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CONTROLLING EMERGING DISEASES: NEW CONDITIONS ARE NOTIFIABLE BY LABS AND HOSPITALS FROM DECEMBER 1, 1996

Following an amendment to the Public Health Act 1991, hospital and laboratory staff will be required to notify some new conditions from December 1, 1996. Laboratories will be required to notify cases of **botulism, cryptosporidiosis, verotoxin-producing *Echerichia coli*** (e.g. *E. coli* O157 or O111) **infections** and **blood lead levels ≥0.72 µMol/l** (15µg/dl), and hospitals will have to notify cases of **botulism** and **haemolytic uraemic syndrome.** These conditions should be notified to Public Health Units.

The addition of these conditions to the list of notifiable conditions follows recommendations by the NSW Infectious Diseases Advisory Committee and Public Health Unit directors. Here we outline the salient features of each condition, and the public health response that should follow a notification.

BOTULISM

Botulism is characterised by neurological symptoms including blurred or double vision, drooping eyelids, dry mouth and weakness, reflecting a symmetrical flaccid paralysis starting with the facial muscles and progressing downward. Patients are often alert, and symptoms may be accompanied by constipation, vomiting or diarrhoea. In infants, symptoms often begin with constipation, followed by lethargy, listlessness, difficulty feeding, a weak cry, ptosis, and generalised weakness (the 'floppy baby' syndrome).

Botulism is caused by ingestion or other exposure to a toxin produced by *Clostridium botulinum*. *C. botulinum* spores are common in soil and elsewhere in the environment, and can survive indefinitely, even after boiling. Spores can be killed, however, by high temperatures (>120°C) under pressure. Bacterial growth occurs only in an anaerobic environment and in conditions of low acidity (generally pH>4). The toxin, produced as the bacteria multiply, is heat labile and inactivated by boiling for 10 minutes.

Epidemiologically, cases fall into one of three categories. Although all types are potentially fatal and demand aggressive medical intervention, only one (foodborne) is a public health emergency.

1. Foodborne botulism is caused by ingestion of preformed toxin in food. Typically, implicated foods have been low acid, home-preserved foods that were not heated adequately during preservation, e.g. home-preserved vegetables such as asparagus, beans and other vegetables,

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and foods eaten by some community groups, such as fermented fish heads.

2. Infant botulism is the most common form of botulism, and occurs when ingested spores germinate in a gut without mature flora and toxin is produced in situ. Most cases occur in infants <3 months old, rarely adult 'infant' botulism cases are reported.

3. Wound botulism results from a local *C. botulinum* infection at a wound site in devitalised tissue, where semi-anaerobic conditions pertain. The toxin is produced in situ.

The purpose of surveillance is to:

- identify the source of illness and to prevent others from eating that food;
- assist in the diagnosis and treatment of the identified case; and
- identify others who may be at immediate risk of illness because they have also eaten the suspect food and to assure their proper evaluation and care.

Laboratories and hospitals are required to notify cases by telephone immediately on suspicion of diagnosis.

Case follow-up

Suspected foodborne (but not infant and wound) botulism is a true medical and public health emergency and should be investigated as such. The investigation should urgently identify possible food sources, identify other people who may have eaten the suspect food, ensure patient and food specimens are submitted to the lab, and ensure others do not eat the suspect food.

CRYPTOSPORIDIOSIS

Cryptosporidiosis is characterised by mild to severe watery diarrhoea, often accompanied by abdominal cramps, nausea, vomiting and low-grade fever. Illness can be intermittent and prolonged, lasting days to weeks in many patients. Immuno-compromised patients (e.g. people with AIDS) may never clear the infection. Cryptosporidiosis is grossly under-diagnosed, in part because this parasite may not be identified on a routine stool examination for parasites.

Cryptosporidiosis is caused by *Cryptosporidium parvum*, a protozoan parasite. Infected animals and people excrete large numbers of oocysts in stools. The infective dose is probably very low (<10 oocysts). Oocysts are relatively hardy, and can survive in the environment for weeks or months. They are resistant to concentrations of chlorine and other disinfectants commonly used for water treatment, but can be killed by boiling or largely removed by filtration. A wide variety of mammals can be hosts for this parasite. Young livestock, notably calves and lambs, are commonly infected and may excrete huge numbers of oocysts (>10^8/ml); these animals are very important reservoirs for human infections.

Cryptosporidiosis is transmitted by the faecal-oral route. Typical examples include faecally contaminated drinking or recreational waters; person-to-person spread by direct/indirect contact (e.g. in child care centres); and contact with infected animals. Almost all large outbreaks recognised to date have been traced to waterborne transmission.

The purpose of surveillance is to:

- identify sources of major public health concern (for example, a public water supply or a day care facility) and to stop further transmission; and
- identify whether the case may be a source of infection for other people (for example, a child in nappies or child care attendee), and if so, to prevent further transmission.

Laboratories are required to notify cases by telephone or mail.

Case follow-up

Routine case investigation is optional, *unless* the case is <4 years old and attends day care, or the number of recent reports suggests an outbreak may be occurring.

Children with diarrhoea or other symptoms referable to cryptosporidiosis should not attend day care until symptoms have resolved for >24 hours. An exemption should be granted only if separating ill children from well children and special care with hand washing after nappy changing and before food handling can be implemented to prevent transmission.

If the number of reported cases in an area is higher than usual for the time of year, routine follow-up investigations for all cases reported should be done, including cases notified at least two weeks before the apparent upswing.

Haemolytic uraemic syndrome and verotoxin-producing *Escherichia coli* Infection

Haemolytic uraemic syndrome (HUS) is characterised by a triad of microangiopathic haemolytic anaemia, thrombocytopenia and renal failure. About 90 per cent of cases of HUS occur in early childhood and are preceded by a diarrhoeal illness (usually bloody). Verotoxin-producing *E. coli* (VTEC) is the most commonly reported cause of diarrhoea-associated HUS, although *Shigella dysenteriae* type 1 infections are also reported.

Over the past few years, substantial outbreaks of VTEC, in particular *E. coli* O157:H7 have been reported from the United States, Europe and Japan. Infections tend to have a seasonal pattern, with increases during the summer months. Most *E. coli* O157:H7 infections can be traced back to cattle. Outbreak sources have included contaminated meats (hamburgers, unprocessed meat), other contaminated foods (unpasteurised dairy products, apple cider), drinking water and swimming water. Person-to-person transmission

is also well documented. The incubation period of *E. coli* O157 disease is usually 2-8 days but may range up to 12 days. Diarrhoea may become bloody within 48 hours and be accompanied by abdominal cramps, nausea and vomiting. Fever is generally absent or low-grade. In *E. coli* O157 outbreaks, 5-15 per cent of cases develop HUS, usually within 3-10 days of infection.

Until recently, there has been little evidence of VTEC outbreaks in Australia. However, in January 1995, the first reported Australian outbreak of HUS and *E. coli* disease was reported in South Australia associated with *E. coli* O111. This epidemic comprised 23 HUS cases (including one death) and was linked to consumption of mettwurst. Little is known about the association between HUS and *E. coli* O111 and other non-O157 organisms. In particular, it is unknown what proportion of infections lead to diarrhoea, bloody diarrhoea or HUS.

The purpose of surveillance is to:

- identify whether the case may be a source of infection for other people;
- identify outbreaks and potential sources or sites of ongoing transmission in order to reduce possible further transmission; and
- better understand the epidemiology of these conditions (disease profile, risk factors and sources of transmission).

