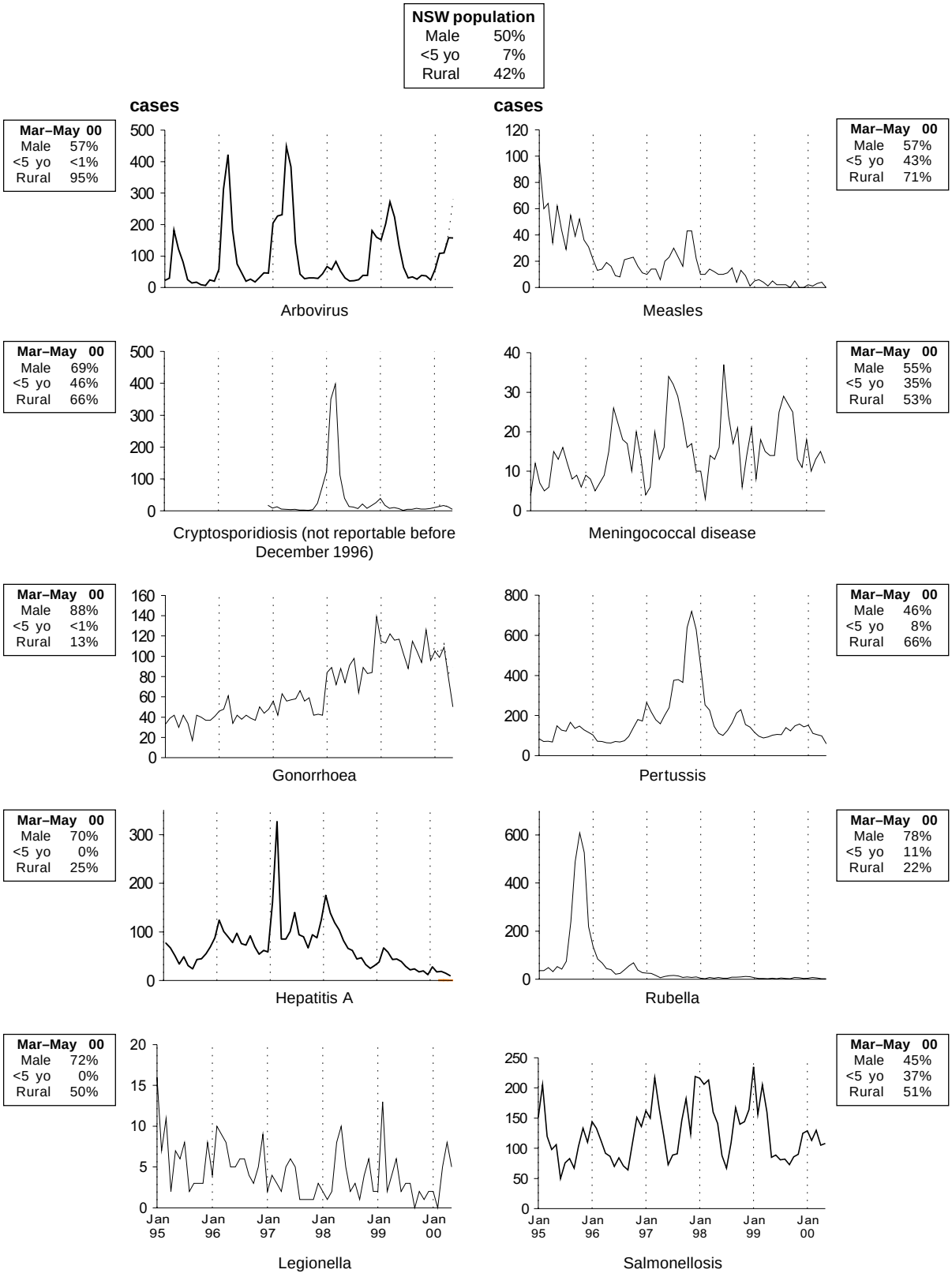

TABLE 7 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN MAY 2000 BY AREA HEALTH SERVICES

