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Lessons from a respiratory illness outbreak in an aged-care facility

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Abstract: This report outlines practical lessons learnt from an influenza-like outbreak in an aged-care facility in NSW, which affected 26 residents, resulted in 14 hospital admissions and was associated with six deaths. No common causative agent was identified. Key recommendations include: encouraging aged-care facilities to establish mechanisms that improve the early identification of outbreaks and timely implementation of outbreak control strategies; identifying strategies to inform general practitioners of outbreaks if they have patients residing in aged-care facilities; and improving the vaccination coverage of the aged-care workforce.

Outbreak in an aged-care facility

Respiratory outbreaks in residential aged-care facilities are the cause of significant mortality and morbidity.[1,2] An outbreak of an influenza-like illness occurred among residents and staff of a multi-purpose residential aged-care facility located on the north coast of New South Wales (NSW) in September–October 2006. At the time of the outbreak, there were 60 residents, ranging in age from 64 to 96 years, and 75 staff. The facility caters for residents with low- and high-care needs, and includes a purpose-built dementia facility. A high proportion of residents had previously been vaccinated against influenza A and B (96%; $n = 58$); however, vaccination coverage was low among staff members (27%; $n = 20$).

Public health response

Course of the outbreak

The outbreak extended over a 7-week period starting on 2 September 2006, with onset of the last case reported on 20 October 2006. A total of 26 residents (43%) and one staff member (1%) were affected. Of these, 14 residents (54%) were hospitalised and six (23%) died.

Immediate actions taken by the public health unit and aged-care facility

On 17 October 2006 the North Coast Area Health Service (NCAHS) Public Health Unit (PHU) was notified of the outbreak. The aged-care facility was advised to implement several strategies to contain the outbreak including: isolating sick residents; excluding sick staff; increasing infection control practices; deferring admissions; and discouraging non-essential visitors to the facility. The facility was also provided with fact sheets and the *Guidelines for the prevention and control of influenza outbreaks in residential care facilities in Australia*.[3]

During a site visit on the day following notification to the PHU, nasal and throat swabs and blood samples were taken from ill residents at the facility and from residents who had been admitted to hospital. In addition, PHU staff collected and reviewed information about affected staff and residents, including basic demographics, symptoms and onset date from those who had experienced symptoms since 2 September 2006. PHU staff also found that the facility was experiencing difficulty in sourcing protective (P2) masks. These are high efficiency, disposable masks with the capacity

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Table 1. Frequency of symptoms among affected residents and staff at a residential aged-care facility on the north coast of NSW, September–October 2006 ($N = 27$)

Symptoms	Number affected
Temperature >38°C	27
Cough	24
Fatigue	25
Weakness	18
Aches and pains	4
Chills	18
Runny nose	5
Sore throat	1
Shortness of breath	1
Diarrhoea	1
Vomiting	2

to filter up to 95% of airborne particles with an aerodynamic diameter of 0.3 micron or more. The PHU was able to provide additional mask stocks.

Approximately 30 general practitioners who provided care to residents of the facility were notified of the outbreak by a letter forwarded by the facility. Antibiotics were prescribed for some symptomatic and asymptomatic residents by their general practitioners. Where possible, convalescent serology was collected 4 weeks later.

Diagnosis and testing

After considering relevant national guidelines, the range of symptoms reported and the occurrence of laboratory-confirmed influenza in surrounding locales, a case definition was formed.[3] For this outbreak, a case was defined as a resident or staff member of the facility who had a fever of 38°C or more, and at least two of the following symptoms with onset after 2 September 2006: chills, cough, fatigue, aches and pains (including severe pain), diarrhoea, vomiting and hypertension. A summary of the symptoms experienced by affected people is presented in Table 1.

Symptomatic residents received point-of-care tests for influenza A and B during an initial site visit. Point-of-care tests, or tests that occur at the patient's beside, allow physicians to diagnose patients more rapidly than traditional laboratory-based testing. The Victorian Infectious Diseases Reference Laboratory tested nasal and throat swabs and blood samples taken from sick residents at a later date, for a range of conditions including: influenza A and B, parainfluenza, respiratory syncytial virus, adenovirus, *Chlamydophila psittaci*, *Mycoplasma pneumoniae* and *Legionella pneumophila*. Test selection was guided by avian and pandemic influenza protocols and *Guidelines for the prevention and control of influenza outbreaks in residential care facilities*, as well as potentially relevant environmental features of the facility, such as water fountains and domestic birds.[3–5]

Ongoing response to the outbreak

Two days after the outbreak notification, on 19 October, a teleconference was convened to discuss: the status of the outbreak, action taken, potential sources of infection and future management of the outbreak. The discussion group consisted of: the facility's Acting Director of Nursing; the Director of Medical Services and the infection control practitioner from the local hospital; and staff from the PHU and the Communicable Diseases Branch of NSW Department of Health. The possibility of further hospital admissions and the range of pathology tests to be undertaken were also discussed.

Throughout the outbreak, the PHU was in regular (at least daily) contact with the facility and the local hospital, and provided updates on the status of the outbreak, the likelihood of further hospitalisations and the health status of residents at the facility.

A second site visit was conducted on 20 October in order to:

- review the effectiveness of the infection control strategies implemented by the facility
- identify potential sources of infectious agents
- interview residents and staff members
- collect additional nasal and throat swabs and blood samples from ill and convalescing residents.

Samples were initially sent to the local pathology service for analysis. However, due to the number of sample batches, the range of tests required and the need for rapid processing, the samples were forwarded to the Victorian Infectious Diseases Reference Laboratory for analysis, as described above. Where possible, convalescent serology was collected 4 weeks after the first visit.

The results of the serological tests were inconclusive and no common causative agent was identified. One of six point-of-care tests performed was positive for both influenza A and B; however, the sensitivity and specificity of these tests is limited.[6] Serology of convalescent patients also failed to identify a common causative agent.

Media issues

The outbreak received considerable media interest. Initially the aged-care facility was reluctant to make any public comment. Following increasing media speculation about the nature and extent of the outbreak, the NCAHS issued a statement on 30 October 2006 confirming that an outbreak of an unidentified infectious respiratory illness had been associated with the death of elderly people in residential care and requesting unwell people to avoid visiting aged-care facilities and health-care facilities. The media statement did not identify the aged-care facility nor provide details of the number of suspected cases or deaths. In response, a local newspaper used funeral notices from the previous week to speculate that up to 13 people over the age

of 80 had died as a result of the outbreak. The following day the parent company of the aged-care facility issued a statement that appeared to conflict with the NCAHS statement regarding the link between recent resident deaths and the respiratory illness. This statement aroused further media interest and led to the involvement of the NSW Shadow Minister for Health. The local Member of Parliament wrote to the NSW Minister for Health requesting that the matter be investigated.

Public health lessons

Interaction between public health units and aged-care facilities

Several valuable lessons were learnt as a result of this outbreak. Most importantly, it highlights the need either to meet with aged-care providers regularly or involve them in periodic emergency management training sessions. This regular contact with aged-care facilities would emphasise the importance of early outbreak notification and close liaison with the PHU on matters such as vaccination and infection control. In this outbreak, the delay in the facility identifying and notifying the PHU of the outbreak may have contributed to difficulties in containing its spread and led to increased media interest. With support from PHUs, aged-care facilities should be encouraged to establish sentinel surveillance systems to improve outbreak recognition and timely implementation of infection control strategies. The NCAHS PHU has written to all residential aged-care facilities in the region to advise them of the resources available to assist in dealing with outbreaks, including notification procedures.

Laboratory testing

Considerable delay was experienced in receiving serological test results. Increased clarity about the tests available at reference laboratories and improved efficiency in transporting specimens to laboratories during an outbreak may shorten turnaround times for test results and aid the identification of appropriate treatment and infection control measures. The management of this outbreak would have benefited from closer liaison between the PHU and pathology laboratories, and the advocacy of the NSW Public Health Laboratory Liaison Officer when laboratory-related issues arose.

Informing general practitioners

Appropriate strategies for informing general practitioners who have patients residing in affected facilities need to be identified. In the case of this outbreak, the only available means of notifying the general practitioners of the residents of the outbreak was by a letter from the facility. This emphasises the need for a collaborative response to outbreaks between aged-care facilities, attending general practitioners and PHUs. It also reinforces the need for collaboration with Divisions of General Practice and aged-care panels and committees, where they exist, and the importance of seeking the consent of residents and the facility for information-sharing between agencies.

Controlling the outbreak

Although influenza was not identified as the likely causative agent in this outbreak, it provides important incidental lessons for influenza control in aged-care facilities. Researchers have reported that elderly residents of aged-care facilities often have an impaired immune response to influenza vaccinations due to age or co-morbidities.[7,8] Influenza outbreaks have been reported in aged-care facilities with high levels of vaccination coverage (more than 85%), even when the vaccine used matches the circulating influenza strain.[9,10] In this facility, influenza vaccination coverage among residents was high (96%); however, vaccination levels among staff were low (27%). This low vaccination rate may in part be because influenza vaccinations are not provided free of charge to staff of aged-care facilities. One high priority for preventing influenza outbreaks in aged-care facilities is to prevent the introduction of the virus into the facility by ensuring high levels of vaccination coverage among all health-care workers and visiting general practitioners. To achieve this outcome, incentives for facilities to fund staff vaccinations could be considered. Increasing vaccination coverage among visitors is harder to achieve. One strategy is to educate the general public about the risks of transmitting respiratory illnesses when visiting health-care facilities. Appropriate signage at aged-care facilities and advice in the media during outbreak events are additional potential strategies that would reinforce these messages.

Liaison between care and health organisations

A standardised notification procedure to a defined point within the Australian Government Department of Health and Ageing would assist PHUs and the NSW Department of Health to provide timely outbreak notification. Identification of an appropriate mechanism to advise the NSW Ambulance Service of outbreaks in private facilities is also necessary.

Managing the outbreak through regular liaison with the affected facility, local hospital and PHU was considered beneficial for all, as this allowed joint problem-solving and information sharing. It also assisted in planning for potential hospital admissions and discharges. It would have been useful for this group to have jointly issued a media release to avoid the controversy that ensued following the perceived contradiction between the PHU and the facility's media releases.

Summary

Respiratory illness outbreaks in residential aged-care facilities cause significant mortality and morbidity. This outbreak resulted in 14 hospital admissions and was associated with six deaths among elderly residents. No common causative

agent was identified. The practical lessons learnt that may be of use to other PHUs include:

- encouraging aged-care facilities to establish mechanisms for effective early identification of outbreaks and timely implementation of infection-control strategies
- identifying effective strategies to inform general practitioners, the NSW Ambulance Service and the Australian Government Department of Health and Ageing of outbreaks in aged-care facilities
- improving the vaccination coverage among the aged-care health workforce.

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Evidence of pertussis clusters in three aged-care facilities in the former Macquarie Area Health Service, NSW

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Abstract: During a limited outbreak of pertussis in an area health service in NSW, three clusters occurred in aged-care facilities – the first reported outbreaks of pertussis in Australian nursing homes. The attack rates across the three clusters were 16.7% for staff and 15.7% for residents. Our investigation revealed that older adults are not immune to pertussis. We suggest methods for controlling a pertussis outbreak in an aged-care facility.

Pertussis is an acute infectious disease that is vaccine preventable but cases still occur because vaccination coverage is not complete and protection wanes with time. Although it is usually regarded as a childhood illness, where severe morbidity and mortality of the disease occur in very young children, pertussis can occur at any age.

Pertussis appears to be endemic in the former Macquarie Area Health Service (now part of Greater Western Area Health Service) in New South Wales (NSW). Epidemics occur in addition to a background of regularly reported cases. We describe here three clusters of pertussis in aged-care facilities (private nursing homes) that occurred as part of an outbreak in 2004. The clusters occurred in two towns within the area health service.

Index case A: aged-care facility 1

On 8 July 2004, the Macquarie Area Health Service Centre for Population Health was notified of a possible case of pertussis in a staff member of an aged-care facility (facility 1) in a town in the northern part of the area. The staff member had a spasmodic cough and a high serum IgA titre to pertussis (IgA positive). The local general practitioner (GP) managed the person as an incident case. The GP initiated treatment and advised the patient to stay at home. This became index case A.

It was initially thought that the positive blood test of index case A represented a persistent IgA response to previous pertussis, notified in 2000. As a precaution, the facility was advised to carry out active case finding and appropriate tests. This strategy involved testing all people in the facility with a cough and those who developed a cough in the two to three weeks following identification of index case A. A diagnosis of pertussis was defined as IgA positivity in the presence of a clinically compatible illness.

Index case B: aged-care facilities 2 and 3

On 9 September 2004, in another town, a case of pertussis (positive IgA) was reported in an assistant in nursing who worked in two aged-care facilities (facility 2 and facility 3), and who had had a coughing illness compatible with pertussis for two to three weeks. The date of onset of the illness was between 16 and 23 August (Epiweek 34). The treating GP had started the patient on erythromycin for 14 days and provided a sick leave certificate for five days.

Public health response

At facility 1, the public health response aimed to ensure that cases and contacts were treated appropriately to limit infection. Symptomatic staff were advised not to attend work until five days of recommended antibiotic treatment had been completed. The facility was advised to implement appropriate infection control guidelines, including: personal hygiene and personal protective equipment; minimising visitors (especially children and pregnant women); and early detection of new cases by testing all staff or residents who developed a cough.

At facility 2 and facility 3, the public health response was similar. With the permission of the patient, both facilities were informed of the case of pertussis in a casual staff member and were sent fact sheets containing information about the disease. Each facility was actively encouraged to seek new cases among staff and residents, and to test anyone who had a coughing illness.