Laboratories are required to report VTEC infections by telephone or mail. Hospital staff will be required to report cases of HUS by telephone.

Case follow-up

Case investigation should be conducted and possible sources of infection identified. Stool cultures of HUS cases should be referred to an appropriate laboratory for tests for *E. coli* organisms. Food handlers, day care workers and health care workers should not attend work as long as they have diarrhoea. Symptomatic children should be excluded from child care facilities. Individuals and carers should be educated on measures to avoid further or future exposures including: to avoid eating raw or undercooked minced meat, especially hamburger, to avoid cross contamination with meat and other contaminated foods, and to wash hands after changing nappies. Advice on improving food handling or day care environments may be indicated.

ELEVATED BLOOD LEAD

Lead intoxication can affect both children and adults, although the effects may vary markedly with age. The most prominent signs and symptoms are neurological.

Acute disease

Symptoms of lead poisoning include seizures, bizarre behaviour, ataxia, apathy, diarrhoea, restlessness, incoordination, vomiting, alteration in consciousness and subtle loss of recently acquired skills. At higher levels lead can cause convulsions, coma or even death. Adults will frequently remain asymptomatic even with chronic exposure until the blood lead level is greater than 2.4 µMol/l (50 µg/dl).

Chronic effects

Recent studies suggest lead absorption is harmful at any concentration. Overt signs and symptoms of lead poisoning are rarely present at low levels, but exposure can cause neurological damage, especially in developing children, including decreased IQ, developmental delays and behavioural disturbances.

Sources of lead exposure

Lead can be ingested or inhaled. The most common source of preventable lead exposure is probably lead-containing dust. The extent of lead uptake is affected by an individual's developmental stage, the route of exposure and the nature of the lead to which the individual is exposed. Nutritional status may also be important; a healthy diet high in iron and calcium and low in fat, for example, may slow the rate of lead absorption in children. Lead absorption rates may vary from 10 per cent in adults to perhaps 50 per cent in children. Uptake from pulverised paint chips or dust from sanded lead-based paint is more efficient than from whole paint chips. The most dangerous exposure is probably to lead vapours (formed whenever lead is melted).

Common sources of lead exposure include:

Paint – Lead was used in significant proportions of paint manufactured in Australia before the mid-1960s. Paint that is peeling, chipping, or chalking is a common source of ingestible lead, particularly by toddlers or other children with pica. Paint dust can be inhaled or swallowed by people living or working in residences or other buildings being renovated or remodelled, unless proper precautions are taken. Renovations conducted on older houses in city areas may also distribute lead dust in the ceiling, which may recirculate or recontaminate the house.

Motor vehicles – The amount of lead petrol used in NSW has declined dramatically since 1985 when new cars were required to use unleaded fuel. However, lead in leaded petrol contributes to elevated air lead levels, which in turn contribute to elevated blood lead levels. About 50 per cent of cars still use leaded petrol. Batteries may contain lead, and occupational exposure may occur among persons who manufacture, repair or recycle these materials. As an occupation, radiator repair is notorious for lead exposure.

Hobby sources – Many hobbies involve lead use, for example making lead shot, sinkers or toy soldiers; working with stained glass, ceramic manufacture and glazing. Heating and melting lead is particularly dangerous, because of the formation of vapours.

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A LINK IN THE CHAIN OF SURVIVAL: NSW AMBULANCE RESPONSE TO CHEST PAIN

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This report reviews the effectiveness of the ambulance transport component of the system for emergency response to acute chest pain in NSW, using routinely collected data.

Chest pain is an important symptom of coronary ischaemia and is the key to early recognition of acute coronary ischaemia (acute myocardial infarction and unstable angina) by the general community. Although the ratio of cases of confirmed acute myocardial infarct (AMI) to non-coronary chest pain may be low[1,2,3], an effective system for management of those with chest pain will be the basis for effective delivery of definitive care of acute ischaemia.

The "Chain of Survival" model[4] of emergency care emphasises the importance of:

- rapid public access to emergency care;
- training in cardio-pulmonary resuscitation techniques;
- early defibrillation; and
- prompt access to advanced cardiac life support.

To this should be added:

- early thrombolysis; and
- prompt access to advanced management and monitoring.

Effective delivery of care according to this model requires attention to issues such as symptom recognition in the community, pre-hospital care (including cardio-pulmonary resuscitation and defibrillation), response time by ambulance services, diagnosis and emergency treatment.

METHODS

The NSW Ambulance Service has a written protocol for the management of chest pain. When the Ambulance Service responds to a call for assistance, patient demographic details, clinical conditions, transportation mode and timing and any pre-hospital care given are recorded in the "ambulance patient report form". These data were entered in a database.

Data were analysed for "casualty calls" (i.e. not booked transport), with a complaint of chest pain, for the period January to June 1994.

Hospital separations for AMI (defined by the ICD.9.CM rubric 410) from the NSW Health Department 1993-94 Inpatient Statistics Collection (ISC) were also analysed to assess utilisation of ambulance services in relation to a discharge diagnosis of AMI.

RESULTS

There were 309,839 ambulance transportations in New South Wales during the study period (the first six months of 1994). Of these, 172,817 were casualty calls (56 per cent).

Of the 172,817 casualty calls, 10,743 (6.2 per cent) involved chest pain, although 751 (7.0 per cent) of these calls were cancelled, and a further 115 (1.1 per cent) were inter-hospital transfers or discharges from hospital to home (possibly miscoded as casualty calls). The remaining 9,877 (5.7 per cent) were confirmed casualty calls, managed by the Ambulance Service and transported to hospitals. Data for those calls formed the basis of our analysis.

Chest pain protocol

Of the 9,877 definite casualty chest pain cases transported by ambulance, 8,336 (84.4 per cent) were managed using the Ambulance Service's chest pain protocol. The rest were mainly managed under their basic protocol. The chest pain protocol recommends that glyceryl trinitrate be given if "myocardial ischaemia is suspected and systolic blood pressure is greater than 100 mm Hg", and that aspirin be given if coronary artery occlusion is suspected and there is no history of aspirin allergy.

Only about one in four (23.7 per cent) patients who were managed under the chest pain protocol were given aspirin, though nearly two-thirds (62.7 per cent) were given glyceryl trinitrate. About one in three (34 per cent) were given neither aspirin nor glyceryl trinitrate while in transit.

Time of call for ambulance assistance

There were no data recording the time from onset of chest pain. The ambulance record shows only the time of requesting ambulance assistance. These records showed a peak in requests for ambulance assistance for chest pain between 7am and noon. Another peak occurred between 6pm and 8pm.

The NSW Ambulance Service received an average of 54 calls a day for chest pain. There was no substantial variation by day of the week, although Monday had the most cases on average (57), followed by Sunday (56). This pattern of variations was similar to the results of the 1982 Ambulance Inquiry[5].

Ambulance performance

The time from call for assistance to ambulance team leaving the depot and the time from call for assistance to arrival at the scene (response time), are important indicators of service quality.

Overall, the time from calling the ambulance to arrival at hospital was less than 30 minutes in 31.3 per cent of cases, less than 45 minutes in 77.7 per cent and less than 60 minutes in 92.7 per cent of cases (Table 1).

The Ambulance Service is considering standards[6] which have been developed and are used in the United Kingdom (UK) based on a metropolitan service. These standards have not been validated in NSW.

