The blue book

Guidelines for the control of infectious diseases

Acknowledgements

These guidelines have been developed by the Communicable Diseases Section, Public Health Group. *The Blue Book – Guidelines for the control of infectious diseases first edition* (1996) was used as the basis for this update.

We would like to acknowledge and thank those who contributed to the development of the original guidelines including various past and present staff of the Communicable Diseases Section.

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Disclaimer

These guidelines have been prepared following consultation with experts in the field of infectious diseases and are based on information available at the time of their preparation.

Practitioners should have regard to any information on these matters which may become available subsequent to the preparation of these guidelines.

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Contents

Introduction

The blue book: guidelines for the control of infectious diseases has been published by the Communicable Diseases Section, Public Health Group, Victorian Department of Human Services, to assist public health practitioners in the prevention and control of infectious diseases.

The Department of Human Services is committed to enhancing and protecting the health and well-being of all Victorians. Our challenge, together with public health practitioners, is to reduce community risk from communicable disease in Victoria through the implementation of patient focused and population focused control strategies based on surveillance and risk assessment.

Information for each disease in this edition covers:

- Victorian statutory requirement for notification and exclusion
- infectious agent(s)
- identification
- incubation period
- public health significance and occurrence
- reservoir
- mode of transmission
- period of communicability
- susceptibility and resistance
- control measures for patients and contacts
- outbreak measures
- international measures if applicable
- sources of further information if applicable

The first edition of *The blue book: guidelines for the control of infectious diseases* (1996) was used as the basis for this second edition. As treatments change over time health care workers should consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) for up to date information. *The blue book* is not intended to replace appropriate referral or the need to seek further professional advice.

References

The following sources were used in the preparation of the guidelines:

- Therapeutic Guidelines Limited 2003, *Therapeutic guidelines – antibiotic*, version 12.
- Heymann D, 2004 *Control of communicable diseases manual*, 18th edn, American Public Health Association (the 17th edition, Chin J ed. 2000, was used in compiling *The blue book*).

Further information

Together with the references above, the following resources provide further information.

Victorian Department of Human Services

Fact sheets, surveillance reports, Departmental policies and guidelines, and online ordering of resources can be accessed at http://www.health.vic.gov.au/ideas/

- Better Health Channel, information for the public, www.betterhealth.vic.gov.au
- Clinicians Health Channel, www.health.vic.gov.au/clinicians
- Health Translations Directory, links to health information that has been translated into various different languages, www.healthtranslations.vic.gov.au
- Victorian Department of Human Services 1998, *Guidelines for the investigation of gastrointestinal illness*, http://www.health.vic.gov.au/ideas/

Other sources

- Australian Government Department of Health and Ageing 2004, *Infection control guidelines for the prevention of transmission of infectious diseases in the health care setting*, http://www.icg.health.gov.au/
- Centers for Disease Control and Prevention, Atlanta USA, www.cdc.gov
- Food Standards Australia New Zealand, http://www.foodstandards.gov.au/
- National Health and Medical Research Council 2003, *The Australian immunisation handbook*, 8th edn, www.immunise.health.gov.au
- National Health and Medical Research Council 2001, *Staying healthy in child care*, 3rd edn, Australian Government Department of Health and Aged Care, http://www.health.gov.au/nhmrc/

Notification of infectious diseases

Notifiable infectious diseases are included in Schedule 3 of the Health (Infectious Diseases) Regulations 2001 and are divided into four groups on the basis of the method of notification and the information required.

Notification forms are available from the Department:

Telephone 1300 651 160

Or print or order online at www.health.vic.gov.au/ideas

All notifications and related inquiries should be directed to:

Communicable Diseases Section
Public Health Group
Department of Human Services
Reply Paid 65937
Melbourne VIC 8060

Telephone 1300 651 160
Facsimile 1300 651 170

For urgent notifications after hours:

Contact the Duty Medical Officer via pager service 13 22 22 and quote pager number 46870

Group A diseases

Group A diseases require notification to the Department of Human Services by telephone or fax upon initial diagnosis (presumptive or confirmed) with written notification to follow within five days.

- Anthrax
- Arbovirus infections
  - Japanese encephalitis virus
  - Murray Valley encephalitis virus
- Botulism
- Cholera
- Diphtheria
- Food-borne and water-borne illness (two or more related cases).
- Haemolytic uraemic syndrome (HUS)
- Legionellosis
- Measles
- Hemophilus influenzae, type B infection (meningitis, epiglottitis, other invasive infections)
- Meningococcal infection (meningitis or meningococcaemia)
- Plague
- Poliomyelitis
- Rabies
- Severe acute respiratory syndrome (SARS)
- Smallpox
- Tularaemia
- Typhoid and paratyphoid fevers
- Viral haemorrhagic fevers
- Yellow fever

Group B diseases

Group B diseases require written notification only within five days of diagnosis.

- Arbovirus infections
- Ross River virus
- Barmah Forest virus
- Dengue virus
- Kunjin virus
- other Arbovirus infections
- Brucellosis
- *Campylobacter* infection
- Creutzfeldt-Jakob disease (CJD)
- Cryptosporidiosis
- Giardiasis
- Hepatitis A
- Hepatitis B
- Hepatitis C
- Hepatitis D
- Hepatitis E
- Hepatitis viral (not further specified)
- Influenza (laboratory confirmed)
- Leprosy
- Leptospirosis
- Listeriosis
- Lyssavirus – Australian bat lyssavirus
- Lyssavirus – other (specify)
- Malaria
- Mumps
- *Mycobacterium ulcerans*
- Pneumococcal infection (invasive)
- Psittacosis (ornithosis)
- Pertussis
- Q Fever

- Rubella (including congenital rubella)
- Salmonellosis
- Shigellosis
- Tetanus
- Tuberculosis
- variant Creutzfeldt-Jakob disease (vCJD)
- Verotoxin-producing *Escherichia coli* (VTEC)

Group C diseases

Group C diseases include the sexually transmissible diseases and should be notified using the same form. To preclude identification of the patient, only the first two letters of the given and family name of the patient are required.

- Chlamydia trachomatis genital infection
- Donovanosis
- Gonococcal infection
- Syphilis/congenital syphilis

Group D diseases

Group D diseases include HIV infection (human immunodeficiency virus) and AIDS (acquired immunodeficiency syndrome) and written notification is required within five days of confirmation of diagnosis. A separate form is used for this purpose due to the need to have national uniformity in collection of data. Copies of this form are available from the Communicable Diseases Section, telephone 1300 651 160.

- Acquired immunodeficiency syndrome (AIDS)
- Human immunodeficiency virus (HIV) infection

Laboratory notification

Around Australia and overseas it has been recognised that laboratory notification of infectious diseases should be an integral part of any disease surveillance system.

The Health (Infectious Diseases) Regulations 2001 require laboratories to notify tests indicating the probable presence of a human pathogenic organism associated with an infectious disease listed above. The notification should state the laboratory finding, the family name and given name of the patient (except for Group C diseases), the age, sex and postcode of the patient, and the name, address and telephone number of the doctor requesting the test.

In addition to the above, the Health (Infectious Diseases) Regulations 2001 require notification from laboratories of the following micro-organisms isolated or detected in food or water supplies:

- *Campylobacter* spp
- *Cryptosporidium* spp
- *Salmonella* spp
- Verotoxin producing *Escherichia coli* (VTEC)
- *Vibrio* spp
- *Giardia* cysts
- *Listeria monocytogenes*
- *Cyclospora* spp

Immediate notification must be made by telephone followed by notice in writing within 5 days specifying the micro-organism isolated or detected, date of isolation or detection, source (food or water) and any batch identification (if appropriate).

Abbreviations used

ADT	adult diphtheria tetanus vaccine
ALT	alanine aminotransferase
anti-HBc	hepatitis B core antibody
anti HBs	hepatitis B surface antibody
CF/CFT	complement fixation test
CNS	central nervous system
CSF	cerebro-spinal fluid
CT	Scan computerised tomography
DTP	diphtheria tetanus pertussis vaccine
DTPa	diphtheria tetanus acellular pertussis vaccine
dTpa	adult/adolescent formulation diphtheria tetanus acellular pertussis vaccine
EBV	Epstein-Barr virus
EEG	electroencephalogram
EIA	enzyme immunoassay
ELISA	enzyme-linked immunosorbent assay
EM	electron microscopy
FA	direct fluorescent or immunofluorescent antibody test
HAV	hepatitis A virus
HBIG	hepatitis B immune globulin
HbsAg	hepatitis B surface antigen
HbeAg	hepatitis B e antigen
HBV	hepatitis B virus
HCV	hepatitis C virus
HDV	hepatitis D virus
Hib	Haemophilus influenzae type b
HIV	human immunodeficiency virus
IG	immune globulin
IHA	indirect haemagglutination
IM	intramuscular
IV	intravenous
MDU	Microbiological Diagnostic Unit
MIF	micro immunofluorescent test
MMR	measles-mumps-rubella vaccine
MRI	magnetic resonance imaging
NHMRC	National Health and Medical Research Council
PCR	polymerase chain reaction
VIDRL	Victorian Infectious Diseases Reference Laboratory
WHO	World Health Organization

Acute bacterial conjunctivitis

Victorian statutory requirement

Infections with *Neisseria meningitidis* (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Infections with *Chlamydia trachomatis* (Group C disease) must be notified in writing within five days of diagnosis.