Table 1. Cases of pertussis in three aged-care facilities in the former Macquarie Area Health Service (now part of Greater Western Area Health Service) in NSW, 2004

	Number of staff	Number of residents	Staff cases	Resident cases	Staff attack rate (%)	Resident attack rate (%)
Facility 1	22	24	2	7	9.10	29.20
Facility 2	90	80	21	15	23.30	18.80
Facility 3	67	62	7	3	10.50	4.80
Total	179	166	30	25	16.70	15.70

Response at facility 1

Three weeks after the notification of index case A, a further eight cases were reported from facility 1. A number of residents and staff had developed a cough and had had blood tests. Seven out of 24 residents and one additional staff member were IgA positive and reported to have a cough (Table 1).

Over the next week, other concerned asymptomatic staff members arranged to be tested and four more asymptomatic staff were found to be IgA positive. Being asymptomatic, these people were not regarded as cases.

Response at facility 2

Facility 2 actively identified and tested residents with a cough. A number of IgA positive tests among the residents were reported on 15 September 2004 (Table 1).

In response, an investigation team visited facility 2 to obtain epidemiological information about the people who tested positive in order to support the facility and advise on appropriate infection-control procedures. Detailed information about the cases was difficult to obtain as age, dementia and illness meant that most of the people could not remember or communicate their immunisation status or could not remember having previously had a whooping cough-like illness.

The team recommended that all residents who had developed a cough in the preceding three weeks should receive antibiotic treatment regardless of IgA status. All residents who developed a cough in the following week were treated empirically (i.e. treatment was initiated before a diagnosis of pertussis was confirmed).

Symptomatic staff were advised to be tested, to be treated and to take leave from work until they had completed five days of antibiotic treatment. A fact sheet was distributed to visitors, and signs were posted to exclude high-risk category visitors (children less than one year of age, children between 12 months and 5 years who were not fully immunised and women in the last month of pregnancy). This outbreak and the infection control procedures generated local media interest and a media release was issued and followed up with television and radio interviews.

Response at facility 3

Despite having received the same information and advice as facility 2, facility 3 did not actively identify residents and staff with a cough. Facility 3 had fewer symptomatic residents and staff. A few people developed a cough in the following weeks and tested positive (Table 1).

Results

The number of staff and residents found to have pertussis and the attack rates in each of the three aged-care facilities are shown in Table 1. In each facility, a significant proportion of the staff was affected. Facility 2 and facility 3 were situated in the same town. In both facilities, a significantly larger proportion of staff were affected than residents.

A review of the notifications for pertussis in facility 2 and facility 3, and a calculation of their onset dates, revealed five cases with onset before index case B.

Discussion

These three clusters arose during 2004, a high incidence year for pertussis in the former Macquarie Area Health Service. The clusters occurred in two different towns in the area: facility 2 and facility 3 were situated in the same town and had a significantly larger proportion of staff affected than residents.

Pertussis has generally been considered a disease of children. In NSW, there has been a shift in the age distribution of new cases of pertussis from pre-teens to adolescents.[1] Pertussis is not assumed to be significant in adults. Our data clearly show that outbreaks of pertussis can occur in elderly people. Other researchers have found an incidence rate of serologically defined infection of 19.7 per 100 person-years in a group of 100 people aged over 65.[2] Pertussis in adults produces considerable morbidity and, in addition, provides a significant reservoir of infection for children.

Outbreaks of infectious diseases such as influenza are well known to occur in nursing homes.[3,4] Pertussis outbreaks have less commonly been reported: an outbreak of pertussis in a Wisconsin nursing home in the 1980s and an outbreak in severely handicapped patients in a neurological ward have previously been described.[5,6] This occurrence

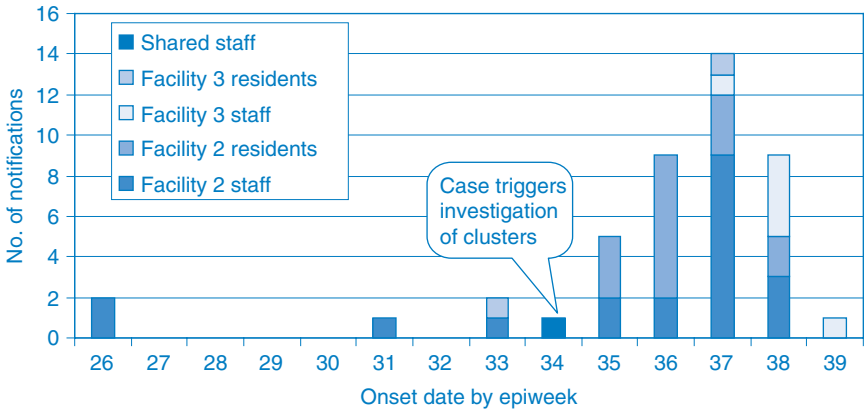

Figure 1. Week of onset of pertussis cases in aged-care facilities 2 and 3 in the former Macquarie Area Health Service (now part of Greater Western Area Health Service) in NSW, 2004. Source: NSW Health Notifiable Disease Database.

is the first reported in an Australian nursing home. In 2005, the United States' Centers for Disease Control and Prevention (CDC) reported outbreaks of pertussis in hospitals in three states.[7]

Importantly, outbreaks of pertussis in residential institutions have also involved staff. In an investigation of pertussis in a residential facility for handicapped people, the epidemiological evidence suggested that the infection was introduced and spread by staff.[8] It is common for staff to work in more than one aged-care facility and this movement facilitates disease transmission. In facility 2 and facility 3, the review of notifications of pertussis and the calculation of their onset dates revealed five cases in these two facilities reported before index case B (Figure 1). Because staff members had pertussis before the index case triggered investigation, it is most likely that the infection was introduced to the facilities by staff. As pertussis is endemic in this area, undiagnosed cases maintain the disease in the community and may be responsible for outbreaks.

The outbreak was controlled by rapid identification and treatment of people with a cough. The implementation of prevention strategies also played an important role in the control of the outbreak. Although it has been reported that prophylactic antibiotics have a role in controlling the extent of pertussis outbreaks, mass prophylactic antibiotics were not used in this situation as they are not recommended by NSW Health guidelines.[9,10] Hospital outbreaks reported by CDC did use prophylactic antibiotics for contacts and, although further cases did not occur, the role of prophylactic antibiotics remains uncertain.[7]

Vaccination is the mainstay of pertussis prevention although one report indicated that the attack rate in immunised residents was the same as for non-immunised residents.[8] Until recently, there has not been a pertussis vaccine recommended for use in adults: normally, immunisation of this age group would have occurred many years earlier in childhood and immunity may have waned. The efficacy of pertussis vaccination in adults during an outbreak has been suggested but has yet to be established.[2,7,8]

These three clusters also underline the limitation of detection using specific IgA alone. IgA may persist for some time after the acute infection and may have delayed the identification of the initial case.[11] However, IgA may also be positive without symptoms and a diagnosis of pertussis depends on IgA positivity in the presence of a clinically compatible illness. In facility 2, a number of asymptomatic staff were tested and found to be positive for IgA.

Conclusion

This investigation has shown that pertussis outbreaks can occur in adults in semi-enclosed communities, such as nursing homes, and that staff can readily transmit the disease. With the present availability of a multivalent vaccine containing pertussis that is suitable for adults, routine vaccination of staff at aged-care facilities should be considered.

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Hospital and non-hospital costs for fall-related injury in community-dwelling older people

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Abstract: Objectives: This study determined the cost of fall-related health care in a cohort of community-dwelling people. **Methods:** 578 people aged 75 years and over were monitored for falls and related health-care costs for one year. **Results:** The mean cost per injurious fall was $1600 (year 2000 dollars). Hospital costs accounted for 67% of the total cost, even though only 4% of injurious falls required hospital admission. The cost of non-hospital health care was also substantial. **Conclusion:** This study highlights the economic burden of falls and the importance of injury prevention strategies.

Falls in older people are common, costly and potentially debilitating. Falls are the leading cause of injury-related hospitalisation and death in people aged 65 years and older, and often result in high economic and social costs.[1,2] Depending on the population under study, between 22 and 60% of fallers suffer injuries from their falls.[3,4] Major injuries including soft tissue damage, head trauma, dislocations and fractures occur in 5 to 15% of all falls in any given year. Furthermore, it has been estimated that 20% of older people who experience a fall sustain injuries that require medical attention.[5,6]

Studies undertaken in Sweden, the United States and the United Kingdom have drawn attention to the significant direct health-care costs required for the treatment of fall-related injury.[7–12] Two recent studies have used aggregated data to examine the current and projected costs of fall injuries in Australia.[13,14] One study examined all injury categories and found that fall injuries were the most costly of any injury mechanism.[13] The second study, which was undertaken on behalf of the Australian government, found that ageing of the Australian population in the next 50 years will have a significant impact on the health system due to the increased number of older people suffering fall-related injuries.[14] The study concluded that prevention strategies will need to deliver a reduction in falls incidence of approximately 66% in order to maintain cost parity with current health system costs. A third Australian study estimated fall-related costs in more detail by assessing costs for 79 older people admitted to acute hospital care due to a fall and then discharged to the community.[15] Community and informal care costs were derived from daily diaries completed by participants in the three-month period following hospital discharge and the results showed that hospital costs accounted for most of the post-fall care costs, but that community and personal costs were also substantial. A larger, subsequent study by the same authors found that more than half of the total fall-related health-care cost was attributed to hospital inpatient care when costs were examined for people attending emergency departments (EDs) in Western Australia over a one-year period.[16] A limitation of these study designs, however, is that they only included subjects who had attended a hospital ED following a fall-related injury, thereby limiting the focus to the small percentage of falls that result in serious injury.

The current study builds upon the previous population-aggregated cost studies by broadening the scope of costs included in the analysis to include non-hospital health care in addition to costs associated with hospital admission for all injurious falls, regardless of severity. We examined the cost and cost components of all individual injurious falls that occurred in a large sample of community-dwelling people aged 75 years and over, over a one-year period. For each fall we ascertained costs related to hospitalisation, ambulance, ED presentation, non-hospital medical and allied health care, pharmaceutical and diagnostic investigation and other out-of-pocket expenses to provide accurate costs of individual falls and the costs of the various health-care components required for their treatment.

Methods

Participants

The study population comprised community members aged 75 years and over who were randomly drawn from a membership database of a private health insurance

company and invited to take part in a randomised controlled falls-prevention trial, conducted in northern Sydney between 1999 and 2002.[17] Exclusion criteria included minimal English, blindness, Parkinson's disease or a Short Portable Mental Status Questionnaire (SPMSQ) score <7. People were also excluded from further participation in the study if, after initial assessment, they were found to have a low falls risk (as measured by the Physiological Profile Assessment), since the aim of the trial was to test an intervention for high-risk fallers.[18] Six hundred and twenty people were enrolled in the study; however, during the study year, 42 people were lost to follow-up due to ill-health, death, leaving the study area or withdrawal of consent. Thus, 578 participants aged between 75 and 98 years (mean = 80.2; SD = 4.4) were included for cost-of-falls analysis. Informed consent was obtained from all participants prior to participation and approval was given by the Human Studies Ethics Committee at the University of New South Wales (approval number CEPIHS 98048).

Falls definitions and data collection

Falls experienced by all participants for a period of 12 months were measured prospectively, using monthly fall calendars. Details of each fall, including the injuries sustained and consequent medical treatment, were collected via telephone interview. Injuries were classified using the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM). The participants returned the calendars to the research team at the end of each month and participants who did not return calendars were contacted via telephone.

Health-care use and cost data

Participants who experienced a fall that resulted in health-care utilisation were contacted via telephone on a weekly basis to collect information relating to ongoing medical, health-care and associated costs. This follow-up continued until the fall-related treatment had ceased (range 1–20 weeks). Information was collected on all aspects of the participants' medical treatment, including hospital costs, outpatient services from medical practitioners and allied health professionals and the cost of pharmaceutical medications and diagnostic investigations. Costs obtained from subjects were total costs and did not take account of Medicare or health fund rebates. The data were collected between the years 1999 and 2002, and the costs calculated using year 2000 fees and charges.

Hospital costs were based on the Diagnosis Related Group (DRG) allocated to each specific fall-related injury, since the authors did not have access to hospital cost information directly from the source. For cases where the length of stay differed from the average length of stay for each DRG, adjustments to the average ward costs were made, i.e. cost = DRG figure × (patient length of stay/DRG average length of stay). Estimates from the NSW Health Services Comparison Data were used to calculate missing costs.[19]

Patients who presented at a hospital ED and were discharged without admission were allocated the average cost of a visit to the ED ($238 for a principal referral hospital and $271 for a major metropolitan hospital), plus additional costs for individual services such as pathology ($170 principal referral and $95 major metropolitan hospital) and imaging ($174 principal referral and $123 major metropolitan hospital), where relevant. These cost estimates were obtained from the NSW Health Services Comparison Data.[19]

The cost of ambulance travel in relation to a fall was also included in the analysis and an average fee for service was used ($143), which was determined through direct contact with the Ambulance Service of NSW.

Costs for services provided by medical practitioners and allied health professionals were obtained from the participants. Where participants were not able to provide specific information, the standard fee-for-service was used. For medical practitioners, standard fees were obtained from the Medicare Benefits Schedule.[20] For allied health professionals, the following amounts were used (obtained from the relevant representative professional organisations): physiotherapy and occupational therapy, $50 initial visit, $40 subsequent visit; home nurse, $50 per hour.

The cost of fall-related pharmaceuticals was ascertained through the Schedule of Pharmaceutical Benefits.[21] The cost of associated diagnostic investigations such as x-rays was obtained directly from the participant or, when this was not possible, from the Medical Benefits Schedule.[20]

Other out-of-pocket expenses included in the analysis were the repair of broken glasses, purchase of non-prescription analgesics, installation of safety rails in the home and purchase of bandages and other items for wound protection. The amounts for these items were obtained directly from the study participants.

Costs that were beyond the scope of the study and therefore not ascertained included indirect costs such as informal care from friends and family, transport-related costs such as taxi fares to and from medical appointments, and participant opportunity costs such as time away from work.