The NSW ambulance data show that, in about 80 per cent of cases, the ambulance left the depot within three minutes of receiving the request (Table 2). In about 37 per cent of cases in NSW, the ambulance arrived at the scene within seven minutes of receiving the call and in 84 per cent of cases, the ambulance arrived at the scene within 14 minutes. Comparisons can be made between the NSW performance and the UK standards for urban and rural areas (Table 2). For example, in NSW urban areas the ambulance reached the scene within seven minutes of an emergency call in 32 per cent of cases, compared to the UK urban standard of 50 per cent. In NSW rural areas the corresponding proportion was 54 per cent within seven minutes, compared

TABLE 1

PROPORTION (%) OF AMBULANCE CASUALTY CALLS FOR CHEST PAIN WITHIN DEFINED CATEGORIES OF TIME FROM REQUEST FOR AMBULANCE TO ARRIVAL AT HOSPITAL, JANUARY TO JULY 1994.

	Within 15 min	Within 30 min	Within 45 min	Within 60 min	Within 75 min	Within 90 min	Within 105 min	Within 120 min
Urban	0.8	27.8	78.3	94.4	97.9	98.9	99.4	99.6
Rural	2.5	42.9	75.8	87.0	93.4	96.6	98.1	98.8
NSW	**1.2**	**31.3**	**77.7**	**92.7**	**96.7**	**98.4**	**99.1**	**99.5**

TABLE 2

PROPORTION NSW AMBULANCE SERVICE CASUALTY TRANSPORTS FOR CHEST PAIN FALLING WITHIN UK URBAN/RURAL STANDARD TIMES FROM CALL TO LEAVING THE DEPOT AND ARRIVING AT THE SCENE, JANUARY TO JULY 1994.

	From call to leaving depot		From call to arrival at scene		From call to arrival at scene	
	NSW study % (time)	UK standard % (time)	NSW study % (time)	UK standard % (time)	NSW study % (time)	UK standard % (time)
Urban	83 (3 min)	95 (3 min)	32 (7 min)	50 (7 min)	85 (14 min)	95 (14 min)
Rural	69 (3 min)	95 (3 min)	54 (7 min)	50 (8 min)	82 (14 min)	95 (18 min)
Total	**80 (3 min)**	n/a	**37 (7 min)**	n/a	**84 (14 min)**	n/a

TABLE 3

PROPORTION (%) OF NSW AMBULANCE SERVICE CASUALTY CALLS FOR CHEST PAIN FALLING WITHIN SPECIFIED ACTIVATION AND RESPONSE TIMES, JANUARY TO JULY 1994.

	From call to leaving depot (5 min)	Response time (10 min)	Response time (15 min)
Urban	94	63	88
Rural	90	72	84
NSW	**93**	**65**	**87**

Data source: NSW Ambulance Service 1994 (HOIST), Epidemiology Branch, NSW Health Department.

to the UK rural standard of 50 per cent within eight minutes.

If the conventional five-minute intervals are used to measure performance, the ambulance data show that in 93 per cent of cases, the ambulance left the depot within five minutes of receiving the request. About 87 per cent of the time the ambulance arrived at the scene within 15 minutes, and about 96 per cent of the time within 30 minutes, of the time of receiving the request (Table 3).

There were some rural and urban differences in time from ambulance call to arrival at scene (Tables 1 and 2), with time from call to leaving depot being longer and response time shorter in rural than urban areas.

Hospitalisation: ambulance-transported AMI patients

In 1993-94 there were 10,839 hospital separations in NSW where AMI was the principal diagnosis. Of these, 1,088 (10 per cent) had an ambulance number in their patient record, indicating they were brought in by an ambulance. The majority (90 per cent) of the 10,839 people with an AMI arrived at hospital using their own transportation arrangement.

DISCUSSION

These data show that, though there is room for improvement, the response times of the NSW Ambulance Service appear to be very good overall, even in relation to the UK standards. These are based on the UK experience and need to be validated in a local context.

Observed differences between rural and urban areas may be due, at least in part, to geographic conditions. It should be noted that, if the call is between midnight and 6am in rural areas there may be a slight delay as most officers are called in from home to attend the case.

A more significant issue may be the number of people who access the ambulance system at all. According to the 1993-94 NSW ISC, the ambulance services transported only 10 per cent of the AMI patients admitted to NSW hospitals. The recording of an ambulance number in the ISC almost certainly underestimates the number of people who arrive by ambulance. Nevertheless, even if this underestimation is of the order of 100 per cent, it seems that only about one in

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five people who have an AMI arrive by ambulance. Data from the USA estimate that only about 50 per cent of people presenting to emergency departments with chest pain arrive by ambulance[7].

The next step in the "chain of survival"[4] is entry to the emergency care system. This system should enable rapid assessment of patients in the "field" (out of hospital by ambulance staff or in emergency departments) according to simple protocols. Protocols should include rapid transfer to a hospital equipped to manage patients with AMI.

It is not possible to determine from these data the appropriateness of applying the Ambulance Service Chest Pain Protocol. The cases recorded as "chest pain" where glyceryl trinitrate or aspirin were not used may have been judged to be non-ischaemic in origin, such as trauma or other medical conditions, or did not fulfil the criteria that indicate coronary ischaemia.

Approximately one in four patients were given aspirin, though almost two-thirds were given glyceryl trinitrate, for suspected coronary ischaemia. The concern is not so much the failure to give aspirin in itself. The best evidence at present, however, is that it should be commenced within 24 hours of the infarct, and commencement in the ambulance does not necessarily confer additional benefit[8].

Early recognition of symptoms and entry into the system of emergency care is crucial to ensuring desired health outcomes for people with AMI. In addition, those in whom a thrombolytic is contraindicated and those with unstable angina will benefit from early assessment and treatment to limit disease progression, loss of cardiac function and possible mortality. Nevertheless, delays in delivery of definitive treatment for acute coronary ischaemia have been documented in many studies[9,10,11,12,13,14,15]. The major source of delay seems to be before the decision to seek emergency assistance[14]. Of some concern is the suggestion that people are less likely to seek care expeditiously when they have been diagnosed with coronary heart disease[16,17].

There are many possible reasons for these delays, including patient and family denial. A decision to seek medical help from the local doctor may significantly increase the time to hospital and definitive care[13,14].

Measures to encourage people (especially those known to be at high risk, such as people with diagnosed CHD) to respond appropriately to persisting chest pain and to minimise delays in assessment and definitive management of people in the emergency department have the potential to substantially reduce avoidable mortality and minimise loss of cardiac function.

A systematic approach to management of acute coronary ischaemia is needed to address these issues. The NSW Rural and Metropolitan Critical Care Plans could serve as a model for such an approach. Though these plans have not explicitly addressed coronary ischaemia, they do deal with many generic issues and could provide the architecture for the effective delivery of services for the management of acute coronary ischaemia in NSW.

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GASTROENTERITIS OUTBREAK LINKED TO FOOD HANDLER

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This article reports on an investigation to determine the cause of an outbreak of gastroenteritis in a large Sydney institution employing some 1,100 people, and on control measures that were implemented.

On January 17, 1995 the occupational physician at the institution reported that seven employees were ill with nausea, abdominal cramps, diarrhoea and vomiting, and that 50-60 other employees may also have been ill. His initial inquiries suggested the only common exposure of the seven was that they had eaten together in the institution's cafeteria on January 13.

Foodborne disease outbreaks constitute a significant public health problem in NSW. In 1993, 24 outbreaks involving some 516 cases were reported in the State; this was probably a small fraction of the true number. Viral agents were suspected in five outbreaks and Norwalk-like agents accounted for half of all cases[1]. Factors that contribute to foodborne outbreaks from contaminated food include infected food handlers, inadequate cooking temperatures, improper holding temperatures and contaminated equipment. Most incidents can be easily avoided by adherence to basic rules of food hygiene[2].