Infections with *Neisseria gonorrhoeae* (Group C disease) must be notified in writing within five days of diagnosis.

Other pathogens are not notifiable.

School exclusion: exclude until discharge from the eyes has ceased.

Infectious agent

Haemophilus influenzae and *Streptococcus pneumoniae* are the most common causes but *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Neisseria gonorrhoeae*, *Neisseria meningitidis* and *Chlamydia trachomatis* (trachoma serovars A-C) can occasionally be implicated.

Identification

Clinical features

The clinical syndrome ranges from mild redness of the conjunctivae to corneal infiltration and visual disturbances in neglected cases. A purulent exudate is almost always present. Trachoma should be suspected in the presence of lymphoid follicles and diffuse conjunctival inflammation or trichiasis (inturned eyelashes). Specialist ophthalmological advice should be sought in this case.

Method of diagnosis

Mild conjunctivitis is rarely investigated and is usually treated empirically. Microscopic examination of a stained smear or culture of the discharge is required to differentiate bacterial from viral or allergic conjunctivitis.

Incubation period

The incubation period is usually 24–72 hours. In the case of trachoma incubation is 5–12 days.

Public health significance and occurrence

Acute bacterial conjunctivitis is widespread throughout the world. Outbreaks of gonococcal conjunctivitis have occurred in northern and central Australia. Infection due to *Chlamydia trachomatis* (trachoma) continues to be a significant public health concern in Aboriginal communities and is a major cause of preventable blindness worldwide.

The epidemiology of acute bacterial conjunctivitis in Australia due to causes other than trachoma and gonococcal infection is not well documented. Infections are most common in children under five years of age and incidence decreases with age.

Reservoir

Humans.

Mode of transmission

Infection is transmitted via contact with the discharge from the conjunctivae or upper respiratory tract of infected persons. Neonates may acquire infection during vaginal delivery. In some areas flies have been suggested as possible vectors.

Period of communicability

It is infectious while there is discharge.

Susceptibility and resistance

Everyone is susceptible to infection and repeated attacks due to the same or different bacteria are possible. Maternal infection does not confer immunity to the child.

Control measures

Preventive measures

Preventative measures include careful treatment of affected eyes and personal hygiene, particularly hand washing.

Control of case

Conjunctivitis due to bacterial infection may be difficult to distinguish clinically from allergic or viral conjunctivitis or that due to physical irritation. Therefore, empirical antibiotic therapy is often used. Patients with significant eye pain, loss of vision or photophobia require immediate referral to an ophthalmologist.

In mild cases propamidine eye drops are the usual treatment.

In moderate and severe cases a combination of treatments may be used. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). An eye ointment may be used at bedtime. Soiled articles should be discarded or disinfected. Rigorous hand washing before and after eye examinations and toilets is important in preventing further transmission. Children should not attend school and child care settings until discharge from the eyes has ceased.

Control of contacts

With the exception of gonococcal or meningococcal conjunctivitis, contact tracing is not applicable in most situations in Victoria. Refer to the relevant sections for the management of persons in contact with these infections.

Control of environment

Dispose of contaminated articles carefully.

Outbreak measures

Public health action in an outbreak is dependent on the type of infection and the setting in which it has occurred.

Amoebiasis

Victorian statutory requirement

Notification is not required.

School exclusion: exclude until diarrhoea has ceased.

Infectious agent

Entamoeba histolytica is a protozoan parasite that exists in two forms: an infective cyst and a potentially pathogenic trophozoite. It should not be confused with the morphologically identical non-pathogenic *Entamoeba dispar*.

Identification

Clinical features

Most infections are asymptomatic but occasionally clinically important intestinal or extra-intestinal disease may result.

Intestinal disease varies from an acute form with diarrhoea which may be bloody and associated fever and abdominal discomfort (amoebic dysentery) to mild abdominal discomfort with diarrhoea containing blood or mucus alternating with periods of constipation or remission.

Intestinal amoebiasis may rarely be complicated by:

- granuloma of the large intestine
- colonic perforation and haemorrhage
- perianal ulceration.

Dissemination via the bloodstream may lead to extra-intestinal amoebiasis. This is most commonly manifested as abscess formation in the liver. This can occur less commonly in the brain or lungs.

Method of diagnosis

Diagnosis is confirmed by microscopic examination for trophozoites or cysts in:

- fresh or suitably preserved faecal specimens
- smears of aspirates or scrapings obtained by proctoscopy
- aspirates of abscesses or other tissue specimens.

Repeated stool specimens may be needed to establish a diagnosis as cysts are shed intermittently in asymptomatic and mild infections. The presence of trophozoites containing red blood cells is indicative of invasive amoebiasis.

Serology using indirect haemagglutination (IHA) and enzyme immunoassays (EIA) is useful in the diagnosis of extra-intestinal disease such as liver abscesses, when stool examination is often negative. Serology is also important in the differentiation between strains of the pathogenic *E. histolytica* and strains of the non-pathogenic *E. dispar.*

X-ray, ultrasound and CT scans are also useful in the identification of amoebic abscesses and can be considered diagnostic in the presence of a specific antibody response to *E. histolytica*.

Incubation period

The average incubation period is two to four weeks. Patients may present months to years after the initial infection.

Public health significance and occurrence

Occurrence is worldwide. Prevalence rates tend to be higher in:

- areas with poor sanitation
- institutions for the intellectually disabled
- men who have sex with men
- travellers returning from developing countries.

Amoebiasis most commonly affects young adults and is rare below the age of five years. Amoebic dysentery is very rare under the age of two years when dysentery is more commonly due to *Shigella*.

Reservoir

Humans are often asymptomatic carriers.

Mode of transmission

Amoebiasis can be transmitted by:

- ingestion of water contaminated with faeces containing amoebic cysts
- ingestion after faecal contamination of hands
- contaminated raw vegetables
- unprotected oral-anal sexual contact.

Period of communicability

Cases are infectious as long as cysts are present in the faeces. In some instances cyst excretion may persist for years.

Susceptibility and resistance

All non immune people are susceptible to infection. People with *E. dispar* do not develop symptoms. Reinfection is possible but rare.

Control measures

Preventive measures

General public health measures to prevent disease transmission focus on:

- public education on the importance of personal hygiene
- providing information to intending travellers about the risk involved in eating uncooked vegetables and fruits and drinking contaminated water
- public education about the possibility of transmitting the disease via sexual contact.

Control of case

Treating clinicians should consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and seek expert advice. Metronidazole and either diloxanide furoate or paramomycin are the usual treatment. Neither diloxanide furoate or paramomycin are registered for use in Australia - contact the NPS Therapeutic Advice and Information Service on 1300 138 677.

For amoebic liver abscess metronidazole should be continued for 14 days and specialist advice should be sought. Passage of *Entamoeba* cysts or trophozoites in the absence of acute dysenteric illness does not warrant antimicrobial therapy.

Surgical aspiration of abscesses may be necessary. Cyst eradication with diloxanide furoate may be indicated in cyst carriers. Seek expert advice.

Control of contacts

Consider faecal screening for household members and institutional contacts. Faecal screening is advised for fellow travellers of a confirmed case. Confirmed carriers should also be treated.

Control of environment

Environmental measures to control disease transmission focus on:

- protecting public water supplies from faecal contamination
- investigation of the food preparation practices of any implicated local food premises.

Outbreak measures

In the event of a cluster of cases, public health measures involve:

- confirmation of laboratory results
- undertaking an epidemiological investigation to determine source of infection and common mode of transmission
- taking appropriate measures to eliminate any common vehicles of transmission, such as contaminated food or water, to prevent further cases.

Special settings

Persons who are suspected of having acquired their infection in an institutional setting should be investigated as appropriate by the Department of Human Services.

Additional sources of information

- Centers for Disease Prevention and Control, Atlanta USA, http://www.cdc.gov/ncidod/

Anthrax

Victorian statutory requirement

Anthrax infection (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion is not required.

Infectious agent

Bacillus anthracis is a gram-positive, aerobic rod-shaped bacterium that is encapsulated, spore-forming and non-motile.

Identification

Clinical features

Anthrax is an acute bacterial disease that usually affects the skin. It may rarely involve the lungs after inhalation or the intestinal tract after ingestion.

Cutaneous anthrax

This form accounts for over 95% of anthrax cases. Lesions usually occur on exposed skin and often commence with itchiness. They pass through several stages:

- papular stage
- vesicular stage with a blister that often becomes haemorrhagic
- eschar stage that appears two to six days after the haemorrhagic vesicle dries to become a depressed black scab (malignant pustule) which may have surrounding redness and extensive oedema (swelling).

Anthrax lesions are usually painless but pain may result due to surrounding oedema. Untreated lesions can progress to involve regional lymph nodes. An overwhelming septicaemia can occur in severe cases.

Untreated cutaneous anthrax has a case fatality rate of 5–20% but death is rare with early appropriate treatment.

Pulmonary (inhalational) anthrax

This is very rare and often presents with mild and non-specific symptoms including fever, malaise and mild cough or chest pain (upper respiratory tract symptoms are rare). Early symptoms may be confused with a flu-like illness.