Statistical analysis

Descriptive statistics were used to collate the falls data and compare the incidence of falls and the types of injuries suffered between the men and women in the study group. Average costs were calculated for falls that resulted in

Table 1. The number of falls according to injury status and receipt of medical treatment in study participants between 1999 and 2002 in Sydney, NSW

Fall type	Men		Women		Total	
	(*n* = 190)	(%)	(*n* = 348)	(%)	(*N* = 538)	(%)
Without injury	105	55.3	92	26.4	197	36.7
No injury but had medical check-up	2	1.0	1	0.3	3	0.6
Injury, no medical attention	56	29.5	160	46	216	40.1
Injury, medical attention, excluding hospital	22	11.6	74	21.3	96	17.8
Injury, medical attention, including hospital	5	2.6	21	6	26	4.8

Table 2. The total and adjusted cost of falls injuries that required hospital admission, treatment at an emergency department or other non-hospital services between 1999 and 2002 in Sydney, NSW

Treatment type	Cost ($)				
	Average	Minimum	Maximum	Total (year 2000)	CPI adjusted total* (year 2006)
Hospital admission (*n* = 13)					
In-hospital services#	10 003	1530	26 262	130 041	175 165
Non-hospital services	2328	11	5200	30 269	40 772
Emergency department** (*n* = 13)					
Emergency department services	424	147	1164	5512	7425
Non-emergency department services	756	101	2371	9828	13 238
Other non-hospital services (*n* = 96)	203	10	1759	19 533	26 311
Total for all services				195 183	262 912

#Average length of stay of 19.5 days (range 1–49 days).
*Year 2000 cost multiplied by the cumulative change in the Consumer Price Index (CPI) between the years 2000 and 2006.[22]
**Excludes subjects subsequently admitted to hospital after attendance at an emergency department.

hospital attendance and those that resulted in medical attention without hospital attendance. The costs obtained were then adjusted to reflect year-2006 costs by multiplying them by the cumulative change in the Consumer Price Index (CPI) between the years 2000 and 2006.[22] For this calculation, the figure used was the percentage change of the weighted average of eight Australian capital cities for each year for the CPI health group only. The data were analysed using SPSS 11.5 software.[23]

Results

In the 12-month study period, 48% of the study participants fell one or more times. The total number of falls over this period was 538. In the follow-up year, 303 people (52%) suffered no falls, 145 (25%) suffered one fall and 130 (23%) suffered two or more falls. The proportion of men and women in each fall category was similar ($\chi^2 = 3.39$, 3 d.f., $p = 0.34$).

Table 1 illustrates the number of injurious falls that occurred during the study period and the subsequent type of medical treatment that was sought. More women than men suffered an injury ($\chi^2 = 18.14$, 1 d.f., $p < 0.001$) and suffered falls that required medical treatment ($\chi^2 = 14.29$, 1 d.f., $p < 0.001$).

Of the 538 falls, a total of 458 injuries occurred, including bruising with intact skin surface (39% of the injuries), sprains and strains of joints and muscles (17%) and superficial injuries (15%). Fractures accounted for 5% of the injuries, including two hip fractures. Women were more likely to suffer fall-related fractures than men, with 83% of the fractures occurring in women. Men had a higher rate of open wound injuries, accounting for 33% of all men's injuries compared with 16% in women.

Table 2 presents the average, minimal, maximal, total and year-2006-adjusted costs for falls injuries that required inpatient hospital, ED and non-hospital services. The overall cost of fall-related health care for the study population in the follow-up period was $195 183. Sixty-seven percent of this cost was due to services utilised while in hospital, 3% was due to services utilised while in an ED and the remaining 30% was due to services utilised from non-hospital sources.

Table 3. The total and adjusted cost of falls in study participants according to major injury categories between 1999 and 2002 in Sydney, NSW

Injury category	Cost ($)				
	Average	Minimum	Maximum	Total (year 2000)	CPI adjusted total* (year 2006)
Fracture ($n = 23$)	5719	28	30 867	131 529	177 170
Sprain/strain ($n = 36$)	656	11	11 382	23 601	31 791
Other injuries ($n = 62$)	646	10	13 480	40 053	53 951

*Year 2000 cost multiplied by the cumulative change in the Consumer Price Index (CPI) between the years 2000 and 2006.[22]

The cost of falls for the major injury categories: fractures, sprains and strains, and other injuries are summarised in Table 3. Fractures were the injury type that incurred the greatest medical costs, with the highest overall individual cost of $30 867 being for a hip fracture. Fractures comprised 5% of all the injuries suffered, yet accounted for 67% of the total cost of fall-related injury.

Discussion

In the 12-month study period, 48% of the participants fell one or more times, a rate that would be anticipated for a sample aged 75 years and over, which had excluded people with a low risk of falling.[2,3] The proportion of falls that resulted in injuries (63%) and injuries requiring medical care (23%) are also consistent with previous research assessing similar populations.[2]

The total cost of fall-related treatment was $195 183, which equates to a total of $262 912 using year-2006-adjusted figures. This translates to an average cost of $1600 per person for the 122 participants who sustained falls requiring medical care in the study period. This figure is lower than the average costs reported in Australian studies by Potter-Forbes and Aisbett ($5688), Hall and Hendrie ($4291) and Hendrie et al. ($4619).[13,15,16] The lower cost is most likely due to a greater capture of people who had minor injuries that required treatment only from a medical practitioner or allied health worker: $1600 is therefore likely to represent a more accurate estimate of all injurious falls suffered by older people in community. When the analyses were restricted to participants attending the ED (and possible subsequent hospital admission), the average cost was $6756. This figure is more comparable to the previous studies that used indirect means for estimating non-hospital care or recruited their study population from ED attendees only.[13,15,16]

Only 4% of the falls that resulted in injuries led to admission to hospital. Despite this, hospital costs accounted for 67% of the total cost of injury-related health care. This figure is midway between the findings of Hendrie et al. (53%), Potter-Forbes and Aisbett (55%) and Hall and Hendrie (80%).[13,15,16] Again, when only those requiring ED and hospital care were considered, hospital costs accounted for a similar percentage of total costs (74%) as reported by Hall and Hendrie.[15] These findings confirm that a small minority of falls that result in serious injury are responsible for the bulk of health care costs for this injury mechanism.

The major strengths of the current study were that it was comparatively large, population based and used individual rather than aggregated data. Also, associated costs data were collected prospectively with a rigorous protocol of follow-up interviews until health care utilisation had ceased. This approach is optimal for maximising the recording of all falls and for calculating non-hospital costs, as routine data collection is not undertaken.[24] The findings indicated that non-hospital costs were substantial and accounted for 19% of the costs of falls that required admission to hospital and 64% of the costs of falls requiring treatment from an ED. The overall proportion of costs attributed to non-hospital services for all falls was 30%. These findings, therefore, complement the research undertaken using aggregated data by providing accurate estimates of costs of all sources of health care.

The study also has a number of limitations. Participants were recruited from a private health insurance database and only those classified as 'high risk fallers' were included, so the study population may not be representative of the older population as a whole. It is possible that this could skew the degree and modality of treatment sought for falls injuries. Nevertheless, the findings are similar to recent Australian studies that have assessed costs in general community populations.[13,15,16] A further limitation is that indirect costs were not ascertained. Previous research has shown indirect costs to be quite substantial in relation to fall injury, so the figures from this study do not provide the total extent of the economic burden of falls injuries.[13]

In conclusion, this study provides detailed cost estimates of hospital and non-hospital treatment for fall-related injuries suffered by community-dwelling older people who were at an increased risk of falls. The inclusion of

treatment from non-hospital services provides a broader estimate of the cost of fall-related injury compared with previous Australian studies. This information may assist health-care planners to guide the allocation of funding priorities and provide estimates of falls-injury treatment categories for use in cost-effectiveness studies of falls prevention strategies.

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Incidence of falls and fall-related outcomes among people in aged-care facilities in the Lower Hunter region, NSW

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Abstract: This article describes the rate of falls and adverse outcomes following falls, such as fracture, hospitalisation and death, among residents of 88 aged-care facilities in the Lower Hunter region of NSW from July to December 2005. A high rate of falls was observed with a crude incidence of 171 falls per 1000 beds per month. Around 40% of the falls resulting in hospitalisation were for fractured neck of femur. Estimated 3-month survival following fractured neck of femur was low, with a rate ratio of around 1 : 3. The data indicate an urgent need for falls injury prevention in aged-care facilities.

Injuries resulting from falls, particularly hip fractures, are a major public health issue and will become more so with the ageing of the population. Of all falls resulting in hip fracture, about 35% occur in residents of aged-care residential facilities.[1,2] Each year, the John Hunter Hospital, the major tertiary referral hospital for what was previously the Hunter Area Health Service, admits around 400 patients aged 65 years and over with a fractured neck of femur (R Gibberd, personal communication, February 2006). Our experience suggests that around one-third of these patients are admitted from residential care facilities. One-third of this group comes from hostels and utilises about 4500 bed-days, while the two-thirds that come from nursing homes have much shorter stays and use about 1500 bed-days (Kichu Nair, personal communication, April 2007).

On this basis, we calculate that, at a cost of $500 per bed-day, hip fractures from aged-care facilities in the former Hunter Area Health Service could cost around $3 000 000 each year. The potential savings from the prevention of hip fractures are large in terms of health care resources and cost. Moreover, the incidence of falls can be up to three times higher among residents of aged-care facilities compared to the local community, with rates in aged-care facilities being reported as 1.4 falls per person per year, rising to 6.2 falls per person per year in a psychogeriatric ward.[3] In an Australian study, 625 residents of hostels and nursing homes in three states were followed for two years. Over that period, 1555 falls were reported among 355 of these residents.[4]

The aim of this study was to describe reports of falls and fall-related outcomes among people in residential aged-care facilities in the Lower Hunter region of New South Wales (NSW).

Methods

All aged-care facilities in the former Hunter Area Health Service and the Lower Mid North Coast Cluster of the Hunter New England Area Health Service were invited to participate in this study. Facilities with 20 or more beds were included (facilities with less than 20 beds were considered to be atypical). In June 2005, each facility provided data on the number of beds by type. All beds were assumed to be occupied and the number of beds, assumed to be fixed, was used to determine the denominator in the analyses. An index of the turnover of individuals within this fixed size population was obtained from monthly data on the number of new permanent and respite admissions. Further, the characteristics of participating facilities were determined at a census in January 2006. In this census, each facility provided details of all permanent residents, including their date of birth, sex, whether they were ambulant or not, whether they were residing in a dementia-specific unit or not and resident classification scale (RCS). Each new resident was assigned to one of eight RCS categories designed to reflect their level of need.

Using a standard form, each facility provided monthly aggregate data describing the number of falls (not the number of residents who experienced a fall, as each resident could register multiple falls) and the number of falls reported in residents' charts that resulted in adverse events such as fracture, hospitalisation or death. Each facility designated a senior staff member to supervise the project

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within their facility and to collect the monthly data. The staff member audited residents' charts to identify 'incident records', which report details of fall incidents including date, time, injury, immediate treatment and medical treatment by facility staff. Cumulative data from the monthly falls reports showed the total number of reported falls for that month as well as: the number of falls resulting in fracture; fractured neck of femur; fractured neck of femur within three months of admission; hospitalisation due to a fall-related injury; and the number of deaths occurring within three months of fractured neck of femur. Where a facility failed to provide a monthly falls report, the designated staff member was sent a second form by email or fax then followed up by telephone.

Not all facilities returned forms every month. The rate of falls and related events occurring each month was determined by dividing the number of events by the total number of beds for all facilities that had returned forms for that month. The total number of beds for each month was summed to provide the overall denominator as bed-months of observation.

Results

In total, 98 facilities were identified in the Lower Hunter region. Of these, six were ineligible due to small size, four declined to participate in the study and 88 consented to participate (consent rate of approximately 96% if ineligible facilities are excluded). Characteristics of the facilities are shown in Table 1 and characteristics of the residents are shown in Table 2.

The median and mean number of new permanent admissions each month at each facility was 2 and 2.3 (range: 0–28), and the median and mean number of new respite admissions each month at each facility was 2 (range: 0–10).

Falls data forms were collected and collated for the period July to December 2005. Over this 6-month period, forms were returned by 75–93% of facilities each month, with an overall return rate for the period of 84%. There was no systematic bias in returns according to type of facility. The total bed-months of observation was 28 536.

The trend in the rate of falls and related events for every 1000 beds is shown in Figure 1. The overall crude incidence rate was 171 falls: 2.4 falls with fractured neck of femur; and 2.2 falls with other fracture injury for every 1000 bed-months of observation. The crude overall incidence rate for hospitalisation from a fall was 6 per 1000 bed-months of observation.

The outcomes of the falls are shown in Table 3 where rates are expressed for every 1000 falls. Overall, 3.6% of falls required hospital admission, 1.4% of falls resulted in a fractured neck of femur and a similar proportion resulted in other fracture. For every 14 fractured neck of femur events, 1.8 occurred within the first three months following admission (13%); that is, a rate ratio of 1.8 : 14. The rate of death within three months of a fractured neck of femur was 35% of the rate of falls with fractured neck of femur – that is, a rate ratio of around 1 : 3 – indicating a high mortality rate among residents experiencing fractured hip.

Discussion

These data were collected before an intervention for falls prevention in aged-care facilities was implemented at the

Table 2. Characteristics of the residents in 88 aged-care facilities in the Lower Hunter region of NSW at the time of the census, January 2006 ($N = 5354$)

Age in years: median (range)	85 (27–107)
Female (%)	73
Ambulant* (%)	70
Dementia-specific care (%)	21

*Ambulant defined as anyone who can stand and walk with or without assistance.