METHODS

Epidemiological investigation

The occupational health physician initially conducted telephone inquiries to identify employees who were ill. Subsequently we obtained demographic information from employees, and asked them about their symptoms (including duration), and food consumption on January 13. Cafeteria food handlers were asked about illness before January 13.

We defined a case as an employee who ate at the cafeteria and developed vomiting or diarrhoea and at least one of the following symptoms after January 13: nausea, abdominal cramps, fever, headache, muscle aches or pains. The date was determined on the basis of information on food consumption before onset of symptoms.

We conducted a case-control study in which the controls were employees who ate at the cafeteria and reported no symptoms, identified as a convenience sample. Food consumed and other exposures by cases and controls were compared.

Environmental investigation

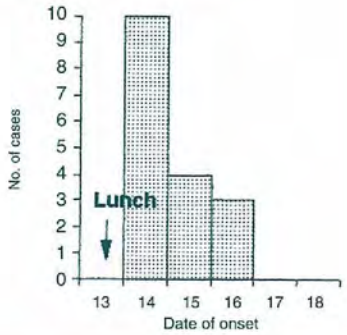
We asked cafeteria managers about food items served on January 13, possible staff illnesses, and food handling and hygiene practices. We inspected the cafeteria to determine food preparation methods, hygiene and storage practices, potential for cross-contamination and temperature abuse. Food handlers were interviewed about food storage, preparation and transportation.

Laboratory investigation

On January 18 we collected samples remaining from food served on January 13 (including seafood salad, salmon, curried egg, ham, salami, tuna, corned beef, pastrami, egg mayonnaise, milk, cooked chicken pieces, turkey roll, cottage cheese and coleslaw with prawns). The samples were taken to the Division of Analytical Laboratories where they were analysed by standard bacteriological methods looking for faecal coliforms, *Escherichia coli, Bacillus cereus, Salmonella* and *Clostridium perfringens*. Stool specimens from cases and food handlers were examined for *Salmonella, Shigella, Campylobacter, Yersinia* and parasites including *Cryptosporidium*.

FIGURE 1

CASES OF GASTROENTERITIS AT THE INSTITUTION, JANUARY 1995



Statistical methods

The statistical significance of associations between illness and foods consumed was determined using the chi-square test, or the two-tailed Fisher exact test, with a 5 per cent significance level.

RESULTS

Epidemiological investigation

We identified 17 cases who reported an onset of illness during the period January 14-16 (Figure 1). For 10 cases, the onset of symptoms was on January 14, while for four cases, symptoms began on January 15, and for three, on January 16. There were no new cases after January 16. Ten (58 per cent) were males and median age was 34 years (range 19-58 years). The cases were employed in a variety of work areas within the institution. Symptoms included vomiting (reported by 88 per cent), diarrhoea (77 per cent), nausea (82 per cent), fever (65 per cent) and abdominal cramps (53 per cent).

In the case-control study, age and sex distributions of cases and controls were similar. Among the 17 cases and 13 controls, no single food item was associated with the illness. However, 14 of the 17 cases (82 per cent) consumed sandwiches, compared with 5 of the 13 controls (38 per cent) (odds ratio 7.5, $p < 0.05$) (Table 4).

The mean period from consumption of sandwiches on January 13 to onset of first symptom was 48 hours (range 30-73 hours, median 47 hours).

Environmental investigation

The cafeteria was serviced by a catering company. Employees could also bring food from home or other shops at least three kilometres away.

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Gastroenteritis outbreak

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One of the food handlers in the cafeteria reported an illness characterised by abdominal cramps, vomiting and diarrhoea beginning on January 12. Despite this illness the food handler was able to continue working in the food preparation area on the morning of January 13. Two other food handlers reported developing similar symptoms on January 14 and 15.

Storage temperatures of some of the potentially high-risk fillings in the sandwich bar were above the recommended temperature of 5 °C (ham and salami 20°C, chicken 11°C). Inspection of the cool-rooms revealed that towels were used to wrap different types of cold cuts. The hand-washing basin at the time of inspection was being used to cool boiled eggs.

TABLE 4

CHARACTERISTICS OF CASES AND CONTROLS AT INSTITUTION, JANUARY 1995

	Cases (n=17)	Controls (n=13)	Odds ratio
Median age (range)	30 (19-58)	37 (20-63)	–
Sex Male	10 (59%)	9 (69%)	0.6
Female	7 (41%)	4 (31%)	
Foods Consumed			
Hot foods	7 (41%)	9 (60%)	0.3
Chips	7 (41%)	5 (38%)	1.1
Pies/pastries	2 (12%)	4 (31%)	0.3
Sandwiches	14 (82%)	5 (38%)	7.5*
Eggs	2 (12%)	–	–
Salami	2 (12%)	–	–
Ham	3 (18%)	2 (15%)	1.2
Pastrami	1 (6%)	–	–
Chicken	4 (24%)	1 (8%)	3.7
Corned beef	1 (6%)	–	–
Salad	5 (29%)	3 (23%)	1.4
Cheese	4 (24%)	3 (23%)	1.0
Turkey	1 (6%)	–	–
Snack bar	1 (6%)	2 (15%)	0.3
Drinks	5 (29%)	5 (38%)	0.7

* $p < 0.05$

Laboratory investigation

Microbiological examination of the food samples showed evidence of faecal coliform contamination in the corned beef, ham, seafood salad, salmon, turkey roll and chicken pieces. *Escherichia coli* was present in the chicken pieces, ham and corned beef. *Salmonella ohio* was detected in the corned beef (Table 5).

Stool specimens from four ill cafeteria staff (collected within 72 hours of onset) were negative for bacteria and parasites.

CONTROL MEASURES

On learning of the possible link to the cafeteria, the catering company voluntarily closed the cafeteria. Food items were removed from sale and food preparation areas disinfected. Ill food handlers were removed from kitchen duties until completely free of symptoms and stool samples were clear.

We provided advice to the food handlers and management on food safety and hygiene. They were advised to ensure that storage temperatures of potentially high-risk fillings in the sandwich bar were kept below 5°C, and to modify food-handling practices (methods of storing and wrapping food items). They were also educated on the need for personal hygiene to minimise the possibility of contamination spread (use of hand-washing basin and sinks).

DISCUSSION

The symptoms, incubation period and absence of other pathogens in human specimens suggest this outbreak was caused by a Norwalk-like virus passed from an infected food handler to sandwiches. The laboratory finding of faecal contamination of a number of samples supports the epidemiological finding that consumption of a number of sandwich items led to illness. The isolation of salmonella in one corned beef sample may have been coincidental.

Frequently two or more factors act together to cause contamination and promote survival of pathogens. Examples include contamination of food by infected food handlers and cross-contamination of food from uncooked food items.