This is followed within three to six days by rapid onset of hypoxia, dyspnoea and high temperature, with radiological evidence of mediastinal widening. Meningitis frequently occurs.

The mortality rate approaches 100% with delayed or no treatment. Commencement of appropriate antibiotics during the prodrome significantly decreases the mortality rate.

Intestinal/oropharyngeal anthrax

These are very rare forms of anthrax in developed countries but may occur in large outbreaks in developing countries following ingestion of meat from infected animals.

In intestinal anthrax, gastro-intestinal symptoms may be followed by fever, septicaemia and death. Case fatality rates of 25–75% have been reported.

In oropharyngeal anthrax, fever, neck swelling due to lymphadenopathy, throat pain, oral ulcers and dysphagia may be followed by severe local ulcers and swelling, septicaemia and death. Case fatality rates are similar to the intestinal form.

Method of diagnosis

Laboratory confirmation of anthrax is by demonstrating the presence of *B. anthracis* in blood, lesions or discharges by direct staining of smears using Gram or other special stains, or by isolation of the organism by culture or animal inoculation. Serological and nucleic acid testing are likely to be available in the near future from reference laboratories.

Incubation period

The incubation period is typically one day for cutaneous anthrax and one to seven days for pulmonary anthrax. Evidence from mass exposures indicates incubation periods up to 60 days are possible for pulmonary anthrax, related to delayed activation of inhaled spores. The incubation is typically three to seven days for the gastrointestinal form.

Public health significance and occurrence

Anthrax is primarily a disease of herbivores. Humans usually become infected when they come into contact with infected animals or their products.

Anthrax is primarily an occupational hazard for handlers of processed hides, goat hair, bone products, wool and infected wildlife. It can also be contracted by contact with infected meat, for example in abattoir workers.

New areas of infection in livestock may develop through introducing animal feed containing bone meal. Cutaneous outbreaks sometimes occur in knackery workers and those handling pet meat. Anthrax spores can persist in the soil of certain tracts of land for years such as areas where carcasses of animals dying of anthrax are buried.

Anthrax can also be used as a bio-warfare or bio-terrorism agent, most likely spread as an aerosol. Any new case should be assessed with this possibility in mind, particularly but not exclusively in cases of pulmonary anthrax.

Reservoir

Spores may remain viable in contaminated soil for many years. Dried or processed skins and hides of infected animals may also harbour spores for years.

Mode of transmission

Cutaneous anthrax is usually introduced through a skin injury. It can occur:

- by contact with tissues of animals such as cattle, horses, pigs and others dying of the disease, or in processing after death
- by contact with contaminated hair, wool, hides or products made from them (Hide-porter's disease)
- by contact with soil associated with infected animals and contaminated bone meal used in some gardening products
- possibly by biting flies that have fed on infected animals in some parts of the world but not seen in Australia.

Pulmonary anthrax ('woolsorter's disease') can occur:

- by inhalation of aerosolised spores in industries that inadvertently may deal with contaminated tissues or products such as tanning hides, processing wool or bone products, or by accident in laboratory workers
- by intentional release of spores using a variety of aerosol devices including mail-items.

Intestinal or oropharyngeal anthrax is caused by ingestion of anthrax contaminated undercooked meat. There is no evidence of transmission through the milk of an infected animal.

The deliberate release of anthrax spores through contaminated letters in the USA in October 2001 resulted in 22 cases of anthrax, of which half were cutaneous and half were pulmonary anthrax.

Period of communicability

There is no evidence of direct spread from person to person. Articles and soil contaminated with spores may remain infective for years.

Susceptibility and resistance

Recovery is usually followed by prolonged immunity.

Control measures

Preventive measures

- Immunise high risk persons, usually laboratory workers who are liable to handle *B. anthracis*, with the cell-free vaccine giving annual boosters as recommended. Protection is likely to be greater against cutaneous exposures than pulmonary exposures. The vaccine is not currently licensed for use in the general community.
- Educate employees who are handlers of potentially infected articles in the proper care of skin abrasions.
- Ensure proper ventilation in hazardous industries and the use of protective clothing.
- Sterilise hair, wool or hides, bone meal or other feed of animal origin prior to processing.

Control of case

The following treatment advice is to be used as a guide only. Always consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and seek expert advice from an infectious diseases physician.

Cutaneous/gastrointestinal anthrax

- Ciprofloxacin, penicillin or doxycycline are the drugs of choice, usually given for 7–10 days in cutaneous anthrax. The duration of therapy for gastrointestinal anthrax is not well defined.
- If the case is associated with a bio-terrorist attack involving aerosolised anthrax where the risk is high, ciprofloxacin or doxycycline are recommended and should be given for at least 60 days.
- For patients with signs of systemic involvement, extensive oedema, or lesions on the head or neck, antibiotics should be administered intravenously, as for patients with pulmonary disease.

Pulmonary anthrax

The following recommendations were developed in the USA following experience during the deliberate release of anthrax through the postal system in 2001.

- Recommended initial treatment of pulmonary anthrax is an intravenous multi-drug regimen of either ciprofloxacin or doxycycline along with one or more agents to which the organism is typically sensitive.

- Ciprofloxacin has been recommended on the basis of in vivo (animal) findings. It should be used in preference to doxycycline in cases where meningitis is suspected because of the lack of adequate central nervous system penetration by the latter.
- Bacteremic patients are often initially treated with an empiric multi-drug regimen which provides adequate therapy for *B. anthracis* and other possible pathogens.
- After susceptibility testing and clinical improvement, the empiric regimen may be altered. A penicillin-based antibiotic, such as amoxycillin or amoxycillin/clavulanic acid may then be used to complete the course.
- Treatment should be continued for 60 days in all cases of pulmonary anthrax.

Keys to successful management appear to be early institution of antibiotics and aggressive supportive care. Chest tube drainage of the recurrent pleural effusions, which are typically hemorrhagic, often leads to dramatic clinical improvement.

Control of contacts

Although there is no person to person transmission, the Department of Human Services will trace and follow-up anyone who may have been exposed to the same source as the case, and it may be recommended that they take prophylactic antibiotics.

Control of environment

If an animal anthrax case is suspected, it should be reported to the Department of Primary Industries (DPI). Movement of animals and animal products from the farm is suspended. Appropriate samples are collected and tested at a laboratory. This can take 12–24 hours. If the case occurs on a dairy farm, the dairy factory is advised to suspend collection of milk until the case is investigated and Dairy Food Safety Victoria is advised.

If an animal anthrax case is confirmed, the affected property is quarantined, potentially exposed stock vaccinated, dead animals buried and contaminated sites disinfected. The quarantine is not released until occurrences of anthrax cases have ceased and at least six weeks have elapsed since the last round of vaccinations on the property. DPI staff will liaise with knackeries, local veterinary practitioners, the dairy industry, health authorities, local government and regional emergency services staff.

Decontamination of environments contaminated after a deliberate release of anthrax spores requires full HAZMAT decontamination by appropriately protected trained personnel using strong chlorine-based disinfectants. The risk of secondary aerosolisation is generally thought to be very low, although spores produced for bioterrorism may be deliberately prepared to increase this risk. Although the risk of anthrax can be significantly reduced by environmental decontamination measures, evidence from deliberate release of anthrax spores in other countries suggests that complete environmental decontamination of anthrax spores is extremely difficult.

Outbreak measures

A single case of anthrax should be considered an outbreak and should be managed with great urgency. If one or more patients seem to have been infected in an unusual way, such as no evidence of exposure to infected animals or their products, a deliberate release of anthrax organisms must be considered.

If a focus of infection was identified or a deliberate release of organisms is suspected, outbreak control measures would include:

- coordination with appropriate emergency services including the police force if required
- active case finding
- alerts for medical practitioners and hospitals
- release of appropriate public information
- control of contacts including field workers involved in environmental control measures
- environmental control measures.

Additional sources of information

- Australian Government Department of Health and Ageing fact sheet, http://www.health.gov.au
- Centers for Disease Control and Prevention, Atlanta USA, Public health emergency preparedness and response, http://www.bt.cdc.gov

Ascariasis
(round worm infection)

Victorian statutory requirement
Notification is not required.

School exclusion: exclude from school or children's services centre if diarrhoea is present.

Infectious agent
The infective agents are *Ascaris lumbricoides*, a large intestinal roundworm (the female measuring up to 30 cm in length) and *Ascaris suum*, a similar parasite primarily affecting pigs and occasionally humans.

Identification
Clinical features

Many people have no symptoms and the first indication of ascariasis may be the passage of a worm by the anus, mouth or nostril. The usual life cycle of the worm is described below. Migration of the larval forms of the worm can cause symptoms due to various types of pneumonitis, liver damage or allergy.

Adult worms can cause a variety of abdominal symptoms and occasionally serious complications such as intestinal obstruction or biliary disease. Ascariasis aggravates malnutrition in underdeveloped countries.

Method of diagnosis

Diagnosis can be made by the identification of eggs or the presence of adult worms in faeces. Pulmonary involvement may be confirmed by identifying ascarid larvae in the sputum or gastric washings.

A. lumbricoides are often diagnosed on radiography either as worm shaped radiolucent areas in a barium filled intestine or in cholangiograms. Significant eosinophilia is noted in only about 10% of cases.