Table 1. Distribution and characteristics for low-care, high-care and mixed-care beds in participating resident aged-care facilities in the Lower Hunter region of NSW, 2005–2006

Type of care	Facilities *n*	Median number general beds+		Median number dementia specific beds+		Median number all beds+		Median number new admissions per month*		Median number respite admissions per month*		Median RCS**	
		n	Range	*n*	Range	*n*	Range	*n*	Range	*n*	Range	*n*	Range
Low care	42	40	0–78	0	0–65	40	25–132	1	0–28	2	0–9	5	1–8
High care	24	60	28–136	0	0–96	65	28–216	3	0–12	0	0–9	2	1–7
Mixed (low and high)	22	68	37–130	10	0–46	8	44–160	2	0–17	2	0–10	3	1–8

+Data obtained June 2005.
*Data obtained from monthly reports from facilities.
**Data obtained from a census of residents in January 2006.
RCS: Resident Classification Scale.

beginning of 2006. The data were aggregated in facilities and so are not age specific and include a wide age range. Additionally, the data were reported by the facilities and accuracy, therefore depends on procedures and protocols for incident reporting at each facility. The true number of falls may be higher or lower than reported.

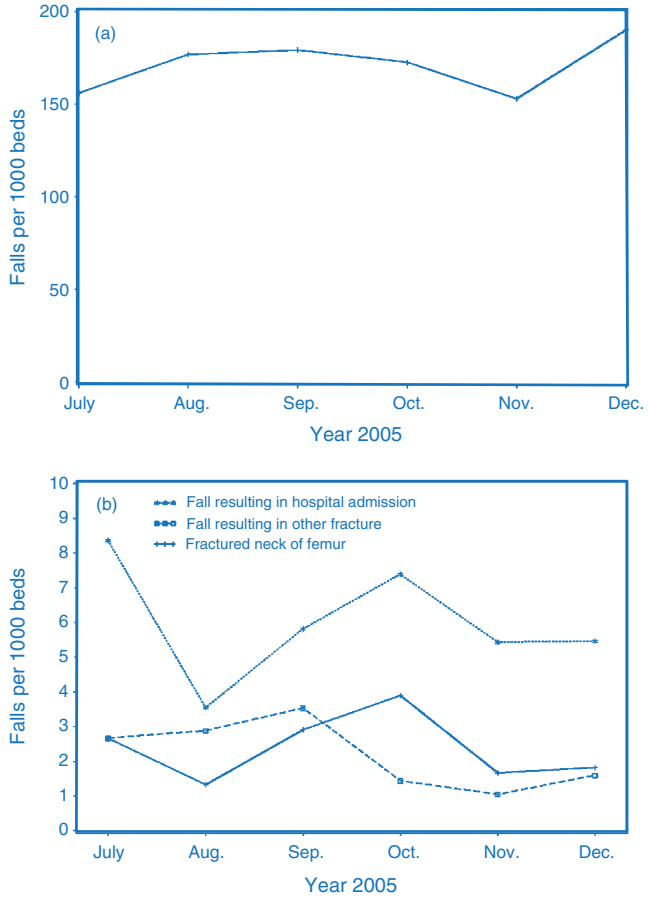

Figure 1. Falls per 1000 beds among residents of aged-care facilities in the Lower Hunter region of NSW for the period July 2005 to December 2005. (a) Rate of total falls. (b) Rate of falls resulting in hospital admission, fractured neck of femur and other fracture.

We assumed that bed numbers remained fixed and that all beds were occupied for the duration of the study. While the assumption of full beds is reasonable, some facilities increased bed numbers during the period, possibly resulting in an overestimation of rates per 1000 beds. Similarly, if falls were under-reported in these data then the denominator for rates per 1000 falls should be larger and the rates may have been overestimated. Furthermore, we observed a decrease in the response rate over time and there was possible under-reporting by facilities where no falls occurred in a month (or by facilities with many falls). Under-reporting would bias both the denominator and the numerator. In contrast, we experienced a high rate of facility enrolment in the study and participation was generally strong (given the limitations of time and resources experienced by these facilities).

Given these limitations, the data demonstrated a rate of 171 falls for every 1000 beds for each month. Extrapolating across a 12-month period, this is equivalent to two falls per bed for every year. This is higher than would be expected in the community; for example, Mackenzie et al. reported a rate of 47 per 1000 people each month.[4,5] The rate of falls observed here in residential settings are comparable to an Australian study by Flicker et al., where a crude rate of 155 per 1000 people every month was observed (calculated from data provided).[6] In addition, a Scandinavian study found a much higher rate of 357 per 1000 person-months among residents with dementia compared with residents of senior citizens apartments (142 per 1000 person-months) and residents of an old people's home (176 per 1000 person-months).[7] Around 3.6% of falls reported in our study resulted in hospital admission. Among those admitted to

Table 3. Monthly total number of beds and falls in residential care facilities and rate of falls outcomes per 1000 falls for July–December 2005 in the Lower Hunter region of NSW

Month	Beds	Falls	Outcomes per 1000 falls					Returns received from facilities contributing data	
	N	*N*	Hospital admission	Fractured neck of femur	Other fracture	Fractured neck of femur within 3 months of admission to facility	Deaths within 3 months of fractured neck of femur	*n*	%
July	5288	830	53	17	17	3.6	3.6	82	93
August	4491	807	21	7.4	17	1.2	6.2	72	82
September	4822	863	32	16	20	1.2	5.8	74	84
October	4898	840	43	23	8.3	1.2	4.8	74	84
November	4786	731	36	11	6.8	0.0	2.7	74	84
December	4251	799	29	10	7.5	3.8	6.3	66	75
Totals	**28 536**	**4870**	–	–	–	–	–	**442**	**84**
Overall monthly rate	–	–	**36**	**14**	**13**	**1.8**	**4.9**		

hospital, around 40% were reported to have a fractured neck of femur, and we observed a high rate of death within three months of fracture. Moreover, we observed that on average over the six months, an estimated 13% of falls that resulted in a fractured neck of femur occurred within three months of admission.

The data revealed a rate of falls among residents of aged-care facilities that can be improved. We intend to work with facilities involved in the study to improve monthly reporting of falls and falls injuries. Additionally, we have enrolled a cohort of around 5000 residents in participating facilities and will be able to observe falls resulting in a fractured neck of femur at an individual level. Cross-checking patient information data for admissions with a fractured neck of femur will allow more accurate reporting of this injury in our routine data collection activities.

The results of this preliminary ecological study indicate a need for falls injury prevention in residential aged-care facilities.

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Environmental health risk assessment of nickel contamination of drinking water in a country town in NSW

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Abstract: Objectives: To assess the health risks associated with consumption of drinking water with elevated nickel concentration in a NSW country town named Sampleton. **Methods:** We used enHealth Guidelines (2002) as our risk assessment tool. Laboratory test results for nickel in water samples were compared with the Australian Drinking Water Guidelines 2004 and the World Health Organization's (WHO) Guidelines for Drinking Water Quality 2005. **Results:** The mean nickel concentration in the drinking water samples tested over a 4-year period (2002–2005) was 0.03 mg/L (95% CI: 0.02–0.04). The average daily consumption of two litres of water by a 70-kg adult provided 0.06 mg (0.03 mg × 2) of nickel, which was only 7% of the lowest observed adverse effect level (LOAEL) based on experiments on nickel-sensitive people in a fasting state. **Conclusions:** The mean nickel concentration in drinking water appears to have no health risks for the inhabitants of Sampleton.

Background and risk identification

Sampleton (not the real name) is a small country town in rural New South Wales (NSW). Until 2001, the inhabitants of the town had been supplied with drinking water sourced from a local surface water catchment. In 2002, the drought prompted the local authority to negotiate with a local colliery to release its extracted underground mining water into the local drinking water catchment. The local government authority (LGA) treated the water before supplying it as drinking water to its residents.

Between 2002 and 2005, the water samples at Sampleton recorded on the NSW Health Drinking Water Database intermittently exceeded the Australian Drinking Water Guidelines (ADWG) value for nickel of 0.02 mg/L.[1,2] The aim of this risk assessment was to assess the potential health risks associated with the consumption of drinking water with an elevated nickel concentration.

Risk assessment methodology

We used the enHealth Guidelines for Assessing Human Health Risks from Environmental Hazards (2002) as the risk assessment tool.[3] The ADWG and the WHO guidelines for nickel were used for the specific guidelines on nickel levels in drinking water.[2,4] Chemical analysis of water samples were undertaken by a laboratory accredited by the National Association of Testing Authorities (NATA).[5] Laboratory test results for nickel in the water samples were compared with the ADWG and the WHO guidelines.

Hazard assessment

Hazard identification

Ground water can contain dissolved metals including nickel (Ni) and chemicals naturally released from rock and soil, which can be harmful to humans.[6] The estimated average daily dietary intake of nickel is between 0.1 mg/day and 0.3 mg/day.[7,8] The intake of nickel from food is estimated to be less than 0.2 mg/day. Drinking water generally contributes 5–25 μg of nickel per day, which is approximately 2–11% of the total daily oral intake of nickel.[4] In Australia, the concentration of nickel in typical drinking water is less than 0.01 mg/L with the highest allowable value of 0.02 mg/L.[9]

Non-occupational sources of nickel exposure include food, air and water, but the amount of nickel found is usually much smaller than that typically found in occupational settings.[10] The primary source of nickel in drinking water is from metal pipes and fittings in contact with drinking water. Nickel concentrations in ground water are influenced by soil type, pH level and sampling depth.[4] Higher concentrations have been reported where drinking water is contaminated with nickel waste discharge from chemical, industrial or mining plants.[9]

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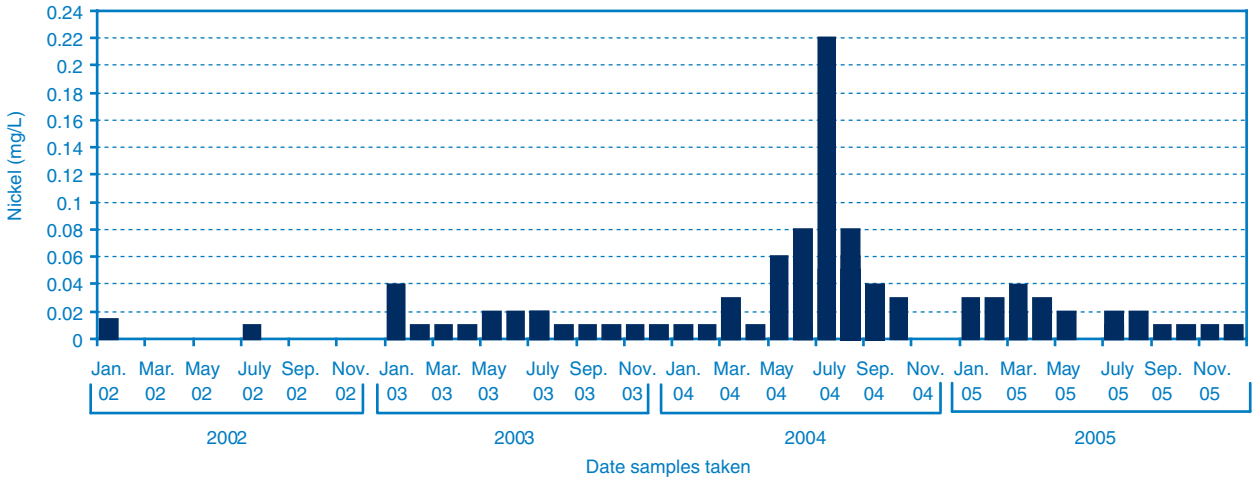

Figure 1. Concentration of nickel in samples of drinking water in Sampleton, NSW, 2002–2005.

The adverse health effects of nickel for humans depend upon the route of administration, water solubility (absorption) of nickel compounds, dose, bodyweight, sensitivity and duration of exposure.[4,11–13] Dermal exposure is the commonest cause of skin irritation to those allergic to nickel, with more females than males being affected.[14] The main adverse effect for this risk assessment is nickel allergic dermatitis, as it occurs at very low levels of exposure. The Expert Group on Vitamins and Minerals estimated that approximately 7–10% of the population in the United Kingdom, predominantly women, have this condition.[15] Allergic contact dermatitis due to nickel sensitivity increases with age and may affect as much as 4–5% of the paediatric population.[16–18] While for some people the reactions are limited to a minor skin irritation, for certain sensitised people the exposure to elevated nickel may cause or aggravate dermatitis. In about half of the sensitive people with vesicular hand eczema, the reactions can be very severe and can lead to loss of working ability.[15]

Dose–response assessment

Assessing the dose–response relationship in terms of nickel sensitivity is complex. Christensen and Lagesson observed wide variations in nickel concentrations in blood and nickel excretion in urine in healthy humans when equal amounts of nickel were ingested.[7] Such variation in sensitivity makes it difficult to estimate the true dose–response effect because a very small exposure to nickel may trigger a rapid response in some people due to their high level of sensitivity. The lowest observed adverse effect level (LOAEL) for an oral dose is reported to be 0.05 mg/kg bodyweight per day when skin is not sensitised.[8] But when the skin is sensitised, an oral intake of 0.012 mg/kg bodyweight per day may provoke contact dermatitis.[13]

Exposure assessment

In Sampleton, the mean nickel concentration in drinking water found in water samples taken between January 2002 and December 2005 was 0.03 mg/L (95% CI: 0.02–0.04) (Figure 1).