Continued on page 108 ►

TABLE 5

MICROBIOLOGICAL EXAMINATION OF THE FOOD SAMPLES (JANUARY 18, 1995)

Sample type	Seafood salad	Salmon	Ham	Corned beef	Cooked chicken	Turkey roll
Standard Plate Count/g	4.5×10^5	2.0×10^6	3.2×10^8	8.4×10^7	1.0×10^9	2.5×10^7
Salmonella	ND	ND	ND	Detected	ND	ND
Faecal coliforms/g	9	4	23	4.3×10^2	93	4
E.coli/g	< 3	< 3	23	1.5×10^2	9	< 3
Clostridium perfringens/g			< 50	< 50	< 50	< 50

NEWS AND COMMENT

BRAIN INJURY REHABILITATION POLICY IN NSW

Magnolia Cardona, Ruth Dewar, Jim Hyde
Health Services Policy, NSW Health Department

NSW Health spends more than $15 million each year maintaining the Brain Injury Rehabilitation Program (BIRP), a network of services across the State. The Motor Accidents Authority of NSW has also made a major financial contribution for the establishment of this network to provide inpatient, outpatient, outreach and transitional living services for people who have had a traumatic or non-traumatic brain injury.

The Health Services Policy Branch is putting together a policy framework to set the direction for brain injury rehabilitation (BIR) services across the State. We aim to assess and address the gaps and overlaps in coordination of referral, integration of services, admission criteria, clinical practice patterns, availability of community support services and cost-effectiveness of existing services.

Among other concerns, we will be considering the following in the consultation towards the draft BIR policy:

- Target groups
- Health Outcomes measurement
- Service databases
- Equity and access issues
- Resource issues
- Training issues
- Quality and best practice
- Role of the public and private sector
- Building partnerships
- Consumer participation

Calls for submissions have been invited from relevant service providers, health planners, academics and consumer groups. Their comments will be incorporated into a directions paper to be circulated for additional feedback. For further details contact Magnolia Cardona, Health Services Policy, on ph: (02) 9391 9008, fax: (02) 9391 9615, or E-mail: mcard@doh.health.nsw.gov.au.

CLUSTERS OF ILLNESS

The recent amendment to the Public Health Act (see page 95 of this issue) provides a stimulus to encourage notification of unusual clusters of illness to Public Health Units. This notification is not required under the Public Health Act 1991, but voluntary notification may lead to effective public health action for a problem that might otherwise not come to attention.

From time to time, clusters of illnesses that are not notifiable occur in the community. Recent examples include:

- a cluster of people with elevated arsenic level linked to consumption of herbal remedies;
- a cluster of people and horses with respiratory illness subsequently identified to be due to a new virus (equine morbillivirus); and
- a cluster of people with adult respiratory distress syndrome in New Mexico, subsequently identified as hantavirus infection.

In many cases, the presence of such clusters can represent a risk to public health. They may indicate a source of infection which can be identified and controlled, thus preventing the further spread of the illness. In some cases, they may represent a previously unidentified pathogen which may be a significant risk to public health.

Medical practitioners, hospitals and laboratory staff are encouraged to contact their local Public Health Unit if they identify such clusters and are of the view that a risk to the public health may eventuate should the matter not be investigated. This notification can be done on an non-identifying basis. The PHU can consult with the NSW Health Department which can initiate a public health inquiry if appropriate.

INFECTIOUS DISEASES

TRENDS

The seasonal increase in **meningococcal infection** began early in NSW this year (Figure 3), with more cases (23) in July than in any other month in the previous twelve months. Reports of meningococcal disease continued to surge in August (23, [Table 6]). Many of these were related to a cluster of cases in the Penrith area (see below). Clinicians are reminded that penicillin should be given by injection at first suspicion of meningococcal diseases (even before transporting the patient to hospital for definitive diagnosis).

There were also more reports of **hepatitis A** than expected (64 reports in August), with cases prominent in the South Eastern Sydney (16), Central Coast (11), Western Sydney (7), Illawarra (7), Central Sydney (6) and Mid Western (4) Areas (Table 6).

Reports of vaccine-preventable disease (***Haemophilus influenzae* type b infection, measles, pertussis** and **rubella**) remained lower than historical levels (Figure 2). Reports from the Eastern Sydney Laboratory Surveillance Program (Figure 4) indicate that **respiratory syncytial virus** (RSV) activity has waned since peaking around June, and **rotavirus** activity has not reached the peak seen in previous years.

Influenza surveillance

Influenza activity declined at the end of August and beginning of September, as expected for this time of year.

Reports from the NSW Sentinel GP Surveillance Scheme[1] show the average consultation rate for influenza-like illness (ILI) in the last week of August was 1.5 per cent (Figure 5), slightly lower than the historical average. Southern NSW was the only area reporting a consultation rate above 2 per cent. The average school absentee rate[2] has continued at levels similar to previous years (Figure 6).

Reports from Westmead, Prince of Wales and Liverpool hospital laboratories indicate that during the last week of August and first week of September the number of diagnoses of influenza A was lower (3 serological, 13 virological diagnoses) than for the preceding two weeks (14 serological, 20 virological). There were no diagnoses of influenza B in either period.

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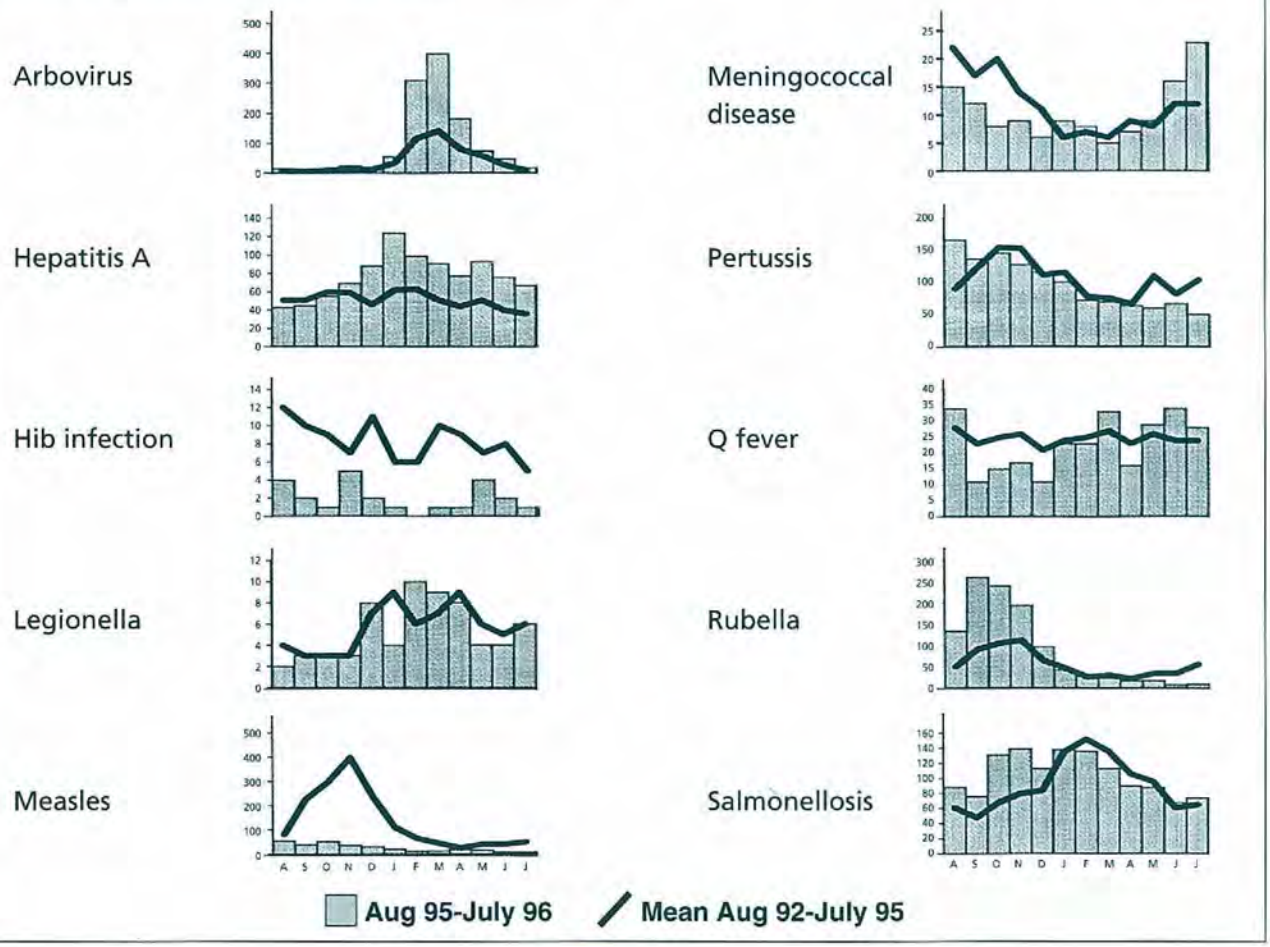
1. Including about 6,000 consultations a week to 50 doctors reported to five Public Health Units.
2. Monitored from 10 schools including about 9,000 students, reported to four Public Health Units.