Incubation period
The lifecycle requires four to eight weeks to complete. *Ascaris* eggs are unsegmented when passed and require a period of two or three weeks outside the host to develop to the infective stage. Mature female worms have been estimated to produce an average of 200 000 eggs per day.

Public health significance and occurrence
Ascariasis infects an estimated one billion people around the world, more than any other parasitic infection.

Roundworm infections are common in temperate or tropical regions of the world including Australia. In communities where poor sanitary conditions exist often 100% of the population will harbour the parasite.

The prevalence and intensity of infection is usually highest in children aged three to eight years. Ascaris eggs are able to survive for months in faecal matter, sewage or even in a 10% formalin solution.

Reservoir
Ascaris eggs in soil or infected humans act as reservoirs.

Mode of transmission
Transmission occurs when eggs are swallowed from soil contaminated with human faeces or from uncooked produce contaminated with soil containing infective eggs. The eggs remain viable in moist soil for several months or years.

Transmission does not occur from direct person to person contact or from fresh faeces. The eggs hatch in the small intestine and larvae pass through the intestinal wall into the blood. They then pass to the liver and heart and to the lungs. In the lungs they have a further period of development. Larvae then penetrate through the alveoli into the airways and migrate up to the pharynx to be swallowed and reach their final destination in the small intestine. This occurs about 14–20 days after egg ingestion. Larvae then mature into adult worms which mate. Females begin to lay eggs 45–60 days after initial egg ingestion.

Period of communicability
Ascariasis is communicable as long as the mature fertilised female worm lives in the intestine. The usual life span is 12 months however it has been reported to be as long as 24 months.

Susceptibility and resistance
Infection does not infer immunity.

Control measures
Preventive measures

Promote effective hand washing, particularly prior to preparing or consuming food.

Control of case

The usual treatment is albendazole, pyrantel or mebendazole. In mixed infections with *Ascaris* and other parasites it is important to initially use a drug that is effective against *Ascaris*, thereby reducing the chances of stimulating the worm into untoward activity. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

When worms obstruct the pancreatic duct or migrate up the biliary tree, surgical or endoscopic removal of the worm may be necessary.

Students with ascariasis should be excluded from school or child care if diarrhoea is present.

Control of contacts

Consider faecal screening of household members to determine if they also require treatment. No school or child care exclusion is required for contacts.

Control of environment

Environmental sources of infection should be investigated.

Outbreak measures

Not applicable.

Additional sources of information

Markell, E, John, D, Krotoski, W 1999, *Markell and Voge's medical parasitology*, 8th edn, ed. Saunders.

Barmah Forest virus disease

Arboviruses are viruses which are spread by the bite of arthropods, particularly mosquitoes. They are divided into alphaviruses and flaviviruses.

Three infective alphaviruses include Ross River, Barmah Forest and Sindbis viruses.

These all have the capacity to cause a similar disease in humans characterised by fever, joint involvement and a rash. Molecular studies of epidemiologically distinct isolates of Ross River and Sindbis viruses have shown changes in isolates from different areas (distinct topotypes). This may explain varying disease patterns which sometimes occur in certain geographic locations and the differing transmissibility of some strains by different vector mosquitoes.

Victorian statutory requirement

Barmah Forest virus infection (Group B disease) requires notification in writing within five days of diagnosis.

School exclusion is not required.

Infectious agent

Barmah Forest virus (BFV) was first isolated in 1974 from *Culex annulirostris* mosquitoes collected in the Barmah Forest near the Murray River in northern Victoria, and simultaneously from mosquitoes collected in southwest Queensland. It has also been isolated from numerous other mosquitoes including the coastal species *Ochlerotatus vigilax* (New South Wales) and *Ochlerotatus camptorhynchus* (Victoria), which enjoy a salt marsh habitat, and from the midge *Culicoides marksi* in the Northern Territory. Subsequently, BFV has been detected in most parts of mainland Australia, and serological surveys indicate that it causes widespread human infection.

Identification

Clinical features

Features include fever, arthritis, arthralgia and rash which are clinically indistinguishable from RRV disease. Like RRV disease there is a high subclinical rate of infection and a low disease rate in children. Recovery usually occurs within several weeks but lethargy, arthralgia and myalgia can persist for over six months. Outbreaks of BFV disease sometimes occur concurrently with RRV disease making diagnosis difficult.

Method of diagnosis

Serology shows a significant rise in antibody titre to the BFV. The virus may be isolated from the blood of acutely ill patients. Virological tests are necessary to distinguish BFV disease from other causes of arthritis.

Laboratory evidence requires one of the following:

- isolation of BFV from clinical material
- detection of BFV by nucleic acid testing
- a significant rise in IgG to BFV
- detection of BFV-specific IgM.

Incubation period

The incubation period appears to be seven to ten days.

Public health significance and occurrence

Since 1988, BFV disease has been reported in Western Australia, Queensland, New South Wales, the Northern Territory and Victoria. Outbreaks have been reported in Victoria throughout the Murray Valley and the Gippsland areas.

Reservoir

Like RRV, BFV disease appears after heavy rains encourage the breeding of mosquito vectors. It is not established, but it is likely, that macropods and other marsupials are the principal hosts for the virus. BFV antibodies have been found in kangaroos, cattle, horses and sheep on the south coast of New South Wales.

Mode of transmission

BFV is transmitted by mosquitoes. *Culex annulirostris* is the major vector in inland areas and *Ochlerotatus vigilax* (New South Wales) and *Ochlerotatus camptorhynchus* in southern parts of Victoria and Tasmania are the vectors in coastal regions.

Period of communicability

There is no evidence of transmission from person to person.

Susceptibility and resistance

Infection with BFV confers lifelong immunity.

Control measures

Preventive measures

BFV infection can be prevented by:

- mosquito control measures
- personal protection measures such as long sleeves and mosquito repellents
- avoidance of mosquito-prone areas and vector biting times at dusk and dawn.

Control of case

Second attacks are unknown. Treatment is symptomatic with rest advisable in the acute stages of the disease. Presently, there is no vaccine available commercially to protect against BFV disease.

Control of contacts

Unreported or undiagnosed cases should be sought in the region where the patient had been staying during the incubation period of their illness. All family members should be questioned about symptoms and evaluated serologically if necessary.

Control of environment

To reduce or prevent virus transmission, interruption of human-mosquito contact is required by:

- suppression of the vector mosquito population
- avoidance of vector contact through personal protection and education.

Outbreak measures

- Conduct a community survey to determine the species of the vector mosquito involved. Identify their breeding places and promote their elimination.
- Use mosquito repellents for persons exposed to bites because of their occupation, or other reasons.
- Identify the infection among animal reservoirs, for example kangaroos, farm and domestic animals.

International measures

Airport vector control in Australia and Papua New Guinea may be necessary to prevent spread from endemic areas to other countries where local vectors such as *Aedes polynesiensis* may transmit the disease.

Additional sources of information

- Boughton C R 1996, *Australian Arboviruses of Medical Importance, A handbook for general practitioners and other clinicians*, RACGP Services.

Botulism

Victorian statutory requirement

Clostridium botulinum infection (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion is not required.

Infectious agent

Clostridium botulinum is a spore-forming anaerobic bacillus. Several serotypes exist, however types A, B and E cause most human disease.

Identification

Clinical features

There are three forms of botulism:

- Classical botulism is a severe and often fatal infection resulting from ingestion of contaminated food. Symptoms include double vision, dysphagia and dry mouth. It can be followed by descending flaccid paralysis which may be associated with respiratory paralysis and result in death. Fever is absent unless a complicating infection occurs.
- Intestinal botulism is the most common form and usually affects infants under one year of age. It can affect adults who have altered gastrointestinal anatomy and microflora. The illness typically begins with constipation followed by lethargy, listlessness, poor feeding, ptosis, difficulty in swallowing and generalised muscle weak-ness ('floppy baby').
- Wound botulism is rare but has been seen after contamination of wounds where anaerobic conditions developed.

Method of diagnosis

Diagnosis is made by culture of *C. botulinum* or demonstration of specific toxin in serum, gastric aspirate, faeces, implicated food or wounds. Electromyography may be useful in corroborating the clinical diagnosis.

Incubation period

Classical botulism occurs within 12–36 hours (sometimes several days) after eating contaminated food. The incubation period for infant botulism is unknown due to difficulty in determining the precise time of ingestion. Shorter incubation periods are associated with more severe disease and higher case-fatality rates.

Public health significance and occurrence

Botulism is a rare disease internationally. However missed diagnoses particularly for intestinal botulism are likely due to low clinician suspicion and limited laboratory diagnostic capacity in many areas.

There have been only six cases of botulism reported in Australia between 1991 and 2003. Two of these occurred in Victoria in 2000 and 2001 (Communicable Diseases Network Australia - National Notifiable Diseases Surveillance System).

C. botulinum has been identified as a potential bioterrorist agent.

Reservoir

It is most commonly found in soil and agricultural products. Spores have been found in marine sediments and the intestinal tracts of animals, including fish.

Mode of transmission

Classical botulism is acquired by ingestion of inadequately cooked food or processed or refrigerated foods in which toxin has formed, particularly canned and alkaline foods. Most cases of wound botulism are due to ground-in soil or gravel. Several cases have been reported amongst chronic drug users.