The high concentration of nickel appears to be a result of the introduction of mine water into the drinking water catchment and the reduction of the natural flow rate within the catchment due to the drought. The lowered flow rate in the catchment due to drought conditions was the main reason that the mine water was accessed to supplement the drinking water supply. The changes in nickel concentrations over the 3-year period could be attributed to changes in natural dilution and the level of demand of water sourced from the colliery to meet the supply requirements.[19]

Risk characterisation

The human health risk of nickel contamination of drinking water in Sampleton has been characterised in consideration of the following two guidelines:

The Australian Drinking Water Guidelines

The ADWG sets a safety standard value for concentration of nickel in drinking water.[2] The guideline value was derived as follows:

$$\frac{5\ \text{mg/kg bodyweight per day} \times 70\ \text{kg} \times 0.1}{2\ \text{L/day} \times 1000} = 0.02\ \text{mg/L}$$

where:

- 5 mg/kg bodyweight per day is the lowest observed adverse effect level (LOAEL) for altered organ-to-bodyweight ratios based on animal studies[20]
- 70 kg is the average bodyweight of an adult
- 0.1 is the proportion of total daily intake attributable to the consumption of water
- 2 L/day is the average amount of water consumed by an adult
- 1000 is the safety factor used for the uncertainty over applying animal studies to humans. In this case, the

safety value is applied as follows: 10 for interspecies variations, 10 for intraspecies variations and 10 to compensate for the lack of adequate studies on chronic effects and for increased intestinal absorption when taken on an empty stomach.[2]

The WHO guidelines

The WHO guidelines for drinking-water quality, based on human challenge studies done by Nielsen et al. (1999), recommend the safe nickel value in drinking water as 0.07 mg/L, which was calculated as follows:[4,13]

$$\frac{0.012 \text{ mg/kg bodyweight} \times 60 \text{ kg} \times 0.2}{2 \text{ L/day}} = 0.07 \text{ mg/L}$$

where:

- 0.012 mg/kg bodyweight, derived from a LOAEL based on human challenge studies[13]
- 60 kg is the average weight of an adult
- 0.2 is the proportion of total daily intake (TDI) of nickel from drinking water
- 2 L/day is the average amount of water consumed by an adult.

The WHO guidelines' value for nickel in drinking water is derived from the LOAEL of 0.012 mg/kg of bodyweight based on experiments in fasting adults with single doses on empty stomach.[13] Because this LOAEL of 0.012 mg/kg bodyweight was based on a highly sensitive individuals, WHO did not include an uncertainty factor (intraspecies or interspecies variations) to derive the TDI.[4] Using the LOAEL of 0.012 mg/kg bodyweight, the LOAEL for a 70-kg adult would be 0.84 mg/L per day (0.012 mg/kg bodyweight × 70 kg).

To our knowledge, there are no data available on the LOAEL for nickel consumption in children. However, it is likely that the LOAEL for children would be greater than the adult LOAEL of 0.012 mg/kg bodyweight as this value was calculated on a highly nickel-sensitive adult population.[13] Children are less likely to have been sensitised to nickel as nickel sensitivity increases with age.[16–18] The average intake of 0.03 mg nickel, assuming consumption of one litre of water each day and using the adult LOAEL of 0.012 mg/kg bodyweight would result in a childhood LOAEL of 0.16 mg/day (0.012 mg/kg × 13 kg) for a 13 kg child.[2]

The mean nickel content (0.03 mg/L) in the drinking water in Sampleton was one and a half times higher than the ADWG value but accounts for only 43% of the WHO guidelines value. The higher nickel level in Sampleton water was intermittent, with one-third of the total water samples tested exceeding the ADWG value over a 4-year period. The ADWG is based on animal studies that, when applied to humans, provide only persuasive rather than hard evidence for effects on humans.[2,20] The animal data may also not be sufficiently protective of people sensitised to nickel. The WHO guidelines value (0.07 mg/L), on the other hand, is the maximum effect value based upon experiments on nickel-sensitive people at a fasting state. Assuming a 70-kg adult drinks 2 L of water per day, the average daily intake of 0.06 mg (0.03 mg × 2 L) of nickel from drinking water in Sampleton was approximately 7% of the LOAEL of 0.84 mg (0.012 × 70 kg) based on experiments on nickel-sensitive adults at fasting state.[13] In children, it is difficult to provide a meaningful calculation of the risk as childhood bodyweights and consumption of drinking water are highly variable. However, assuming consumption of one litre of water per day, the average daily intake of 0.03 mg of nickel from drinking water in Sampleton was approximately 19% of the LOAEL of 0.16 mg/day (0.012 mg/kg × 13 kg) estimated for a 2-year-old child weighing 13 kg.

Assuming that the dietary intake of nickel in the Sampleton population is no different from that in the Australian population, and that the residents of Sampleton are all nickel-sensitive (which is unlikely), the mean nickel concentration of 0.03 mg/L (95% CI: 0.02–0.04) appears to have no health risks for the inhabitants of Sampleton.

The single high reading in the 4-year period of 0.22 mg/L in July 2004 (Figure 1) could have been due to a sampling error or measurement (laboratory) error. The particular sample was taken from the same outlet as all the other samples over the sampling period, so it is unlikely that there was an increase in nickel level due to other contamination sources such as plumbing. However, the nickel value of 0.22 mg/L at the daily intake rate of 2 L a day of drinking water for a 70-kg adult would still be only 52% of the LOAEL of 0.84 mg/day (0.012 × 70 kg) for adults based on studies of nickel-sensitive people.[13] For children, the observed nickel value of 0.22 mg/L at the daily intake of 1 L a day for a 13 kg child would be 138% of the childhood LOAEL of 0.16 mg/day (0.012 mg/kg bodyweight × 13 kg).[9] However, the LOAEL for highly-sensitive adults used in this calculation provides a safety factor for young children as they are less likely to be highly sensitised to nickel. Therefore, it is unlikely that this level of nickel in drinking water would have a significant effect on the younger population.

Risk management

Although the risk assessment found no obvious threat to the health of Sampleton residents, the LGA has been informed of the need to continue to monitor the water supply for nickel levels. The Council was advised that, under the NSW Health Drinking Water Monitoring Program, monitoring of the quality of drinking water should rotate between designated sample sites throughout the distribution system and over time.[1]

Conclusion

An enhanced surveillance of the chemical concentrations in the town water supply system has been recommended while mine water is being directed to the drinking water catchment. The use of alternative sources to supplement drinking water supplies during drought conditions, such as mine water in this case, may become more common in rural and regional towns as the drought conditions and water supply levels continue to fluctuate. While this risk assessment provides some reassurance that small increases in nickel in this town water supply are not a hazard to human health, the study does highlight the need for continued vigilance in relation to water quality when water scarcity forces supply authorities to choose alternative sources.

Acknowledgment

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Managing environmental lead in Broken Hill: a public health success

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Abstract: Objectives: To describe locality-specific changes in blood lead levels of 1–4-year-old children in Broken Hill, NSW between 1991 and 2007. **Methods:** Annual age-sex standardised mean blood lead levels, blood lead screening clinic attendance rates and lead-dust levels for five lead-risk zones were calculated from routinely collected data. **Results:** Blood lead levels were similar in all localities in 2002, 2003, 2005 and 2006, after having been consistently higher in localities with highest environmental lead since 1991. **Conclusions:** Combining health promotion with a targeted clean-up has reduced the effect of locality on blood lead levels. Results are consistent with reduced contamination due to effective soil stabilisation and storm-water control.

"For whoever has, to him more shall be given; and whoever does not have, even what he has shall be taken away from him" is an apt description of many health programs, in that while everyone benefits, those who have least need of the program tend to benefit most, and those who need the program most, often do not benefit at all.[1] This paper, however, describes the outcomes of a program for which this has not been the case. We describe the trend for locality to have a reduced impact on blood lead levels of young children living in Broken Hill, New South Wales (NSW), first observed in 2002.[2]

Lead has been mined in Broken Hill since 1884. Lead poisoning was evident among the early miners and their families, but was seen as mainly an occupational health problem.[3] This was reinforced by a survey of school-aged children in 1982, which found all had blood lead levels below 40 μg/dL, the then level of concern in Australia.[4,5] However, a subsequent study of apparently healthy Broken Hill dogs found blood lead levels similar to levels found in dogs in the town of Port Pirie, South Australia, which has an active lead smelter.[6] Local concern was further increased in Broken Hill by the recommissioning of open-pit mining in the centre of town, a drought in the late 1980s and the birth of three babies with delayed visual maturation (usually caused by exposure to high lead levels *in utero*) between 1988 and 1990.[7] A survey of 1–4-year-old Broken Hill children in 1991 found 86% had blood lead levels of 10 μg/dL or above (the current level of concern with regard to health effects) and 38% had very high lead levels of 20 μg/dL or above.[2]

In 1994, a state government-funded lead management program was established to address this situation. The program (described elsewhere) comprised health promotion, case finding and management, and remediation of contaminated public land.[2] It was underpinned by an active research and evaluation program. Extensive land remediation work in the highest lead-risk zones was largely completed by 1997, with final works undertaken in 2003 and 2004. Mining leases adjacent to industrial and public land, including footpaths and vacant blocks, were comprehensively targeted as were some residential blocks considered to pose a hazard; all were within two streets of the mines. Land remediation mainly consisted of covering contaminated soil with an appropriate material (clean soil, clay, mulch, concrete, crushed metal). Where necessary, work was also carried out to prevent storm-water or vehicles from disturbing remediation work. Hardy local native shrubs and grasses were planted in some areas to further stabilise soil and railway trucks transporting lead concentrate were covered.

Since 1991, all 1–4-year-old children in Broken Hill have been offered at least annual blood lead screening. Screening is voluntary; a combination of reminder letters, promotions and advertising in the local media is used to encourage attendance at the lead screening clinic for at least one blood lead test each year.

Methods

Blood samples are collected for screening at a single laboratory. Trained nurses collect samples by venipuncture

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according to the standard procedure.[8] Samples are stored at 4°C and transported overnight to Adelaide by air for testing the next day. Analysis by electro-thermal atomisation atomic absorption spectrometry is undertaken by the Adelaide Women's and Children's Hospital laboratory.

The number of 1–4-year-old children attending screening each year between 1991 and 2007, their ages, blood lead levels and addresses were obtained from the records of the blood lead screening program. Where children had multiple tests in a year, only the results of the first test were used to avoid regression to the mean. Address was coded into one of five previously described lead-risk zones, based on lead levels in soil (surveyed in 1992) and indoor dust (surveyed in 1995).[2] Geometric mean soil lead level for the five lead-risk zones (ordered highest to lowest risk) is 1967, 794, 621, 365 and 262 ppm, respectively; geometric mean indoor dust deposition is 946, 717, 490, 216 and 201 mg/m^2 per 30 days respectively.[2]

Geometric mean blood lead level (measured as micrograms (μg) lead per decilitre – 100 mL – of blood) was calculated for each lead-risk zone in each year. For comparative purposes these levels were directly age-sex standardised to the 2001 population of 1–4-year-old children. The percentage of children attending for screening was estimated for each lead-risk zone in each year, with estimates of the total eligible population within each zone based on 1991, 1996, 2001 and 2006 census data.[9–12]

Between 1991 and 1999 (after which it was discontinued), a network of dust deposition gauges was maintained in Broken Hill to measure lead flux and concentration. These were constructed and located according to the Australian standard.[13] The gauges consist of a 150-mm-diameter glass funnel inserted through a rubber stopper into a glass bottle of at least 4-L capacity. Gauges are located on stands so that the top of the funnel is approximately 2 m above ground height and placed in clear areas, away from trees and buildings. The contents of the gauges were collected monthly and all gauges were visited within a few days of each other. The contents were analysed for lead content according to the standard procedure by the Mineral Resources Development Laboratory at Lidcombe, NSW.[8] For the 17 gauges with data for the period 1991 to 1999, geometric mean total lead (i.e. water-soluble plus acid-soluble lead reported as mg lead per square metre per month) was calculated for each gauge for each of two periods, 1991–1994 and 1995–1999. Changes in deposited lead across time were assessed by linear regression of yearly averages of $\log_{10}$ transformed total lead against year.

Results

Between 1991 and 2007, age–sex standardised geometric mean blood lead levels among 1–4-year-olds declined by 65%, from 16.3 μg/dL in 1991 to 5.8 μg/dL in 2007. Mean blood lead levels of children living in the highest risk zone decreased by 70%, from 27.3 μg/dL in 1991 to 8.3 μg/dL in 2007. Similar declines were experienced in all areas of town until 2001. The average blood lead level of children living in the highest risk zone dropped markedly in 2002, and was similar to that of children living in other areas of town in 2002, 2003, 2005 and 2006, after having been consistently at least 50% higher (Figure 1). Mean blood lead levels for children living in the highest lead-risk zone spiked in 2004 and again in 2007.

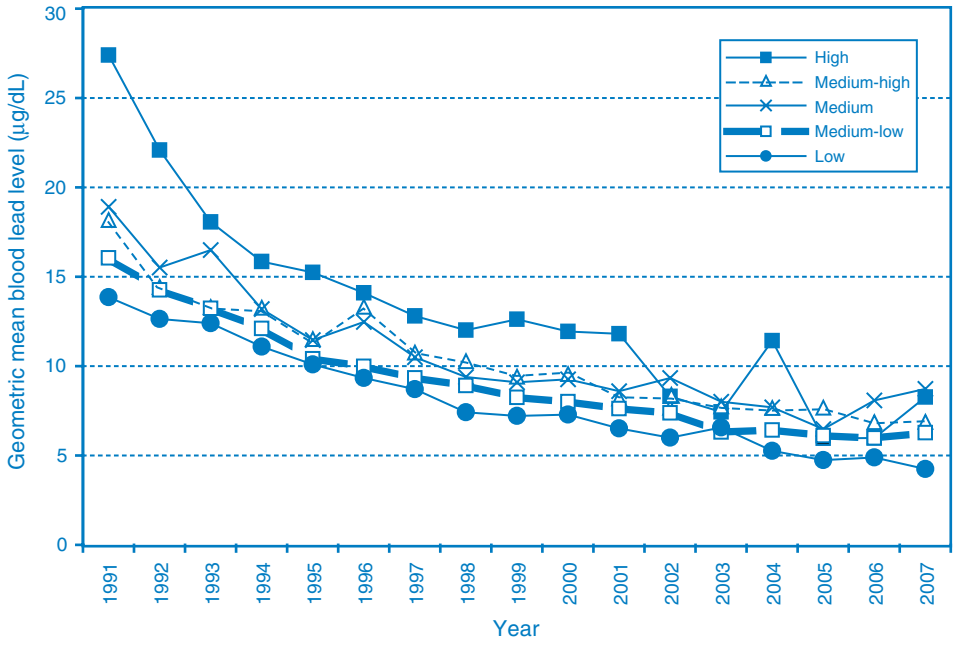

Figure 1. Age–sex standardised geometric mean blood lead levels* for 1–4-year-old children living in different lead-risk districts of Broken Hill, NSW, 1991–2007. *If a child had multiple blood tests in a year, only the first was used. **Risk areas based on lead levels in soil and dust.[2]**

Table 1. Estimated annual participation of 1–4-year-old children in the blood lead screening program in Broken Hill, NSW, 1991–2007, by lead-risk zone

Year	Number and estimated percent of children											
	High risk*		High-medium risk		Medium risk		Medium-low risk		Low risk		Overall risk	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
1991	54	69	103	67	105	52	194	48	324	50	780	52
1992	51	69	99	68	103	54	171	43	304	48	728	50
1993	45	65	76	56	78	43	131	33	207	34	537	39
1994	67	104	108	86	128	77	233	61	413	71	949	72
1995	46	79	87	77	88	58	216	59	343	63	780	63
1996	40	70	71	65	67	47	148	43	242	48	568	49
1997	53	96	91	86	81	59	179	51	328	64	732	63
1998	56	108	116	117	101	79	199	58	341	69	813	73
1999	60	124	90	97	88	72	182	54	313	64	733	67
2000	48	104	65	56	79	56	158	49	281	62	631	59
2001	45	102	73	62	74	49	142	44	280	65	614	58
2002	50	111	79	72	57	39	138	45	257	60	581	56
2003	44	94	68	67	69	49	98	33	214	50	493	49
2004	39	80	61	64	61	44	132	48	201	46	494	50
2005	33	63	71	82	58	44	121	46	228	53	511	53
2006	27	48	56	70	50	38	106	43	192	44	431	46
2007	30	54	65	81	74	57	89	36	175	40	433	46

*Note the estimated number of children living in the highest risk zones is small; this results in imprecise estimation of participation rates and in some years the number of children attending is higher than the estimated resident population.