FIGURE 2

REPORTS OF SELECTED INFECTIOUS DISEASES, NSW, 12 MONTHS TO JULY 1996 BY MONTH OF ONSET (WITH HISTORICAL COMPARISON)



Invasive meningococcal disease outbreak in western Sydney

Bin Jalaludin[a], Marianne Kerr[a], Jane Jelfs[b], Tim Heath[a,c], Oanh Nguyen[a], Moira Hewitt[c,d], Rosemary Munro[b], Anthony Capon[a], Jeremy McAnulty[e]

The Western Sector Public Health Unit (WSPHU) was notified of 14 cases of invasive meningococcal disease (IMD) between August 3 and September 11, 1996.

The cases presented to the emergency departments of four hospitals in western Sydney, with 11 presenting to one emergency department.

Of the 14 cases, eight were males, ages ranged from 2-66 years (eight were aged 14-21 years), 10 lived in the Penrith area, and eight were associated (directly or indirectly) with attendance at a single nightclub in Penrith. The first three cases all attended the nightclub on July 31, 1996. In five subsequent cases, household or other close contacts also went to the nightclub, at least three of them on the night of July 31. Staff estimated about 500 people attended the nightclub that night (Figure 3).

Neisseria meningitidis serogroup C was isolated from 11 cases (a further three cases were diagnosed on clinical grounds alone). Meningococcal isolates from 10 of the 11 cases were phenotypically similar (serotype 2a subtype P1.5).

Symptoms of IMD include sudden onset of fever, headache, stiff neck, nausea, weakness, drowsiness and a purpuric rash. The organism is spread directly from person to person by droplets or discharge from the nose or throat of a carrier. Up to 20 per cent of people in some communities can carry meningococci in their nasopharynx. The incubation period (time between infection and illness) is usually 3 to 4 days, but can be up to 10 days. The illness can be effectively treated with antibiotics in hospital.

Cases of IMD generally increase each year in late winter and spring. In NSW, 116 cases of IMD were reported in 1995, and by the end of August 1996, 101 cases had been

a Western Sector Public Health Unit.
b South Western Area Pathology Service.
c National Centre for Epidemiology and Population Health.
d Department of Public Health and Community Medicine.
e AIDS/Infectious Diseases Branch

FIGURE 3

CASES OF MENINGOCOCCAL DISEASE, WESTERN SYDNEY, JULY-SEP 1996, BY DATE OF ONSET AND REPORTED CONTACT WITH THE NIGHTCLUB

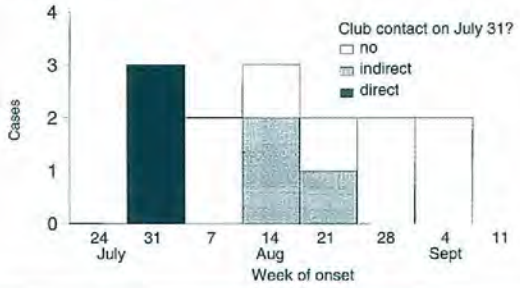


FIGURE 4

LABORATORY REPORTS OF SELECTED INFECTIOUS DISEASES, EASTERN SYDNEY LABORATORY SURVEILLANCE PROGRAM, 13 X 4 WEEK REPORTING PERIODS (1 YEAR) TO END AUGUST 1996 (AND HISTORICAL COMPARISON)

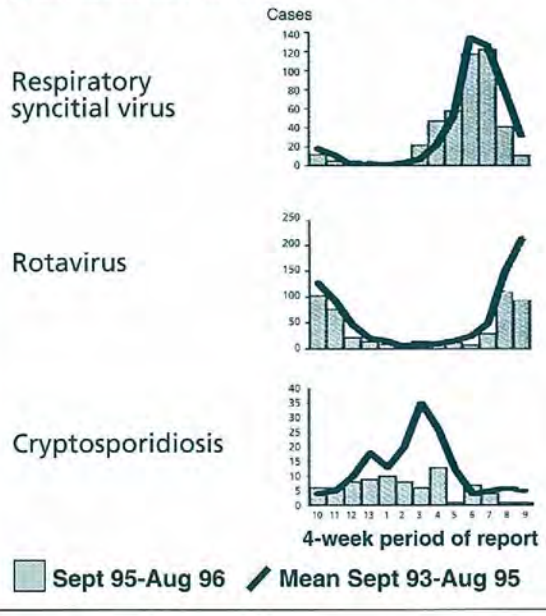


FIGURE 5

NSW GP SENTINEL SURVEILLANCE INFLUENZA-LIKE ILLNESS 1996

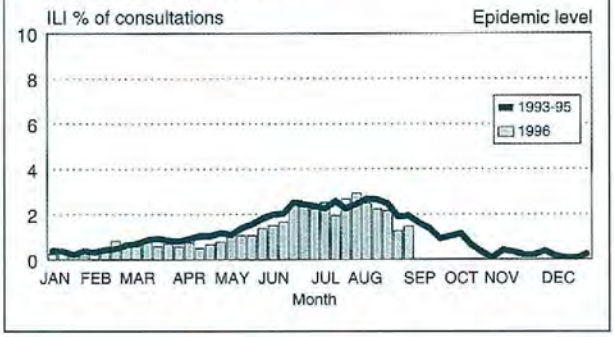
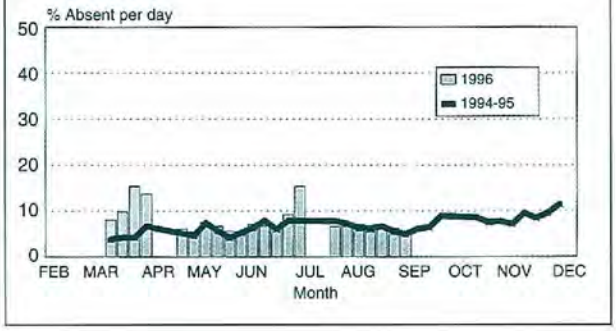


FIGURE 6

SCHOOL ABSENTEE RATE SURVEILLANCE NSW 1996



reported. In western Sydney, six cases were reported in 1995, and at the time of writing, 20 cases had been reported in 1996 (including the 14 cases reported in this cluster).