Infant botulism arises from ingestion of spores rather than pre-formed toxin. Sources of spores include foods such as honey and dust. Honey has been described in the US literature as a source of infection but never implicated in Australia and surveys of Australian honey have failed to identify *C. botulinum*.

Period of communicability

Secondary transmission has not been documented.

Susceptibility and resistance

Everyone is susceptible to infection.

Control measures

Preventive measures

Ensure effective control of processing and preparation of commercially canned and preserved foods.

Educate people undertaking home canning and other food preservation techniques about cooking time, pressure, temperature, adequate refrigeration and storage. The absence of a bulging lid on tinned food does not preclude *C. botulinum* contamination.

Control of case

Botulism is a medical emergency. Suspected cases should immediately be referred for specialist care and trivalent botulinum antitoxin (types A, B, E) administered as soon as possible. A limited supply is available from CSL Limited. Antitoxin is not used in infant botulism due to the risk of anaphylaxis. Antibiotics do not affect the course of the disease.

For wound botulism, in addition to antitoxin the wound should be debrided or drained, and appropriate antibiotic prophylaxis against other potential infections should be administered.

Isolation or quarantine is not needed but hand washing is indicated after handling soiled nappies. Usual sanitary disposal of faeces from infant cases is acceptable.

Any implicated food should be retained for collection and investigation by public health authorities. Contaminated utensils should be cleaned by boiling or with household bleach.

Control of contacts

Those who have eaten incriminated food should be purged with emetics, gastric lavage or high enemas. Administration of polyvalent antitoxin to asymptomatic individuals should be considered carefully, assessing potential protection against the risk of sensitisation and severe reactions to horse serum.

Control of environment

Environmental health officers and food safety officers should coordinate the appropriate disposal of implicated food.

Outbreak measures

An outbreak of botulism is defined as one or more cases of disease. The immediate aim is to identify possible sources of the disease and other people possibly exposed. Recall any implicated food immediately and send samples to the Microbiological Diagnostic Unit for analysis. Take sera and faeces from cases as well as exposed but asymptomatic persons for analysis, before administration of antitoxin.

Undertake efforts to recover and test implicated foods. This should be coordinated through Food Standards Australia New Zealand (02) 6271 2222.

Brucellosis (undulant fever, Malta fever)

Victorian statutory requirement

Brucellosis (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not required.

Infectious agent

The following infectious agents cause brucellosis:

- *Brucella abortus* (biovars 1–6 and 9)
- *Brucella melitensis* (biovars 1–3)
- *Brucella suis* (biovars 1–5)
- *Brucella canis*

Identification

Clinical features

Brucellosis is a systemic bacterial disease with acute or insidious onset. Localised suppurative infections may occur. Subclinical and unrecognised infections are frequent.

Fever is the most common symptom and may be associated with a variety of other complaints.

Osteoarticular complications are common. Orchitis, epididymitis, osteomyelitis and endocarditis are less common. The case-fatality rate in untreated brucellosis is approximately 2%, mostly due to endocarditis from *B. melitensis* infections.

Method of diagnosis

Laboratory confirmation of the diagnosis is made by isolating the infectious agent from blood, bone marrow, other tissues or discharges of the patient. Serological testing for *Brucella* is useful but often difficult to interpret. Current serological tests allow a precise diagnosis in over 95% of cases, but it is necessary to combine a test (Rose Bengal and seroagglutination) detecting agglutinating antibodies (IgM, IgG and IgA) with others detecting non-agglutinating antibodies (Coombs-IgG or ELISA-IgG) developing in later stages. Except in the case of *B. canis*, where diagnosis requires tests detecting antibodies to rough-lipolysaccharide antigens.

Incubation period

The incubation period is highly variable. It is most commonly one to two months but ranges from five to sixty days.

Public health significance and occurrence

B. abortus was successfully eradicated from Australian cattle herds during the national eradication campaign in 1989. *B. suis* is still isolated occasionally from feral pigs in Queensland and represents a risk to people who hunt and butcher feral pigs. Notifications of brucellosis in Victoria are now rare and generally represent imported infections or undiagnosed chronic infections.

Brucellosis occurs worldwide. The sources of infection and responsible organism vary according to geographic area. Affected regions include the Mediterranean countries, North and East Africa, Western Africa, the Middle East, India and Central and South America.

Reservoir

The most important reservoirs for human infection are cattle, swine, goats, sheep and dogs. Infections may also occur in other wild ungulates.
B. canis has occasionally been identified in laboratory dogs.

Mode of transmission

Brucellosis can be transmitted by contact with infected tissues, blood, urine, vaginal discharges, aborted animal foetuses and especially placentae. It can also be transmitted by ingestion of raw milk and dairy products from infected animals.

Outbreaks are generally attributed to inhalation of aerosols which may occur in animal pens and stables, abattoirs and laboratories, or through ingestion of unpasteurised milk products. A small number of cases have occurred following accidental self-inoculation of the strain 19 animal *Brucella* vaccine.

Period of communicability

There is no evidence of communicability from person to person.

Susceptibility and resistance

Everyone is susceptible to infection. Severity and duration of clinical illness are subject to wide variation. Duration of acquired immunity is uncertain.

Control measures

Preventive measures

Educate the public, particularly travellers, against drinking unpasteurised milk or eating dairy products produced from such milk. Boiling milk is effective in killing the organisms when pasteurisation is not available.

Educate farmers and handlers of potentially infected animals such as feral pigs to reduce exposure and exercise care in handling placentae, discharges and foetuses. Search for and investigate livestock at risk of infection.

Control of case

Treatment should be age appropriate. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and seek expert infectious diseases.

Control of contacts

Although there is no person to person transmission of brucellosis, contact tracing is done as part of the case investigation to identify those people who have been exposed to the same implicated source of *Brucella* infection as the case. These people are advised of the early signs and symptoms of brucellosis to aid early diagnosis and treatment.

Control of environment

The Department of Primary Industries is notified of any new, non-imported case of brucellosis so that appropriate animal investigations and control measures can commence.

All incriminated products are recalled. Restriction on the distribution of unpasteurised milk and milk products is enforced.

Outbreak measures

Trace source of infection such as contaminated unpasteurised milk products and institute appropriate control measures.

Additional sources of information

- Australian Quarantine and Inspection Service, www.aqis.gov.au
- Victorian Department of Primary Industries, phone 136 186, www.dpi.vic.gov.au

Campylobacter infection

Victorian statutory requirement

Campylobacter infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude cases from child care and school until after diarrhoea has ceased.

Laboratories are required to notify *C. jejuni*, *C. coli* or *C. lari* isolated from water supplies or *C. jejuni* detected in food.

Infectious agent

The most common types of *Campylobacter* species that cause infection are *C. jejuni, C. coli, C. fetus, C. lari*, and *C. upsaliensis*.

Identification

Clinical features

Campylobacter infection may be subclinical or cause disease of variable severity. *C. jejuni* infection typically results in abdominal pain, fever and diarrhoea which may be mucopurulent or bloody. Symptoms usually last two to five days.

Campylobacter infection has been associated with rare sequelae including reactive arthritis and Guillain-Barré syndrome (polyneuritis). Human infection with *C. fetus* may cause localised abscesses or generalised sepsis particularly in immunosuppressed persons.

Method of diagnosis

Infection is diagnosed by culture of *Campylobacter* spp. from faeces, blood or other clinical specimens.

Incubation period

The incubation period is usually two to five days, with a range of one to ten days.

Public health significance and occurrence

Campylobacter infections are now the most commonly notified of the enteric pathogens in Victoria and over 14 900 cases were reported Australia-wide in 2003 (Communicable Diseases Network Australia – National Notifiable Diseases Surveillance System). The incidence of infection appears to be increasing, a trend observed internationally. All age groups are affected. The most commonly affected are children less than five years of age and young adults. Most cases in Australia appear sporadic but food and water-borne outbreaks occur and it is likely that many outbreaks are not detected.

Reservoir

Many animals, especially birds, are carriers of *Campylobacter* spp. Domestic animals are another possible source of infection.

Mode of transmission

Infection occurs most commonly by ingestion of the organism via contaminated foods, particularly raw or undercooked meats (especially poultry). Person to person transmission via the faecal-oral route is common. Infection may also occur through contact with infected animals.

Period of communicability

Cases are infectious throughout their illness. Excretion of organisms may continue for some weeks after symptoms resolve.

Susceptibility and resistance

All non immune people are susceptible to infection. Immunity to serologically related strains may follow infection and may be more common in high incidence regions.

Control measures

Preventive measures

Prevention is dependent on good personal and food hygiene. Raw meats should be cooked thoroughly and refrigerated after cooking, especially poultry. Wash utensils used to prepare raw meats and poultry in hot soapy water before using them to prepare non-cooked food such as salads. Unpasteurised milk and dairy products should not be consumed. Recognise pets as sources of infection and encourage hand washing after handling animals.

Control of case

Treatment is largely symptomatic. However, antibiotics may be indicated for severe illness or where prompt termination of faecal excretion is desired. Antibiotics are not indicated for diarrhoeal disease in which the causative pathogen is not known, except in some very severe illnesses when empirical treatment may be considered.