Condition	Area Health Service (2000) CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	Total for May**	Total To date†
Blood-borne and sexually transmitted																			
AIDS	-	-	-	1	-	-	-	-	4	1	-	1	-	-	-	-	-	7	60
HIV infection*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	90
Hepatitis B - acute viral*	-	-	1	-	-	-	1	-	1	-	-	-	-	-	-	-	-	3	32
Hepatitis B - other*	52	36	45	5	80	1	7	3	38	3	-	8	2	4	2	7	4	300	1,857
Hepatitis C - acute viral*	-	-	-	-	-	1	1	-	-	-	-	-	1	-	-	-	-	3	28
Hepatitis C - other*	75	25	115	39	45	25	51	23	92	37	13	11	11	37	2	21	19	648	3,787
Hepatitis D - unspecified*	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	2
Hepatitis, acute viral (not otherwise specified)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	25	16	20	6	4	7	29	12	43	17	12	11	5	5	8	8	3	231	1,146
Gonorrhoea*	23	4	5	-	4	-	-	3	52	5	-	4	1	-	1	-	-	104	509
Syphilis	7	-	-	1	5	-	-	-	10	3	1	2	1	2	3	-	-	36	222
Vector-borne																			
Arboviral infection (BFV)*	-	-	-	-	-	1	2	2	-	7	20	-	1	-	1	-	-	34	102
Arboviral infection (RRV)*	-	-	-	2	-	7	37	2	1	11	27	36	35	9	22	11	5	205	476
Arboviral infection (Other)*	-	1	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	2	22
Malaria*	1	-	-	-	4	-	1	-	2	2	2	-	-	-	-	-	-	12	76
Zoonoses																			
Brucellosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Leptospirosis*	-	-	-	-	-	-	1	-	-	4	1	1	-	1	-	1	1	12	24
Q fever*	-	-	-	-	-	-	-	-	-	5	3	-	1	-	-	-	-	9	48
Respiratory and other																			
Blood lead level*	7	1	-	2	13	-	138	1	3	-	-	-	-	-	1	1	-	167	421
Legionnaires' Longbeachae*	-	-	-	-	-	-	-	-	1	-	-	-	-	1	-	-	-	2	4
Legionnaires' Pneumophila*	1	-	1	-	-	-	-	2	1	-	-	-	1	-	-	-	-	6	15
Legionnaires' (Other)*	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1	2
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Meningococcal infection (invasive)	1	1	2	-	1	1	1	2	1	-	1	-	-	-	-	-	-	11	69
Mycobacterial tuberculosis	3	7	2	1	1	-	1	2	7	-	-	-	1	1	-	-	-	26	167
Mycobacteria other than TB	5	7	-	1	-	1	4	-	4	2	-	-	-	1	-	1	2	29	141
Vaccine-preventable																			
Adverse event after immunisation	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1	4
H.influenzae b infection (invasive)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Measles	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	10
Mumps*	1	1	5	1	4	-	-	-	1	-	-	-	-	-	-	-	-	13	30
Pertussis	8	10	12	10	11	7	33	2	7	2	5	15	7	11	-	6	3	149	667
Rubella*	-	-	-	-	-	-	1	-	2	-	-	-	-	-	-	-	-	3	22
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Faecal-oral																			
Botulism	-	-	-	-	-		-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera*	-	-	-	-	-		-	-	-	-	-	-	-	-	-	-	-	-	-
Cryptosporidiosis*	1	-	-	1	-		-	-	2	1	1	-	-	-	-	1	-	7	61
Giardiasis*	9	10	10	4	1		5	2	13	13	1	6	4	3	-	1	-	90	459
Food borne illness (not otherwise specified)	-	-	-	-	-		-	-	26	-	-	-	-	-	-	-	-	26	88
Gastroenteritis (in an institution)	-	-	-	-	-		7	-	-	-	-	-	-	-	-	-	-	7	55
Haemolytic uraemic syndrome	-	-	-	-	-		-	1	-	-	-	-	-	-	-	-	-	1	4
Hepatitis A*	3	2	3	-	-		-	-	4	-	1	-	-	2	-	-	-	16	97
Hepatitis E*	1	-	1	-	-		-	-	1	-	-	-	-	-	-	-	-	3	6
Listeriosis*	-	-	-	-	-		-	-	-	-	-	-	-	-	-	-	-	-	5
Salmonellosis (not otherwise specified)*	16	26	-	3	9	9	8	6	30	18	7	8	1	7	-	17	1	168	646
Typhoid and paratyphoid*	-	-	3	-	-		-	-	1	-	-	1	-	-	-	-	-	5	17
Verotoxin producing Ecoli*	-	-	-	-	-		-	-	-	-	-	-	-	-	-	-	-	-	-

* lab-confirmed cases only

† includes cases with unknown postcode

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