It is likely that on the night of July 31, a patron who was carrying the bacteria transmitted it to others by coughing in a densely crowded, smoke-filled room. Smoking – either actively or passively – substantially increases the risk of meningococcal disease. Examination of nightclub staff found that none of the 37 tested had evidence of the infectious strain. Consultation with experts suggests that carriage of group C meningococcus by close contacts of cases for long periods is rare; people who acquire the organisms either become ill, or clear it within a short time.

In response to this outbreak, Public Health Units throughout NSW initiated active surveillance for cases of IMD, and sought information from cases about contact with the Penrith nightclub. WSPHU staff interviewed cases and contacts about exposures; initiated rifampicin prophylaxis for household and close contacts who may have been at increased risk of carrying the organism; wrote to all general practitioners in western Sydney alerting them to the outbreak and emphasising early use of antibiotics for suspected cases; and distributed fact sheets on IMD to some schools in western Sydney. On August 28, 1996, the NSW Health Department issued a press release warning that a cluster of IMD cases had been identified in western Sydney, and urged people – especially those who had been at, or who had been in contact with persons who had been at the nightclub on July 31, 1996 – to seek early treatment if they developed symptoms.

At the time of writing this outbreak report, four cases were still hospitalised. No deaths had been reported, although one case was critically ill.

Thanks to Drs Mohamed Patel, John Tapsall and David Isaacs for advice in this investigation.

MEETINGS

Laboratory Surveillance Advisory Committee (LSAC)

At its meeting in August, LSAC discussed:

- Establishing a committee consisting of representatives from Areas and the Health Department to compare public health laboratory needs and existing services, with view to designating sites to perform Statewide functions.
- A planned survey of pathology laboratories in NSW to determine the range and types of tests performed in NSW for public health purposes.

CIRCULARS

The Department released a circular on vancomycin resistant enterococci (VRE) on September 13, 1996 (Circular 96/70). The circular is an adaptation of recommendations by the United States Hospital Infection Control Practices Advisory Committee (see the July issue of the *NSW Public Health Bulletin*), and will be reissued after review by key stakeholders in NSW.

ERRATUM

In the August edition of the *NSW Public Health Bulletin*, certain graphs in the Infectious Diseases section were unclear, due to excessively dark shading. We apologise for this error.

PUBLIC HEALTH EDITORIAL STAFF

The editor of the *NSW Public Health Bulletin* is Dr Michael Frommer, Director, Centre for Research and Development, NSW Health Department. Dr Lynne Madden is production manager.

The *Bulletin* aims to provide its readers with population health data and information to motivate effective public health action. Articles, news and comments should be 1,000 words or less in length and include a summary of the key points to be made in the first paragraph. References should be set out using the Vancouver style, the full text of which can be found in *British Medical Journal* 1988; 296:401-5.

Please submit items in hard copy and on diskette, preferably using WordPerfect, to the editor, *NSW Public Health Bulletin*, Locked Mail Bag 961, North Sydney 2059. Facsimile (02) 9391 9029.

Please contact your local Public Health Unit to obtain copies of the *NSW Public Health Bulletin*. The *Bulletin* can be accessed via the Internet from the NSW Health Department's World Wide Website, at

http://www.health.nsw.gov.au/public-health/phb/phb.html

Back issues can be obtained from the Better Health Centre, Locked Mail Bag 961, North Sydney 2059. Telephone: (02) 9954 1193, Facsimile (02) 9955 5196.

TABLE 6

INFECTIOUS DISEASE NOTIFICATIONS FOR NSW IN AUGUST 1996, RECEIVED BY AREA HEALTH SERVICE

Condition	Area Health Service																	Period	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	Total for Aug	Year to date
Blood-borne and sexually transmitted																			
AIDS	1	4	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	6	186
HIV infection**	HIV data are reported bi-monthly																		
Hepatitis B – acute viral**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	23
Hepatitis B – other**	40	14	25	1	25	5	2	5	40	–	1	3	–	4	–	–	1	166*	2,992
Hepatitis C – acute viral**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	9
Hepatitis C – other**	44	18	1	–	31	15	33	30	43	19	18	15	7	9	2	13	21	319*	5,335
Hepatitis D – unspecified**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Hepatitis, acute viral (NOS)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Gonorrhoea**	1	2	2	–	–	2	–	–	17	–	–	–	1	1	1	–	–	27	331
Syphilis	6	1	6	–	6	1	1	1	11	3	–	1	1	1	2	–	–	41	523
Vector-borne																			
Arboviral infection**	–	–	–	–	–	–	1	–	–	8	2	1	1	–	2	3	–	18	1,107
Malaria**	2	2	1	–	–	1	–	–	2	–	1	–	–	–	–	2	–	11	150
Zoonoses																			
Brucellosis**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Hydatid disease	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	9
Leptospirosis**	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	22
Q fever**	–	–	–	–	–	–	–	–	–	1	1	3	2	–	2	1	1	11	197
Respiratory/other																			
Legionnaires' disease	–	2	1	–	1	–	1	–	–	–	–	–	–	–	–	–	–	5	50
Meningococcal (invasive) infection	–	1	3	9	2	1	–	3	1	–	–	1	1	–	–	1	–	23	101
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
Mycobacterial tuberculosis	1	2	–	–	–	–	2	–	–	–	–	1	–	–	–	–	–	6	235
Mycobacteria other than TB**	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	220
Vaccine-preventable																			
Adverse event after immunisation	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1	–	2	34
H.influenzae (invasive) infection	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	10
Measles	–	–	2	4	–	–	4	1	3	3	1	–	–	–	–	–	1	19	120
Mumps**	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	17
Pertussis	1	8	1	1	2	–	3	–	3	2	1	2	–	–	–	1	–	25	517
Rubella**	4	1	1	1	–	–	–	3	1	–	–	–	–	–	–	–	–	11	156
Faecal-oral																			
Cholera**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Foodborne illness (NOS)	–	8	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	8	54
Gastroenteritis (instit)	31	63	13	13	–	–	–	–	–	–	–	–	–	–	–	3	–	123	305
Hepatitis A	6	1	7	–	2	11	3	7	16	–	–	2	2	4	1	1	1	64	693
Listeriosis**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	9
Salmonellosis (NOS)**	2	6	5	3	2	2	3	1	7	–	1	2	–	1	–	–	1	36	752
Typhoid and paratyphoid**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	23

* includes acute
** Lab confirmed cases only

Abbreviations used in this Bulletin:
CSA Central Sydney Health Area, SES South Eastern Sydney Health Area, SWS South Western Sydney Health Area, WSA Western Sydney Health Area, WEN Wentworth Health Area, NSA Northern Sydney Health Area, CCA Central Coast Health Area, ILL Illawarra Health Area, HUN Hunter Health Area, NRA Northern Rivers Health Area, MNC Mid North Coast Health Area, NEA New England Health Area, MAC Macquarie Health Area, MWA Mid West Health Area, FWA Far West Health Area, GMA Greater Murray Health Area, SA Southern Health Area, OTH Interstate/Overseas, U/K Unknown, NOS Not Otherwise Stated.

Please note that the data contained in this Bulletin are provisional and subject to change because of late reports or changes in case classification. Data are tabulated where possible by area of residence and by the disease onset date and not simply the date of notification or receipt of such notification.

Controlling emerging diseases

► Continued from page 97

Folk medicines – Many home remedies, particularly popular in some communities, may contain lead. Examples that may contain as much as 75 per cent lead by weight include *greta* and *azarcon* remedies, used in Latin American communities for stomach ailments ('*empacho*'), or 'pay-loo-ah' used similarly in South-east Asian communities.