To prevent further transmission the importance of hand washing and personal hygiene should be stressed, particularly with respect to food preparation. Health care workers, child care workers, food handlers and children in school and child care centres should be excluded from work or school until diarrhoea has ceased. As asymptomatic excretion may persist, diligent personal hygiene is required.

Isolation is not required for hospitalised patients and standard precautions apply.

Control of contacts
The diagnosis should be considered in symptomatic contacts. Investigate related cases to identify a common source.

Control of environment
Isolation is not required for hospitalised patients and standard precautions apply.

Outbreak measures

Two or more related cases should be reported immediately, particularly in institutions. Obtain food histories and investigate other recognised vehicles of infection such as pets or farm animals to identify a common source.

Chickenpox or shingles
(varicella/herpes zoster)

Victorian statutory requirement
Notification is not required.

School exclusion differs according to case or contact status:

- cases should be excluded until full recovery or for at least five days after the first eruption appears. Some remaining scabs are not a reason for continued exclusion
- any child with an immune deficiency or receiving chemotherapy should be excluded for their own protection. Otherwise contacts are not excluded.

Infectious agent
Human herpesvirus 3 (alpha) or varicella zoster virus (VZV) is the causative agent.

Identification
Clinical features

Varicella (chickenpox)

Chickenpox generally presents with a low-grade fever, malaise and a rash. The rash is firstly maculopapular then becomes vesicular (blistered) and progresses to crusted lesions over about five days. Lesions appear in three or four crops. They are most numerous on the trunk and less so on the face, scalp, limbs and mucous membranes of the mouth. Some cases (about 5%) are subclinical or exceedingly mild in nature.

Adults tend to suffer with more severe disease than children. Rarely, the disease may be fatal.

Complications include secondary bacterial infection of the skin lesions, primary varicella pneumonia, aseptic meningitis, encephalitis and Reye's syndrome (acute encephalopathy with fatty infiltration and dysfunction of the liver).

Newborns and immunosuppressed patients are at greatly increased risk of severe chickenpox.

Herpes zoster (shingles)

Herpes zoster or shingles is characterised by a predominantly unilateral vesicular eruption within a dermatome. It is often associated with severe pain that may precede lesions by 48–72 hours. The rash lasts up to several weeks depending on severity. The rash is often more widespread and persistent in immunosuppressed patients.

Patients must be carefully evaluated to ensure that there is no eye involvement when the rash involves the ophthalmic area of the face. Specialist treatment is mandatory in this case as blindness can result.

Incidence increases with age and children under 12 are rarely affected unless immunosuppressed or infected as infants.

A debilitating complication of herpes zoster in many (especially elderly) patients is prolonged pain (post-herpetic neuralgia) which may persist for months after resolution of the skin lesions.

Method of diagnosis
Confirmation of the diagnosis is generally only required when the clinical picture is atypical. It is made by:

- isolation of the virus in cell cultures
- visualisation by electron microscopy
- serological tests for antibodies
- immunofluorescence on lesion swab or fluid
- nucleic acid testing or PCR.

Incubation period
The incubation period is from two to three weeks and is usually 14–16 days. This may be prolonged in immunosuppressed persons or following immunoglobulin administration as passive immunisation against varicella.

Public health significance and occurrence
Chickenpox is a highly contagious but generally mild disease and is endemic in the population. It becomes epidemic among susceptible individuals mainly during winter and early spring. More than 90% of cases are children under 15 years of age.

Herpes zoster (shingles) occurs in 20% of people, mostly when they are elderly due to the reactivation of latent virus from the dorsal root ganglia.

Reservoir
Humans.

Mode of transmission

Chickenpox transmission is mainly person to person by airborne respiratory droplets but also by direct contact with vesicle fluid of chickenpox cases, or contact with the vesicle fluid of patients with herpes zoster. Indirect contact occurs through articles freshly soiled by discharges from vesicles of infected persons. Scabs are not infective.

Period of communicability

It is usually communicable for one to two days (up to five days) before the onset of the rash, continuing until all the lesions are crusted. Communicability may be prolonged in patients with altered immunity.

Those with zoster are considered infectious for a week after lesions appear when they are moist.

Susceptibility and resistance

Chickenpox is highly infectious, herpes zoster much less so. Over 80% of non-immune household contacts of a case of chickenpox will become infected. Non-immune people exposed to shingles cases will develop chickenpox (not zoster) if they become infected.

Second attacks of chickenpox are rare but do occur.

Infection remains latent and can recur years later as shingles.

Patients who are at high risk of severe disease/complications if they do not have immunity include:

- infants less than one month old
- pregnant women
- immunosuppressed individuals including those with haematological malignancies, on chemotherapy, high dose steroids or with HIV infection.

Control measures

Preventive measures

In Australia, the varicella vaccine is recommended for non-immune, healthy individuals aged 12 months or older.

It provides protection against infection in 70–90% of individuals.

Non-immune individuals who should be specifically targeted for vaccination include:

- household contacts of immunosuppressed people
- health care workers
- those working with young children
- women contemplating pregnancy
- parents of young children.

Vaccination is contraindicated in immunosuppressed people and pregnant women. For further details see the current edition of the *Australian immunisation handbook* (National Health and Medical Research Council).

Immunosuppressed people and newborns should be protected from exposure. If exposure has occurred in these persons varicella zoster immune globulin (VZIG) is effective in modifying or preventing the disease if given within 96 hours of exposure. VZIG is available from the Australian Red Cross.

Control of case

Varicella (chickenpox)

In the non-hospitalised patient with a normal immune system and uncomplicated varicella, aciclovir is not recommended because the benefits are only marginal. In immunocompromised patients with severe disease and in normal patients with complications of varicella (such as pneumonitis or encephalitis) aciclovir may be used. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

General measures include:

- tepid bathing or cool compresses may help to alleviate itching
- exclude from school until fully recovered, or at least five days after eruption first appears. Some remaining scabs are not a reason for continued exclusion
- advise adults to stay away from work for the same period
- avoid contact with high risk susceptible persons.

Aspirin should never be given to children with varicella due to a strong association with the development of Reye's syndrome.

Herpes zoster (shingles)
Some antiviral medications (famciclovir, valaciclovir or aciclovir) have been shown to be effective for treatment of varicella zoster infections in patients with a rash less than 72 hours old. It gives pain relief, accelerates healing and may be of benefit in reducing the incidence of post-herpetic neuralgia. The addition of corticosteroids in selected patients may also speed resolution.

More intensive treatment is warranted in high risk patients. Consultation with an infectious diseases physician is advised. Adequate analgesia should not be forgotten.

Control of contacts
Significant contact is defined as face-to face contact for at least five minutes, being in the same room for greater than one hour or household contact.

Varicella vaccination
Vaccination may be used to prevent or attenuate illness if given to susceptible contacts within five days (preferably 72 hours) of first exposure.

Varicella zoster immunoglobulin (VZIG)
High risk susceptible contacts where vaccination is not indicated such as neonates, pregnancy and immunosuppressed persons should be offered varicella-zoster immune globulin (VZIG) within 96 hours of exposure. If vaccination is not contraindicated this should follow at least 3 months later. (See the current edition of the A*ustralian immunisation handbook*, National Health and Medical Research Council, for further details and supply.)

Contacts should not be excluded from school.

Any non-immune person admitted to hospital who has a known exposure to varicella should be isolated during days 10–21 after exposure or up to 28 days if immune globulin given to reduce the risk of spread to immunosuppressed patients.

Special settings
School
Children with chickenpox are excluded for at least five days after the rash appears. A few remaining scabs are not a reason for continued exclusion. Children with shingles can attend school if the lesions can be covered adequately however exclusion from swimming and contact sports should be advised for seven days after the rash appears.

Parents of children with immunosuppressive diseases should be advised of cases of chickenpox in the school as they may wish to voluntarily exclude their own child.

Immunosuppressed and their household contacts
Immunosuppressed people, in particular those with haematological malignancies, are at high risk of more severe infection. VZIG should be offered to these patients if exposed. Recommend vaccination of susceptible household contacts of these patients.

Pregnancy
Varicella infection during the first trimester of pregnancy confers a small risk of miscarriage. Maternal infection before 20 weeks may rarely result in the foetal varicella zoster syndrome, with the highest risk (2%) occurring at 13–20 weeks. Clinical manifestations include growth retardation, cutaneous scarring, limb hypoplasia and cortical atrophy of the brain.

Intrauterine infection can also result in herpes zoster in infancy. This occurs in less than 2% of infants. The highest risk is associated with infection in late pregnancy.

In the third trimester, maternal varicella may precipitate the onset of premature labour.

Severe maternal varicella and pneumonia at any stage of pregnancy can cause foetal death.

Susceptible pregnant women who have been exposed during pregnancy should seek specialist obstetric advice. They may be offered zoster immune globulin (VZIG) and antivirals (famciclovir, valaciclovir or aciclovir), especially where delivery is imminent.

Where chickenpox develops in pregnancy, early medical review within 24 hours of rash onset is indicated to consider treatment options.

Newborns
Where newborns develop varicella before ten days of age or when maternal chickenpox develops within seven days prior to delivery and up to 48 hours postpartum, the neonatal fatality rate is up to 30% without treatment. Treatment of mothers and of babies once born is vital.