Overall community participation in the blood lead screening program declined from 72% in 1994, when there was a major door-knock campaign to recruit children for testing, to 46% in both 2006 and 2007. Participation remained higher in the highest risk zone (Table 1) but the difference in participation with other zones diminished in 2006 and 2007.

Data from the environmental dust gauges show that, compared with 1991–1994, deposited lead was lower in most gauges during 1995–1999. However, statistically significant reductions only occurred in the three highest risk zones. The three most dusty sites, all of which were in the highest risk zones, showed highly significant reductions in deposited lead, especially after 1997 (Figure 2).

Discussion

The variable contribution attributed to where a child lives in Broken Hill on their blood lead level has decreased considerably in recent years. Between 1991 and 1993, the average annual difference between highest and lowest locality-specific blood lead level was 9.55 μg/dL; this difference was reduced to 4.84 μg/dL during 1994–2001 and to 2.61 μg/dL for 2002–2007 (Figure 1).

Children living in the highest risk zone experienced a sudden reduction in blood lead levels in 2002 that has persisted for four out of six years. The reason for the spikes in blood leads level in 2004 and 2007 is unclear against a background of variable but declining blood lead screening rates. Less than 60 children live in the highest lead-risk zone, which results in some imprecision in estimates for children living in that area.[9–12] The spikes may be an artefact of the standardisation process: in both 2004 and 2007, 1- and 2-year-old boys and 4-year-old girls living in the highest risk zone had higher mean blood lead levels than in adjacent years and in other age–sex specific groups; these were also the age and sex groups in that risk zone that had the greatest number of children in the standard population.

The recent differential reduction in blood lead levels observed in children living in the highest lead-risk zone is unlikely to be attributable to changes in screening. Screening participation has declined similarly in all areas, although attendance has remained highest in the higher risk zones, a feature that is consistent with the literature.[14,15]

The decline in blood lead levels for all children in Broken Hill, and particularly those living in the highest risk zone, is consistent with reduced levels of environmental risk. Due to their location, children living closest to the mines would be most likely to benefit from the extensive remediation of contaminated land, the majority of which occurred on the mines and immediately adjacent land and all of which occurred within two streets either side of the mines. Data from the dust deposit gauges (Figure 2) support the

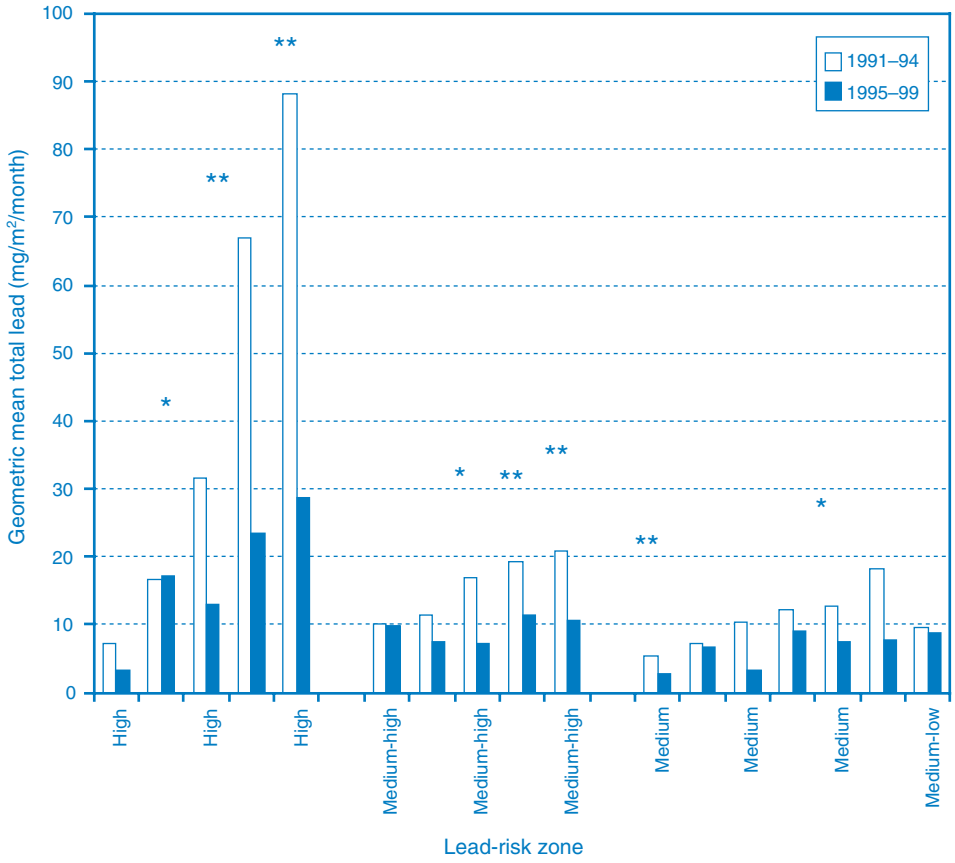

Figure 2. Geometric mean total lead deposition in Broken Hill, NSW, 1991–1999. *Significant at 0.05. **Significant at 0.01.

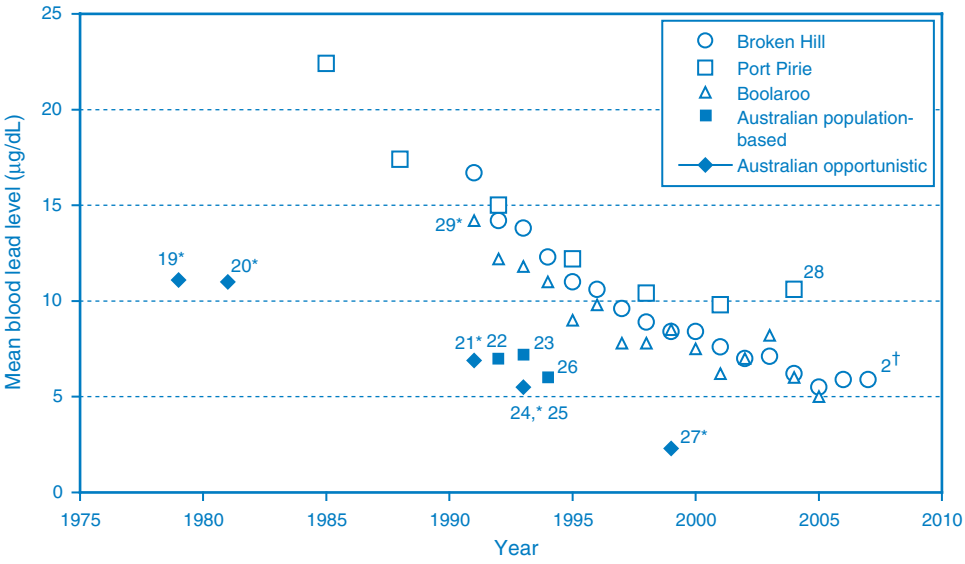

Figure 3. Mean blood lead level of children living in Broken Hill, NSW, compared with other Australian locations. *Indicates arithmetic mean; otherwise geometric. †Data for Broken Hill 1991–2003 from Lyle et al., 2006; data for 2004–2007 from extended analysis by one of the current authors.

hypothesis that areas closest to the mines experienced the greatest reduction in lead deposition. Sudden and persistent declines in blood lead levels after removal of a source of contamination have been reported from other locations within Australia and overseas.[14,16,17] In communities where environmental contamination is widespread, zonal remediation is more likely to be effective than intervening in individual homes.[17,18]

The importance of removing environmental risks is highlighted by Figure 3.[2,19–29] In Australia, lead levels in petrol were lowered in the early 1980s and further reduced in the

1990s. A number of studies indicate that blood lead levels have reduced markedly in the broader Australian community since the late 1970s.[19–27] While these studies were cross-sectional and did not include a remediation assessment component, they provide a benchmark for blood lead levels in young children living outside of point source communities.

While blood lead levels are still substantially above that of the general population, the declines experienced by the three point-source communities of Broken Hill, Port Pirie and Boolaroo, NSW (which had an active lead smelter until 2003) can be attributed to a combination of active local public health programs and broader community-wide action, including reduced lead in petrol and improved control of emissions from mining and smelting.[29] All three communities experienced similar declines until the late 1990s. Since then, levels in Port Pirie have plateaued at about 10 μg/dL but levels in Broken Hill have continued to decline, probably reflecting the impact of different sources and pathways for lead getting into the environment (an ongoing smelting operation versus contamination from waste dumps and historical smelting). Blood lead levels dropped substantially in Boolaroo after the smelter was closed in 2003.[29]

Thus, after nearly a decade of concerted public health action, we appear to have finally minimised the differential impact of locality on blood lead levels in Broken Hill. Active lead management is still required in the community, and the lead management program is currently being reviewed.

Because of the multifaceted approach taken – a combination of large scale land remediation, intensive case management, including home remediation and health promotion – and the limitations of the service-based data set, including a lack of environmental dust data after 1999, it is not possible to separate the effects of the various components of the program. However, the literature suggests zonal remediation is more likely to be effective in reducing blood lead levels than other interventions.[14,16–18] The most likely explanation for the important and sustained reduction in blood lead levels among children living in the highest risk zone of Broken Hill is a mitigation of environmental risk by remediation of contaminated land. While we acknowledge that this analysis of available data does not provide definitive evidence of effect, the observation is noteworthy and consistent with the literature.

Combining whole-of-community approaches to public health action with targeted reduction of environmental risks may similarly benefit other communities.

Acknowledgments

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Addressing decreasing blood lead screening rates in young children in Broken Hill, NSW

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Abstract: This paper outlines the findings of a review commissioned in response to concerns about declining attendance of young children for blood lead screening in Broken Hill, NSW. A review of the literature and feedback from the local community revealed that reasons for reduced screening attendance elsewhere can be applied here, but that any proposed response should take account of local conditions.

Redressing the declining attendance for blood lead screening will require more than raising community awareness, educating carers and enhancing the acceptability of the screening service. A whole-of-community approach to environmental lead management that goes beyond the specific responsibilities of the health sector is required.

Broken Hill is a mining town of over 19 000 people located in far west New South Wales (NSW). The town is built around one of the world's largest silver-lead-zinc ore bodies.[1] Since 1991, children under the age of 5 years have been offered annual screening to assess the level of lead in their blood as part of an environmental lead management program.[2–4] Blood lead screening is the only reliable way to identify children with high blood lead levels so that preventive measures can be taken to reduce the detrimental health effects of lead.[5,6]

The Broken Hill Lead Management Program initially operated out of a dedicated centre, but in 2001 the Program was integrated into mainstream health and other community-based services. The local child and family health centre is now responsible for delivering the Lead Health Program, which involves blood lead monitoring; case finding and management; and community education.

The local area health service was concerned about declining levels of attendance at blood lead screening. The decreased attendance rate was occurring in the context of a program that had been successful in reducing mean blood lead levels, but where around one-quarter of the 451 children tested in 2007 still had elevated blood lead levels ($\geq$10 μg/dL).[7]

We report on a review commissioned to determine why blood lead screening attendance in Broken Hill has declined and propose actions to increase screening rates.

Review methods

We analysed routinely collected data from the Lead Health Program and reviewed the literature to document factors associated with screening attendance.[8,9] We used focus groups to consult with 48 community members who responded to invitations through notices placed in public areas and newspapers. We also sought feedback, using semi-structured interviews, from a range of health professionals and organisations about ways in which their services might contribute to blood lead screening attendance rates. Health promotion models and theories were used to inform the interviews, which explored individual, organisational, social and political factors that may contribute to screening attendance.[10,11]

Key findings

Analysis of routinely collected data showed that attendance rates for screening of pre-school children in Broken Hill have declined significantly from 73% of eligible children in 1998 to 48% in 2007 and that parents are now waiting longer before bringing children in for their first screening test.[8] Screening rates were higher in the youngest children; those living closer to the mines; boys; and the Aboriginal community.[8]

We identified a number of potential factors in the published literature that explain reduced screening attendance, including: the perception of not being at risk and previous low blood lead levels; concerns about the screening

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procedure, especially if the test is invasive or causes discomfort; a previous (self or friend) negative experience; fear of the outcome; and beliefs that nothing can be done to treat the problem. Practical issues can also have a negative impact on attendance, such as: difficulty accessing the service; forgetting that the test is due; and being too busy to attend.[9] Factors associated with higher screening attendance rates include the use of targeted outreach activities, such as screening children in their homes; intensive screening during short periods; and personalised invitations to screening with definite appointment times. People who have attended screening before are more likely to return for the next test and encouragement by general practitioners and health service providers can positively influence screening rates.[9]

Community consultations and interviews with health professionals revealed that many of the reasons for attending or not attending screening reported in the literature can be applied to Broken Hill. Some important issues, however, appeared to be unique to the local conditions, namely the observed decrease in activities to promote lead awareness and cessation of financial support for families to remove lead dust from their homes. Reactions to these observations ranged from an interpretation that environmental lead is apparently no longer a problem in Broken Hill, to an expectation that, as in the past, the government and the mining industry should take responsibility for preventive measures that are beyond the capacity of an individual family.