Housewares – Lead may leach from improperly fired pottery and ceramic dishes, most commonly of overseas manufacture. Lead is also found in expensive crystal; surprisingly high concentrations are found in liquor and other beverages stored in crystal decanters. Pewter houseware is also a potential source of lead exposure.

Soil and dust – Soil around heavily travelled roads or industries that use lead (e.g. smelting, mining or lead factories) can be highly contaminated. Soil around older lead-painted buildings can also have high levels of lead.

Other sources – Contaminated illicit methamphetamine and derivative drugs have been reported, occasionally resulting in common-source outbreaks of lead intoxication among injection drug users. Imported canned foods may be contaminated if lead-containing solders were improperly used to seal the can.

'Secondary transmission' – While lead intoxication is obviously not a communicable disease, household contacts of people with occupational, vocational, or other exposures may be exposed to lead dust or other compounds brought home by the affected individual, e.g. on clothing. People who are occupationally exposed to lead should shower and change clothes before leaving the workplace.

Purpose of reporting and surveillance is to:

- assess the magnitude of the lead exposure problem in NSW;
- identify and control the sources of lead exposure for people with elevated blood lead levels (EBLLs), and to identify and evaluate others who may be at risk from those sources; and
- ensure that people with lead intoxication receive proper medical management, including follow-up until their concentration of blood lead is brought down to acceptable levels.

All individuals with EBLLs ≥0.72 µMol/l (15 µg/dl) must be reported by labs to the PHU by telephone or mail.

Case follow-up

The single most important factor in managing childhood lead poisoning is reducing the child's exposure to lead. Working with the patient's medical practitioner, PHU staff will contact the patient or family to provide information about lead poisoning and referral for therapy, assess the risk to other potentially exposed people and provide counselling about how to reduce exposure. Occupationally-associated cases will be referred to Workcover for follow-up.

Acknowledgment

We thank the Oregon Health Division at the Centre for Disease Prevention and Epidemiology for assistance.

Gastroenteritis outbreak

► Continued from page 102

ingredients[3]. Since the same factors can potentially promote outbreaks caused by a variety of organisms, gastroenteritis outbreaks involving multiple organisms are not unexpected.

The major contributing factors in the outbreak described here were as follows:

- a staff member suffering from gastroenteritis continued to be involved in food handling while ill, thus spreading the organism; and
- poor personal hygiene practices among food handlers (as evidenced by findings of faecal coliforms and *E.coli* in food samples).

Use of a hand-washing basin to store food may have also increased the likelihood of contamination.

This case-control study was limited in its ability to detect differences in exposures by the relatively small numbers of cases and controls. A further limiting factor of the investigation was that food samples were taken several days after contaminated foods were consumed. Despite the absence of viral testing, the epidemiology, clinical and microbiological features of the outbreak points to a viral agent as a likely cause.

Food poisoning is usually an avoidable disease. In most cases it can be prevented simply by applying established hygienic principles in the manufacture, preparation, handling, storage and serving of food. Important measures to reduce the incidence of foodborne disease are:

- training of food service personnel in food hygiene;
- application of appropriate food hygiene legislation; and
- removal of ill workers from food-handling duties while contagious.

Acknowledgments

The authors thank the microbiology section of the NSW Health Department's Division of Analytical Laboratories, staff of the former South Sydney Public Health Unit, and Miss Angela Wong, Food Inspector, South Eastern Sydney Public Health Unit, for her assistance with many aspects of this investigation.

1. Infectious Disease Notifications 1993. *NSW Public Health Bulletin* 1995; 6(S-1):14.
2. Centers for Disease Control. Foodborne Disease Outbreaks: 5-Year Summary, 1983-1987. *MMWR* 1990; 39(SS-1):39-59.
3. Davey GR. Food Poisoning in New South Wales:1977-84. *Food Technology Australia* 1985; 37(10):453-56.

Registration

3rd NSW Public Health Network Conference

December 11-12, 1996

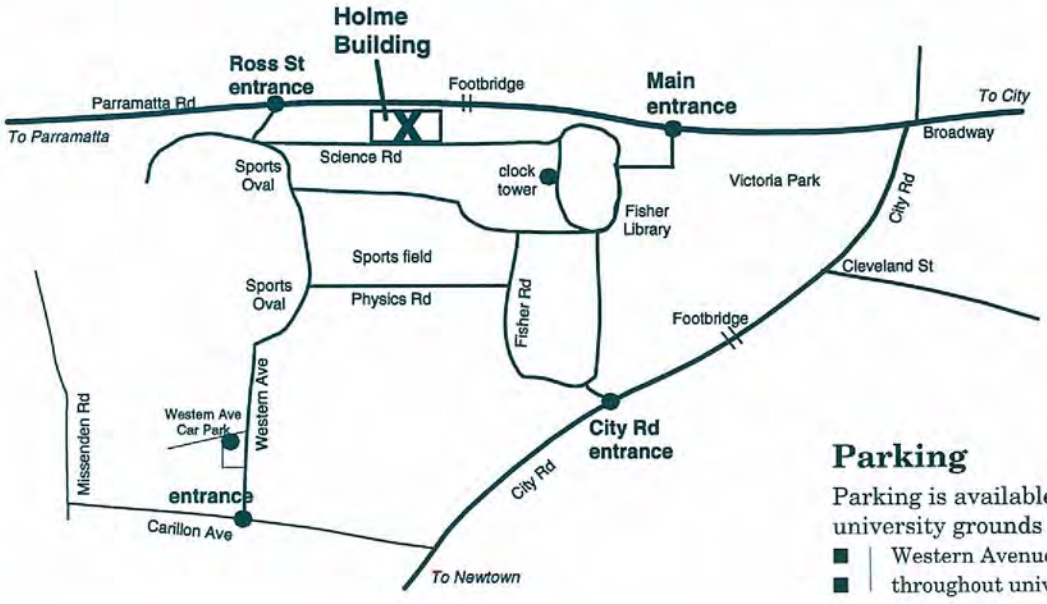
The Holme Building
University of Sydney

One Hundred Years of Public Health

Now is the time to register for the 3rd NSW Public Health Network Conference. Prominent researchers and practitioners in Public Health will contribute to a lively program that will celebrate not only the centenary of the Public Health Act in NSW but also the bicentenary of vaccine development.

The conference will explore the evolution of public health practice in NSW and examine how an expanding range of disciplines (from microbiology and environmental sciences to social epidemiology and health economics) contributes to the solution of public health problems.

Map of Sydney University grounds, showing access to Holme Building



Parking

Parking is available in the university grounds for $7 a day at:

- Western Avenue car park, and
- throughout university

ONE HUNDRED YEARS OF PUBLIC HEALTH

3rd NSW Public Health Network Conference

Registration Form

The registration fee is $80 for both days. This includes lunch, morning and afternoon tea on both days and a social gathering on the evening of December 11.

Registration will be limited to 200 people.

Delegate's name/address details

Name: .....

Position: .....

Organisation: .....

Address: .....

.....

Telephone: .....

Payment *(please tick the appropriate box)*

☐ I enclose a cheque made out to **the NSW Health Department** for $80 *(please do not send cash)*

☐ Payment will be forwarded by my employer

Dietary requirements

☐ No special dietary requirement

☐ Vegetarian

☐ Other *(please specify)*

Accommodation

Special rates for accommodation are available during the conference at the following motels, which are within walking distance of the university. Accommodation bookings are the responsibility of participants.

- Camperdown Travelodge, $110. Ph (02) 9516 1522
- University Motor Inn, $103. Ph (02) 9660 5777

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