Premature babies and infants less than one month old who develop varicella may require specific treatment. Seek expert advice.

Health care workers

On commencement at a new workplace all health care workers with an uncertain history of varicella infection should be serotested and offered immunisation if susceptible.

If a rash develops in the three weeks after immunisation, the worker should be removed from patient contact until varicella is excluded or lesions have crusted over.

If a health care worker is exposed to a confirmed case of varicella or herpes zoster they may continue working with patient contact if they have a history of previous infection or immunisation. They should be advised to report any febrile symptoms or rash developing within three weeks of exposure and then avoid patient contact until varicella is confidently excluded.

If the worker is susceptible and has been exposed, vaccination within five days of exposure is indicated. They should report rash occurring within six weeks of vaccination and avoid patient contact as above. If vaccination is refused, no patient contact should take place between days 10–21 after first exposure.

Shingles in a health care worker

Workers should not care for high risk patients until lesions have crusted over. Other patients can be cared for as long as lesions can be adequately covered.

Outbreak measures

Timely vaccination of susceptible contacts is indicated to contain an outbreak.

Additional sources of information

- Australasian Society for Infectious Diseases 2001, 'Position statement on management of varicella-zoster virus exposure and infection in pregnancy and the newborn period', *Medical Journal of Australia*, vol. 174, pp. 288–291.

Chlamydia (genital infection)

Victorian statutory requirement

Chlamydia (Group C disease) must be notified in writing within five days of diagnosis.

Specific information must be notified under the Health (Infectious Diseases) Regulations 2001. To maintain confidentiality, only the name code (first two letters of the surname followed by the first two letters of the first name) is required. A questionnaire is sent to the diagnosing doctor to collect additional information on the case that is essential for detecting disease trends and informing policy development.

Medical practitioners have a statutory obligation under the *Children and Young Person's Act 1989* to notify the Department of Human Services' Child Protection Service if they believe a child is in need of protection on the basis of sexual abuse.

Infectious agent

Chlamydia trachomatis serogroups D–K cause disease.

Identification

Clinical features

Most women with urethral or endocervical chlamydial infection are asymptomatic. Clinical manifestations may include vaginal discharge, dysuria and post-coital or intermenstrual bleeding. Less frequent manifestations include urethral syndrome (dysuria and pyuria), bartholinitis, perihepatitis and proctitis.

Complications and sequelae may result in chronic pelvic pain, infertility and ectopic pregnancy. Infections during pregnancy may cause preterm rupture of the membranes and preterm delivery. It can also cause conjunctivitis in the newborn and pneumonitis in the young infant.

The primary presentation of chlamydial infection in males is urethritis but infection may be asymptomatic. Possible sequelae and complications of male urethral infection are epididymitis, infertility, Reiter's syndrome and conjunctivitis. Receptive anal intercourse in men who have sex with men (MSM) may result in chlamydial proctitis.

Method of diagnosis

Testing individuals at high risk of chlamydial infection is recommended. High risk individuals include those with a clinical presentation suggestive of chlamydial infection, individuals attending general practitioners for testing of sexually acquired infection (STI), those attending STI and family planning clinics and gay men's health centres and partners of those already diagnosed with an STI.

Laboratory investigations currently available are:

- cell culture (only in specialised laboratories)
- antigen assays including direct immunofluorescence or enzyme immunoassay
- hybridisation assays such as the DNA probe
- amplification assays including PCR and ligase chain reaction (LCR).

The choice of test depends on the specimen type submitted, the cost of the test, the sensitivity and specificity of the test and the expertise and size of the laboratory.

Incubation period

The incubation period is poorly defined but is probably 7–14 days or longer.

Public health significance and occurrence

Infection with *C. trachomatis* has become a major public health problem because of the long term consequences of infection experienced predominantly by women. These include chronic pelvic pain, ectopic pregnancy and infertility. Rarely males may also become infertile.

Chlamydia is the most commonly notified sexually transmissible bacterial disease in Victoria. It affects both genders. The annual number of notified cases has more than doubled since the early 1990s. Approximately 75% of infections are notified from individuals aged less than 30 years.

The prevalence of chlamydial genital infections in Australia has not been comprehensively established but it has been estimated to be 2.5 –14% in STD clinic patients, 5% in family planning clients and up to 15% in commercial sex workers.

While the spontaneous cure rate has been estimated at 7.4%, immunity following infection is thought to be type-specific and only partially protective. As a result recurrent infections are common.

Risk factors for chlamydial infections include a relatively high number of sexual partners, a new sexual partner and lack of use of barrier contraceptive measures.

Endocervical *C. trachomatis* infection has also been associated with an increased risk of acquiring human immunodeficiency virus (HIV) infection and may also increase HIV infectiousness.

Reservoir

Humans.

Mode of transmission

Transmission of *C. trachomatis* occurs primarily by sexual contact. Mother to baby transmission occurs when mothers colonised with C. trachomatis infect their babies as they are born vaginally.

A high proportion of infections in women are asymptomatic resulting in untreated disease, ongoing transmission and an increased risk of sequelae.

Period of communicability

The period of communicability is unknown but may be months to years.

Susceptibility and resistance

Everyone is susceptible to infection.

Control measures

Preventive measures

Preventive measures include education about safe sex practices including use of condoms and early detection of infection by testing of those at risk.

Control of case

Azithromycin or doxycycline are used as first line antimicrobials to treat chlamydial infection. Advice on the treatment of chlamydial infections can be found in *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and the *National management guidelines for sexually transmissible infections* (Venereology Society of Victoria, 2002).

Specialist consultation should be sought for complicated or disseminated infections.

Control of contacts

Sexual partners of individuals with chlamydial infection should be examined and investigated then treated empirically.

Contact tracing assistance can be provided by the Department's partner notification officers (03) 9347 1899.

Control of environment

Not applicable.

Outbreak measures

Not applicable.

Additional sources of information

- Australian Government Department of Health and Family Services 1997, *Contact tracing manual – a practical handbook for health care providers managing people with HIV, viral hepatitis, other STDs and HIV-related tuberculosis*, Australian Government Department of Health and Family Services.
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- Venereology Society of Victoria 2002, *National management guidelines for sexually transmissible diseases*, Venereology Society of Victoria, http://www.mshc.org.au
- Victorian Department of Human Services 2001, *Chlamydia strategy for Victoria 2001–2004*, Victorian Department of Human Services.
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Chlamydophila pneumoniae

Victorian statutory requirement

Notification and school exclusion are not required.

Infectious agent

The infectious agent is *Chlamydophila pneumoniae*, an obligate intracellular bacterium (previously named *Chlamydia pneumoniae*).

Identification

Clinical features

Chlamydophila pneumoniae infection is often mild. The initial infection appears to be the most severe with reinfection often asymptomatic. A spectrum of illness from pharyngitis and sinusitis to pneumonia and bronchitis may occur. Sometimes there is a biphasic illness with initial upper respiratory tract infection symptoms which resolve and then a dry cough and low grade fever.

The organism may be an infectious precipitant of asthma and is implicated in about 5% of episodes of acute bronchitis. Cough occasionally persists for some weeks despite appropriate antibiotic therapy.

Method of diagnosis

Chest X-ray may show small infiltrates. Most cases of pneumonia are mild but the illness can be severe in otherwise debilitated patients.

Laboratory diagnosis is made with serology or culture:

- Serological diagnosis is made by detecting a four fold rise in antibody titre using microimmunofluorescence (MIF). MIF is the only serological test that can reliably differentiate chlamydial species. A single antibody titre is of little diagnostic value on its own as the seroprevalence of antibodies to *C. pneumoniae* approaches 50% in the adult population. Seroconversion may take up to eight weeks in an initial infection but it tends to occur much more quickly in reinfection (one to two weeks). False positive antibody tests can occur in the presence of a positive rheumatoid factor.
- Culture of nasopharyngeal aspirates, throat swabs or bronchial lavage fluid is possible. Swabs should be placed in chlamydia transport medium whilst other specimens can be collected in the usual containers. All samples should be kept refrigerated.

Diagnosis by PCR is available through the Victorian Infectious Diseases Reference Laboratory (VIDRL) but it is currently only being used in investigation of outbreaks of respiratory illness where conventional testing has not revealed the cause of infection.

Incubation period

The incubation period is approximately 21 days.

Public health significance

C. pneumoniae is emerging as a frequent cause of both upper and lower respiratory tract infections. It appears to be a common cause of mild pneumonia, especially in school age children. Up to 10% of cases of community-acquired pneumonia can be attributed to this organism.

Asymptomatic carriage occurs in 2–5% of the population. Only about 10% of infections result in pneumonia. Epidemics of respiratory illness can occur and these usually occur in institutional settings such as military barracks or nursing homes.

Speculation regarding the bacteria's involvement in the pathogenesis of atherosclerotic arterial disease continues. Seroepidemiologic studies have shown an association between evidence of *C. pneumoniae* infection and atherosclerosis but the significance of this is not yet established. Studies are ongoing into the effect of prophylactic antibiotic treatment on prevention of atherogenesis.

Possible links with Alzheimer's disease, arthritis and asthma are also postulated.

Reservoir

Humans.

Mode of transmission

Transmission occurs person to person via respiratory secretions.