Discussion

Reversing the trend of decreasing lead screening rates in pre-school children requires raising community awareness, educating parents and improving accessibility to the screening program. Each of these approaches needs to be incorporated into an effective response strategy. The strategy should also take account of local conditions. A broader approach to increasing blood lead screening rates must be taken, and environmental lead management in Broken Hill must go beyond the specific responsibilities of the health sector. This approach needs to take into account prevailing community views that lead management is no longer a priority issue and that the previous commitment by public agencies to invest in solving the problem has diminished.

Successful lead management programs from Australia and overseas have incorporated contributions from the health sector, local and state government, industry and community groups.[12–16] Such an approach would enable effective interventions to be applied at the individual, neighbourhood and community level, including industry practices in relation to mining and transport of lead ore.

It is important that the Broken Hill community is kept informed about changes to the Lead Health Program and that community members are engaged as participants in the strategy, as it evolves. This inclusive approach would help reverse perceptions that environmental lead is no longer a problem or that nothing more can be done by individual families to address it – two local factors reported as barriers to screening.

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Chronic and infectious diseases in Aboriginal and Torres Strait Islander peoples

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Australia is a rich country with high living standards and a world-class health system. According to the United Nations Development Program, Australia is ranked third in the world in terms of human development and fifth in the world in terms of life expectancy.[1] However, the benefits of high living standards are not evenly distributed to all Australians. Current life expectancy for Aboriginal Australians is 59 years for men and 65 years for women, 17 years less than non-Aboriginal Australians. Compared to the life expectancy of populations in countries around the world, the life expectancy of Aboriginal Australians ranks 123rd (after Bangladesh and Bolivia).[1] Furthermore, the gap in life expectancy between Aboriginal and non-Aboriginal Australians is larger than the gap between indigenous and non-indigenous peoples in similarly developed countries such as the United States, Canada and New Zealand.[2]

According to data from the Australian Bureau of Statistics for Queensland, South Australia, Western Australia and the Northern Territory, the five most common causes of death for Aboriginal Australians between 1999 and 2003 were: diseases of the circulatory system; external causes (injury and self-harm); neoplasms; respiratory diseases; diabetes and chronic kidney disease. Although these are also causes of death for other Australians, Aboriginal Australians are four times more likely to die as a result of diabetes than non-Aboriginal Australians and twice as likely to die of external causes and chronic kidney disease.[3]

Not only do Aboriginal Australians die younger, they also become ill much more often than non-Aboriginal Australians. In fact, hospitalisation rates for Aboriginal Australians are twice that for non-Aboriginal Australians. Aboriginal Australians are hospitalised for care involving dialysis (for the treatment of chronic kidney disease) at 12 times the rate of non-Aboriginal Australians. In addition, Aboriginal Australians are hospitalised for endocrine, skin and respiratory conditions, external causes, circulatory diseases, infectious and parasitic diseases, mental and behavioural causes and complications of pregnancy at between 1.5 and 2.9 times the rate of non-Aboriginal Australians.[3] Note that hospitalisation data have limitations as measures of morbidity: disparities between morbidity in Aboriginal and non-Aboriginal Australians are likely to be greater than indicated by these data. The premature mortality and higher morbidity in Aboriginal Australians are mainly due to chronic diseases primarily attributable to social, economic and educational disadvantage, with associated higher prevalence of negative health-related behaviours.

Addressing the health needs of Aboriginal peoples

Aboriginal Community Controlled Health Services operating in New South Wales (NSW) have developed best practice models for delivering health services to Aboriginal peoples.

The Aboriginal Health and Medical Research Council of NSW (AH&MRC) is the recognised peak body and voice of Aboriginal communities on health matters for over 50 Aboriginal Community Controlled Health Organisations in NSW. The AH&MRC represents and supports its member Aboriginal community controlled health services, develops and delivers public health programs, provides accredited training through its Aboriginal Health College and runs a human research ethics committee registered with the National Health and Medical Research Council.

Important principles of good practice for delivering health services to Aboriginal people advocated by the AH&MRC are: community control and engagement; a focus on capacity building; an integrated, coordinated approach; using evidence; and forming partnerships. Recent public health projects undertaken by the AH&MRC include: research and resource development for sexually transmitted infections and bloodborne viruses; policy work on drug and alcohol harm minimisation; chronic disease workshops focused on promoting adult health checks; developing an ongoing chronic disease program; exploring and responding to gambling issues; and convening a national lupus workshop.

Maari Ma is an Aboriginal Community Controlled Health Service that manages all health services in the western third of the Greater Western Area Health Service (not

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including Broken Hill). In order to address the high prevalence of chronic disease, the service has developed a chronic disease strategy that identifies essential elements needed to deliver high quality chronic disease care including: evidence-based practice; system and organisational approaches to care; record and recall systems; communication and cultural skills; multidisciplinary approaches to clinical care; and population approaches (for example, across the lifespan and addressing the social determinants of health). The three key activities of the strategy are: prevention (health promotion and supportive networks); early detection (screening, health checks and control of risk factors); and management (continuing care, maintenance and self-management).

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Pertussis (whooping cough)

What is pertussis?

Pertussis (or whooping cough) is a disease caused by infection of the throat with the bacteria *Bordetella pertussis*.

What are the symptoms?

- Pertussis usually begins like a cold, with a runny nose, tiredness and sometimes a mild fever.
- Coughing then develops, usually in bouts, followed by a deep gasp (or whoop). Sometimes people vomit after coughing.
- Pertussis can be very serious in small children. They might go blue or stop breathing during coughing attacks and may need to go to hospital.
- Older children and adults may have a less serious illness, with bouts of coughing that continue for many weeks regardless of treatment.

How is it spread?

Pertussis is spread to other people by droplets from coughing or sneezing. Untreated, a person with pertussis can spread it to other people for up to 3 weeks after onset of cough.

The time between exposure and getting sick is usually 7–10 days, but can be up to 3 weeks.

Who is at risk?

- Anyone can get pertussis.
- People living in the same household as someone with pertussis are more likely to catch it.
- Immunisation greatly reduces your risk of infection, but reinfection can occur.

How is it prevented?

Immunise your child on time

- The vaccine does not give lifelong protection against pertussis, and protection is sometimes incomplete.
- Children need to be immunised at 2, 4 and 6 months of age.
- Boosters are needed at 4 years of age and again at 15 years.
- Immunisation is available through general practitioners and some local councils.

Keep your baby away from people who cough

Babies need two or three vaccinations before they are protected. For this reason, it is very important to keep people with coughing illnesses away from your baby so they don't pass on pertussis or other germs.

Get immunised if you are an adult in close contact with small children

A vaccine for adults is available. It is recommended:

- for both parents when planning a pregnancy, or as soon as the baby is born
- for adults working with young children, especially health-care and child-care workers.

If you are a close contact of someone with pertussis:

- Watch out for the symptoms. If symptoms develop, see your doctor.
- Some close contacts at high risk (e.g. children under 1 year, children not fully vaccinated, and women at the end of their pregnancy) and others who live or work with high-risk people may need to take antibiotics to prevent infection.

If you have pertussis:

- Get treated early while infectious, avoid other people and stay away from young children, e.g. at child-care centres, pre-school and school.

How is it diagnosed?

If a doctor thinks someone has pertussis, a swab from the back of the nose, or a blood test may be done to help confirm the diagnosis.

How is it treated?

A special antibiotic – usually azithromycin, erythromycin or clarithromycin is used to treat pertussis. These antibiotics can prevent the spread of the bacteria to other people.

Coughing often continues for many weeks despite treatment.

What is the public health response?

Doctors and laboratories must confidentially notify cases of pertussis to the local public health unit. Public health unit staff can advise on the best way to stop further spread.

Infectious children are restricted from going to pre-school and school. Unimmunised contacts may be excluded from child care unless they take the special antibiotics.

The factsheet is available at: http://www.health.nsw.gov.au/factsheets/infectious/pertussis.html.

NSW HEALTH

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Communicable Diseases Report, NSW, July and August 2008

Communicable Diseases Branch, NSW Department of Health

For updated information, including data and facts on specific diseases, visit www.health.nsw.gov.au and click on Infectious Diseases or access the site directly at: http://www.health.nsw.gov.au/publichealth/infectious/index.asp.

Figure 1 and Tables 1 and 2 show reports of communicable diseases received through to the end of August 2008 in New South Wales (NSW).

Influenza

The expected seasonal increase in influenza cases was reported in July and August. There were 402 laboratory-confirmed cases notified across the state.

A number of influenza outbreaks were identified among pilgrims attending the Catholic World Youth Day event in July. Because of the mass gathering of people, observed rapid spread and significant morbidity among those infected, public health interventions were recommended to reduce the spread of disease and to minimise hospitalisations. These included sick pilgrims wearing masks and being isolated from others. Advice was provided through group leaders to minimise transmission within groups during the event. Further measures were taken to minimise the risk of spread on transport as the event concluded, with the provision of fact sheets and special hygiene packs for those travelling on buses.

Typical of this time of year, a number of outbreaks also occurred in institutions, including residential aged-care facilities, hostels and boarding schools. Such outbreaks often affect staff as well as residents or students, and highlight the importance of immunisation in at-risk groups and those working with them.

A higher prevalence of circulating influenza type B was observed in NSW this season compared with recent years. A similar pattern was reported in a number of other states.[1]

Influenza is formally notified to NSW Health when confirmed by a laboratory test; however, notifications represent a small fraction of the amount of illness in the community from this seasonal infection. General trends are monitored with additional data from three further sources:

- influenza-like illness presentations to 28 emergency departments across NSW
- deaths due to influenza or pneumonia
- outbreaks.

Compared with other respiratory viruses, influenza tends to cause more severe complications, such as pneumonia, particularly in elderly people and other vulnerable groups such as small children and those with concurrent illnesses (including heart disease, lung disease or diabetes).

Suspected Hendra virus infection excluded

In early August, a laboratory test result from a sick horse at a training facility in northern NSW led veterinarians to suspect Hendra virus infection. The NSW Department of Primary Industries (DPI) contacted NSW Health to warn of a possible risk to human health. To contain a possible outbreak, the DPI initiated a number of actions, including placing the stable where the potentially infected horse was located under quarantine.

The local public health unit identified 15 people who had been in recent close contact with the ill horse and arranged testing for them. Advice was given to stable staff to avoid contact with the ill horse unless absolutely necessary and to wear personal protective equipment when in contact with the animal. Similar advice was given relating to healthy horses in the same stable that may have been incubating the illness. However, communication of the level of risk was difficult given that there is still limited knowledge regarding the behaviour of the virus.

Seven contacts accepted the offer of testing; all were negative on standard tests. Further testing eventually confirmed that the test result of the original ill horse was a false positive, with no evidence of true Hendra virus infection. The episode, however, highlighted the benefits of a close working relationship between NSW Health and the DPI.

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Hendra virus spreads from flying foxes to horses. It causes a variety of symptoms and can be fatal to the horse. On rare occasions the virus has spread from horses to humans. One equine case of Hendra virus infection occurred in NSW in 2007; however, there have been no cases of human Hendra virus infection in the state.[2]

Within Australia, there have been only six confirmed occasions, all in Queensland, of the virus spreading from horses to humans. Three people who have contracted the illness have died, the first two in 1994 and 1995.[3] At the time of this episode, another two people were unwell from Hendra virus, one of whom subsequently died. Three people diagnosed with Hendra virus infection have later recovered. All six cases had been in very close contact with sick or dead horses.[3]

Symptoms of Hendra virus infection in humans have included:[3]

- an influenza-like illness, which can progress to pneumonia
- encephalitis (inflammation of the brain) with headache, high fever and drowsiness, which can progress to convulsions or coma.

The incubation period in humans has been estimated to be from 5 to 14 days. However, in one of the three fatal cases, encephalitis subsequently occurred 13 months after the initial exposure. The limited data available have not indicated human-to-human transmission.

Enteric diseases

In July and August 2008, NSW public health units investigated 204 outbreaks of gastroenteritis, including 196 suspected to be caused by person-to-person transmission, and eight suspected to be the result of foodborne transmission.

The 196 suspected person-to-person outbreaks affected a total of 3023 people. One hundred and thirty-six occurred in aged-care facilities and affected 2290 people; 31 occurred in hospitals and affected 413 people; 25 occurred in child-care centres and affected 292 people; one outbreak at a school affected eight people; and three outbreaks in other institutional settings affected 20 people.

Clinical specimens were submitted for testing for 92 of the 196 suspected person-to-person outbreaks. Rotavirus was confirmed in stool samples from four outbreaks, norovirus was identified in 44 outbreaks and in one aged-care facility both rotavirus and norovirus were detected. The causative agent was not determined for the remaining 43 outbreaks.

