



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

**F**ollowing 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 *Escherichia coli*** (e.g. *E. coli* O157 or O111) **infections** and **blood lead levels  $\geq 0.72 \mu\text{Mol/l}$  ( $15 \mu\text{g/dl}$ )**, 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 ( $>120^\circ\text{C}$ ) under pressure. Bacterial growth occurs only in an anaerobic environment and in conditions of low acidity (generally  $\text{pH} > 4$ ). 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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### Correspondence

Please address all correspondence and potential contributions to:

The Editor,  
NSW Public Health Bulletin,  
Public Health Division,  
NSW Health Department  
Locked Bag No 961,  
North Sydney NSW 2059  
Telephone: (02) 9391 9191  
Facsimile: (02) 9391 9029

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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<sup>6</sup>/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  $\mu\text{Mol/l}$  (50  $\mu\text{g/dl}$ ).

#### 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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**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  $\geq 0.72 \mu\text{Mol/l}$  (15  $\mu\text{g/dl}$ ) 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.

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## Gastroenteritis outbreak

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ingredients<sup>3</sup>. 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.

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