Of the eight suspected foodborne gastroenteritis outbreaks:

- Two affected 13 people in whom illness was associated with having eaten oysters at separate private functions in the same geographic region. The NSW Food Authority investigated the source oyster farm and detected norovirus in oysters. This lease was closed as a result.
- Four affected a small number of people after consuming restaurant or takeaway meals. No pathogens were detected in these cases. The premises were inspected but no known sources were identified.
- One outbreak occurred among work colleagues who attended a lunch. Sandwiches from commercial premises were implicated and contamination from a sick food handler was suspected as the source. Food handlers should not attend work until 48 hours after gastrointestinal symptoms have resolved.
- One outbreak was identified in July 2008 following an increase in notifications of a rare serovar of *Salmonella*, S. Anatum. Between 13 May and 2 July, there were nine confirmed cases of S. Anatum infection in two area health services (Sydney South West and Sydney West). The median age of the cases was 26 years; four cases were male. Three of the five cases contactable for interview reported eating a meal from the same restaurant. The NSW Food Authority conducted an environmental investigation at the premises, with food and environmental samples taken. One sample collected from a stainless steel bench in the food preparation area was positive for S. Anatum. One of the food samples was positive for *Salmonella* but not S. Anatum. The source of contamination of the environment could not be identified. There have been no further cases of S. Anatum linked to this premises.

The NSW Food Authority assisted the public health unit of Sydney West Area Health Service in the investigation of several episodes of diarrhoea-predominant illness in residential facilities in western Sydney in July and August. *Clostridium perfringens* was the suspected causative agent in each case, although the mechanism of contamination is not yet clear. Further investigation of this issue is underway.

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Figure 1. Reports of selected communicable diseases, NSW, January 2004 to August 2008, 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 conf, 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 and hospitals than by other institutions.

NSW Population	
Male	50%
<5 y	7%
5–24 y	27%
25–64 y	53%
65+ y	13%
Rural	46%

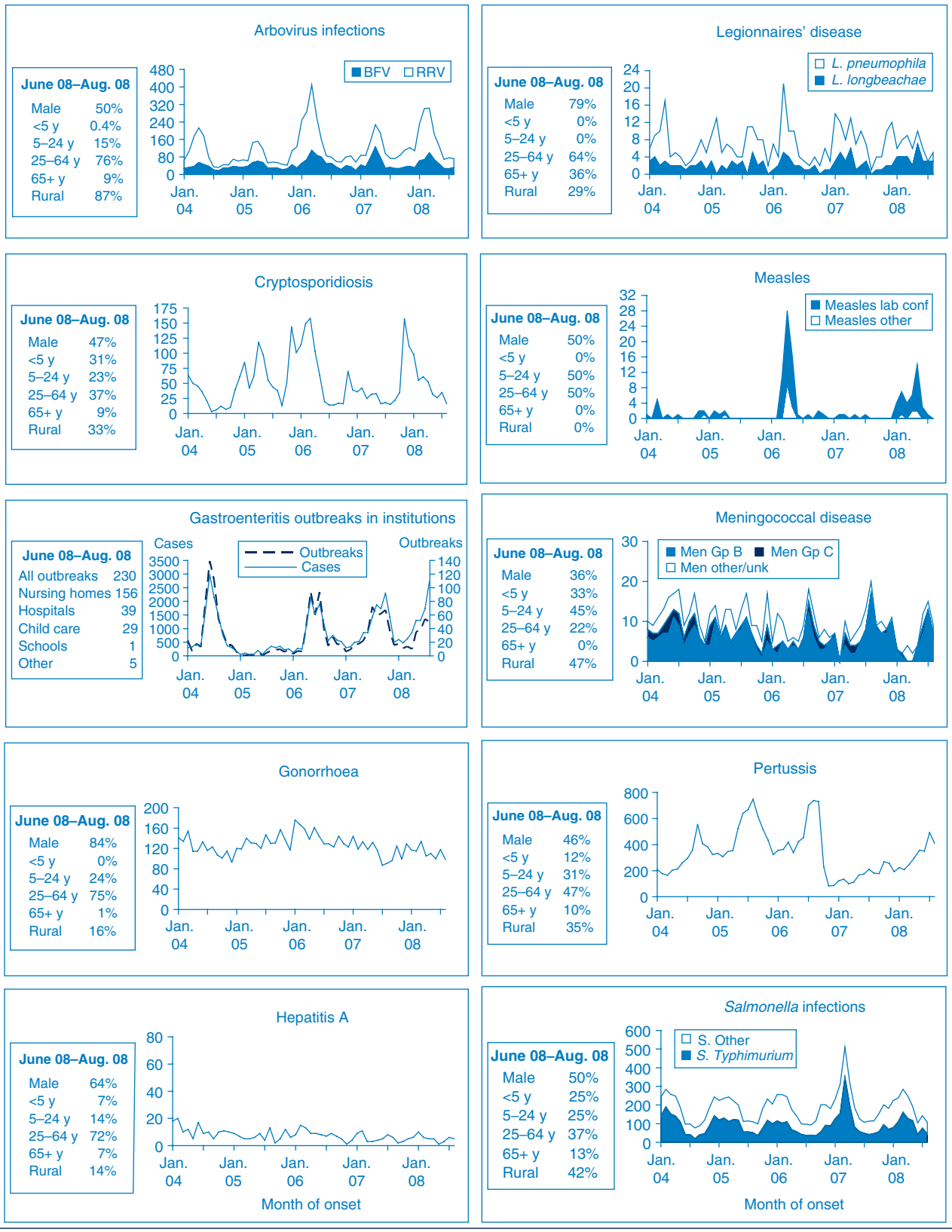

Table 1. Reports of notifiable conditions received in July 2008 by area health services

Condition	Area Health Service (2008)																			Total	
	Greater Southern		Greater Western			Hunter New England		North Coast		Northern Sydney Central Coast		South Eastern Sydney Illawarra		Sydney South West		Sydney West				For July[c]	Year to date[c]
	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA	JHS			
Bloodborne and sexually transmitted																					
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
Chlamydia (genital)[a]	58	28	16	17	26	147	37	30	61	50	90	64	206	–	11	27	104	4		987	8249
Gonorrhoea[a]	–	1	–	2	1	12	–	–	1	–	13	4	41	–	–	2	11	–		88	791
Hepatitis B – acute viral[a]	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	–		2	20
Hepatitis B – other[a]	1	5	3	1	1	2	2	1	2	4	30	6	35	–	2	8	54	2		161	1805
Hepatitis C – acute viral[a]	–	–	2	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–		5	10
Hepatitis C – other[a]	16	12	3	7	9	35	10	20	26	37	19	35	44	–	1	18	20	44		358	3165
Hepatitis D – unspecified[a]	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	1	–		2	7
Lymphogranuloma vanereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
Syphilis	–	4	7	1	–	3	–	1	1	3	7	1	34	1	–	2	8	–		76	675
Vectorborne																					
Barmah Forest virus[a]	1	1	–	2	–	9	–	9	6	–	–	–	–	–	–	–	–	–		28	405
Ross River virus[a]	7	1	3	4	2	8	2	3	9	3	1	1	–	–	–	3	2	–		49	912
Arboviral infection (other)[a]	1	–	–	–	–	2	1	–	2	–	5	1	4	–	–	–	1	–		18	90
Malaria[a]	1	2	–	–	–	3	–	–	–	–	2	–	–	3	–	–	2	–		15	72
Zoonoses																					
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	3
Leptospirosis[a]	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–		1	11
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
Psittacosis[a]	–	–	–	–	1	2	–	1	–	–	–	–	–	–	–	1	1	–		6	25
Q fever[a]	–	1	–	2	–	1	3	–	3	–	–	–	–	–	–	–	–	–		10	90
Respiratory and other																					
Blood lead level[a]	–	1	–	1	1	–	1	–	–	2	–	–	1	–	–	2	–	–		9	152
Influenza[a]	5	1	1	–	1	8	3	1	17	–	8	3	13	–	1	11	36	–		115	510
Invasive pneumococcal infection[a]	6	1	2	2	2	7	1	–	1	6	4	6	4	6	5	6	14	–		73	275
Legionella longbeachae infection[a]	–	1	–	–	–	–	–	–	–	–	–	1	1	2	–	–	–	–		5	26
Legionella pneumophila infection[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	25
Legionnaires' disease (other)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	1
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–		1	3
Meningococcal infection (invasive)[a]	3	–	–	–	–	2	1	–	–	–	–	–	2	–	3	3	–	–		14	40
Tuberculosis	1	–	–	–	–	2	–	–	–	1	3	2	10	1	–	–	17	–		38	248
Vaccine-preventable																					
Adverse event after immunisation	2	1	–	1	1	2	1	–	1	3	3	1	–	1	6	2	3	–		28	182
H. influenzae b infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	7
Measles	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–		1	39
Mumps[a]	–	–	–	–	–	–	–	–	–	–	1	1	–	1	–	–	–	–		3	70
Pertussis	12	4	2	7	3	12	5	11	81	4	33	10	44	18	39	42	114	–		441	2053
Rubella[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–		1	6
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	1
Enteric																					
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
Cryptosporidiosis[a]	2	–	–	–	1	2	–	–	–	4	5	–	9	2	2	7	3	–		37	389
Giardiasis[a]	3	1	1	2	2	17	3	2	–	7	23	12	24	1	1	12	11	–		122	1172
Haemolytic uraemic syndrome	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–		1	9
Hepatitis A[a]	–	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	2	–		4	36
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–		1	7
Listeriosis[a]	–	–	–	–	–	–	–	–	–	–	1	1	–	–	1	–	1	–		4	26
Salmonellosis[a]	6	3	2	2	6	6	5	7	10	4	20	6	12	1	–	15	16	–		121	1457
Shigellosis[a]	–	–	–	–	–	–	–	–	–	–	1	–	–	2	–	–	–	–		3	44
Typhoid[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–		1	23
Verotoxin producing *E. coli*[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	9
Miscellaneous																					
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–		1	2
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	1

[a]Laboratory-confirmed cases only. [b]HIV and AIDS data are reported separately in the Public Health Bulletin quarterly. [c]Includes cases with unknown postcode.

NB: Data are current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation. Historical Area Health Service configurations are included for continuity/comparison purposes and to highlight regional differences.

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

Table 2. Reports of notifiable conditions received in August 2008 by area health services

Condition	Area Health Service (2008)																		Total	
	Greater Southern		Greater Western			Hunter New England		North Coast		Northern Sydney Central Coast		South Eastern Sydney Illawarra		Sydney South West		Sydney West			For August[c]	Year to date[c]
	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA	JHS		
Bloodborne and sexually transmitted																				
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	43	20	13	23	25	141	29	44	58	45	96	49	200	115	85	34	92	11	1129	9621
Gonorrhoea[a]	–	–	–	–	–	4	–	–	4	3	13	3	40	23	5	3	9	1	111	942
Hepatitis B – acute viral[a]	–	–	1	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	2	24
Hepatitis B – other[a]	5	1	1	1	1	6	2	3	–	1	21	3	37	34	49	–	46	8	222	2160
Hepatitis C – acute viral[a]	–	–	1	–	–	2	–	–	–	–	–	–	–	–	–	–	–	–	3	12
Hepatitis C – other[a]	12	13	–	5	10	40	6	22	25	17	14	10	18	17	32	16	23	52	342	3653
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	9
Lymphogranuloma venereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Syphilis	1	–	–	1	1	2	2	2	3	2	5	1	27	17	10	3	7	–	86	764
Vectorborne																				
Barmah Forest virus[a]	–	–	1	–	–	7	1	9	9	1	–	1	–	–	–	–	1	–	30	435
Ross River virus[a]	15	–	2	3	1	5	–	6	4	–	1	3	1	–	–	–	–	–	41	957
Arboviral infection (other)[a]	–	–	–	–	–	–	1	–	–	–	–	–	2	–	–	–	6	–	9	99
Malaria[a]	1	1	–	1	–	–	1	1	–	1	–	–	1	–	–	1	4	–	12	85
Zoonoses																				
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Leptospirosis[a]	–	–	–	–	–	–	–	–	1	–	–	1	–	1	–	–	–	–	3	14
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	2	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	–	–	4	29
Q fever[a]	–	5	2	2	–	1	4	2	3	–	–	1	–	–	–	–	–	–	20	110
Respiratory and other																				
Blood lead level[a]	–	–	2	1	–	6	–	–	1	–	–	–	–	1	–	2	–	–	13	179
Influenza[a]	7	11	–	1	4	44	2	5	63	7	13	12	26	9	27	–	55	1	287	840
Invasive pneumococcal infection[a]	5	3	1	–	4	7	3	4	2	5	11	6	6	3	6	1	8	–	75	352
Legionella longbeachae infection[a]	1	–	–	–	–	–	–	–	–	–	–	1	–	–	–	1	2	–	5	31
Legionella pneumophila infection[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	–	2	27
Legionnaires' disease (other)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Meningococcal infection (invasive)[a]	–	1	–	–	–	2	1	–	–	–	1	–	1	–	2	–	1	–	9	49
Tuberculosis	–	1	–	–	–	–	–	–	–	3	2	–	3	–	–	1	8	–	18	270
Vaccine-preventable																				
Adverse event after immunisation	–	–	–	–	1	1	–	1	–	–	1	–	3	1	3	–	3	–	14	198
H. influenzae b infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	1	8
Measles	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	39
Mumps[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	70
Pertussis	6	3	–	20	11	12	5	18	88	14	45	11	49	30	36	35	131	–	514	2577
Rubella[a]	1	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	–	–	3	10
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Enteric																				
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cryptosporidiosis[a]	–	–	–	–	1	3	–	1	3	2	4	–	3	2	1	1	5	–	26	415
Giardiasis[a]	3	–	–	3	4	18	3	4	–	7	28	7	26	10	10	12	18	–	153	1350
Haemolytic uraemic syndrome	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	9
Hepatitis A[a]	–	–	–	–	–	–	–	–	1	–	1	–	–	–	–	–	2	–	4	40
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	7
Listeriosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	1	27
Salmonellosis[a]	2	9	1	1	3	6	9	4	6	10	30	6	14	11	13	3	11	–	139	1617
Shigellosis[a]	–	–	–	–	–	–	–	–	–	–	2	1	4	–	–	–	1	–	8	52
Typhoid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	2	–	3	26
Verotoxin producing *E. coli*[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	1	10
Miscellaneous																				
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1

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