Period of communicability

Asymptomatic carriers may be an important source of infection. Symptomatic patients can carry the bacteria in the nasopharynx for months after illness.

Susceptibility and resistance

Everyone is susceptible to infection, with the risk of clinical disease increasing in patients with a chronic medical condition. Immunosuppressed patients do not seem to be more susceptible, but older debilitated patients may develop severe disease.

Initial infection occurs in school-age children with up to 50% of the population becoming seropositive by 20 years of age. Infection does not produce complete immunity and reinfection can occur.

Control methods

Control of case

Mild to moderate infections are generally treated with roxithromycin or doxycycline. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). Patients being managed in the community should be reviewed after 24 hours to assess treatment response. Therapy may need to be continued for up to 14 days.

Isolation is not necessary, but the patient should be counselled on good respiratory hygiene, such as coughing into disposable tissues.

Special settings

Institutions

Avoid crowding in living and sleeping quarters.

Cholera

Victorian statutory requirement

Cholera (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Cholera is subject to Australian quarantine.

Infectious agent

Vibrio cholerae serogroups O1 or O139 cause cholera.

Note

Non-O1 *Vibrios*, formerly known as non-agglutinable *Vibrios* (NAG) or non-cholera *Vibrios* (NCV) are now included in the species *Vibrio cholerae*, but the reporting of Non-O1 or O139 infections as 'cholera' is inaccurate.

Most non-O1/O139 strains do not secrete enterotoxin but can cause sporadic disease. The term non-*Vibrio* cholera (NVC) refers to cases of cholera-like illness caused by organisms other than the 01 or 0139 *Vibrio* species. These infections are not notifiable.

Identification

Clinical features

Asymptomatic infection with *V. cholerae* is more frequent than clinical illness and bacteria may be present in faeces for 7–14 days. Mild cases of diarrhoea are common especially among children.

In severe cases disease is characterised by a sudden onset of symptoms with profuse painless watery (rice water) stools, occasional vomiting, rapid dehydration, acidosis and circulatory collapse. In untreated cases, death may occur in a few hours and the case fatality rate may exceed 50%.

Method of diagnosis

The diagnosis is confirmed by the isolation of *V. cholerae* serogroup O1 or O139 from faeces. A presumptive diagnosis can be made by visualisation by dark field or phase microscopy of *V. cholerae*'s characteristic motility, specifically inhibited by preservative-free serotype-specific antiserum.

Incubation period

The incubation period is from a few hours to five days. It is usually two to three days.

Public health significance and occurrence

Cholera can occur in epidemics or pandemics. In any single epidemic one particular biovar tends to predominate. Endemic cholera occurs in parts of Africa, Central Europe and Asia. Cholera appears to be increasing worldwide in both the number of cases and their distribution. Only sporadic imported cases in returned travellers occur in Victoria. *V. cholerae* O1 is established in the riverine environment in some parts of Queensland and New South Wales however human disease is rare.

Reservoir

V. cholerae is often part of the normal flora of brackish water and estuaries and can be associated with algal blooms (plankton). Humans are one of the reservoirs of the pathogenic form of *V. cholerae*.

Mode of transmission

Transmission occurs through ingestion of contaminated water and food. Sudden large outbreaks are usually caused by a contaminated water supply. Direct person to person contact is rare.

Period of communicability

Persons are infectious during the acute stage and for a few days after recovery. By the end of the first week 70% of patients are non-infectious. By the end of the third week 98% are non-infectious. Occasionally the carrier state may persist for months and chronic biliary infection with intermittent shedding of organisms may last for years.

Susceptibility and resistance

Even in severe epidemics, clinically apparent disease rarely occurs in more than two per cent of those at risk. Gastric achlorhydria increases risk of disease. There is some evidence that breastfeeding reduces the risk of infection. Infection results in a rise in antibodies with increased resistance to reinfection. Infection with an O1 strain does not confer immunity against O139 strains and vice-versa.

Control measures

Preventive measures

Travellers to endemic areas should be advised on careful food and water consumption and personal hygiene. Travellers to endemic areas should carry oral rehydration powder available from pharmacies which must be reconstituted with boiled or sterilised water.

Cholera vaccine is a heat-killed suspension of the Inaba and Ogawa serotypes of *V. cholerae* O1. It provides partial protection (approximately 50%) for three to six months. It is not routinely recommended and advice to overseas travellers should emphasise careful selection of food and water rather than

immunisation. Officially, cholera vaccination certificates are no longer required by any country or territory. Unofficially, some countries may still require such a certificate, in which case a single dose of cholera vaccine would satisfy this requirement.

Control of case

Cholera is subject to quarantine conditions under the *Commonwealth Quarantine Act 1908*.

Prompt fluid therapy with adequate volumes of electrolyte solution such as Gastrolyte is critical as life-threatening dehydration may rapidly occur. This is usually all that is required for mild to moderate illness. Patients with severe dehydration require urgent intravenous fluid. Antimicrobial agents to which the strain is sensitive shorten the duration of diarrhoea and the duration of *Vibrio* excretion.

Investigate possible sources of infection, particularly if there is no history of travel to an endemic region.

Control of contacts

Contacts should be observed for five days from the date of last exposure. This may include all fellow travellers of a case. Stool culture of any contacts with symptoms of diarrhoea and stool culture of all household contacts, even if asymptomatic, should be undertaken. Cases should also be looked for among those possibly exposed to a common source. Immunisation of contacts is not indicated.

Control of environment

Severely ill patients should be isolated in hospital, with standard precautions. Less severe cases can be managed at home. Disinfection of linen and articles used by the patient is required. Faeces and vomitus can be disposed of into the toilet without preliminary disinfection, except in areas with an inadequate sewage disposal system. Terminal cleaning of hospital rooms and equipment is required.

In cases with no history of overseas travel, urgent investigation of potentially contaminated food and water supplies is indicated.

Outbreak measures

A single case of cholera in a person with a history of no overseas travel is considered an outbreak. Initiate a thorough investigation to determine the vehicle and circumstances of transmission and plan control measures accordingly. Educate the population at risk about the need to seek appropriate treatment without delay. Adopt emergency measures to assure a safe water supply. Ensure careful supervision of food and drink preparation.

Immunisation of contacts is not indicated, even in the epidemic situation.

International measures

Reporting of cholera to the World Health Organization is mandatory under international health regulations. This will be done by the Department of Human Services through the Australian Government.

Additional sources of information

- *Quarantine Act 1908*, http://www.austlii.edu.au
- World Health Organization, http://www.who.int/csr

Creutzfeldt-Jakob disease (CJD)

Victorian statutory requirement

CJD (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

The infectious agent is a unique abnormal prion protein, designated as PrP. This protein is an insoluble, protease-resistant amyloid form of a normal cellular protein designated PrPc. PrP acts on normal prions, causing them to change into the abnormal infectious form in a cascade like manner.

Identification

Clinical features

CJD belongs to a group of rare diseases known to affect humans and animals called transmissible spongiform encephalopathies (TSE). CJD presents in humans in either a classical or a variant form.

Classical CJD

Classical CJD (cCJD) is one of four rare prion diseases that affect humans. The others are Kuru, Gerstmann-Straussler-Scheinker disease and fatal familial insomnia.

Classical CJD occurs in sporadic, familial and iatrogenic forms. Sporadic cases account for 85–90% of CJD cases and have an unknown cause. Familial cases make up 5–10% and are associated with a genetic mutation. Less than 5% are iatrogenic.

The symptoms of classical CJD usually begin at an average age of 65 years. Most cases occur between 45 and 75 years. The onset is commonly a rapidly progressing dementia, however one third of people may present with cerebellar symptoms such as dysarthria. With disease progression there may be pyramidal or spinal cord involvement, muscle atrophy or fasciculations and frequently myoclonus. Survival beyond one year is unusual with death ensuing after a median of 4.5 months.

Variant CJD

Variant CJD (vCJD) was first described in the United Kingdom in 1996. The disease is strongly linked to the consumption of cattle products infected with the prion protein that causes bovine spongiform encephalopathy (BSE), or mad cow disease. There are clinical differences between vCJD and cCJD. vCJD affects younger people (average 29 years) and the duration of illness is longer (median 14 months). Unlike cCJD, it commonly begins with psychiatric symptoms such as depression and anxiety. Involuntary movements and sensory symptoms such as pain are usually present.

Method of diagnosis

Diagnosis is suspected by the clinical presentation, disease progression and exclusion of other causes. EEG and MRI scans yield distinct results between classical and variant CJD. CSF tests assist diagnosis. Definitive diagnosis is usually made by brain biopsy or at autopsy by detection of the PrP and demonstration of the typical pathological spongiform changes in the brain. However, the diagnosis can also be confirmed by the detection of PrP in other human tissue such as tonsillar tissue by biopsy.

Incubation period

The incubation period is difficult to ascertain and varies from 15 months up to 30 years in iatrogenic cases.

Public health significance and occurrence

Since 1986 over 180 000 cases of BSE in cattle have been reported in the UK. Other countries have reported BSE in cattle imported from the UK. The rates of BSE are now decreasing. BSE has not been reported in Australian cattle. Over 100 cases of human vCJD have now been reported in Britain.

No cases of vCJD have been reported in Australia. Classical CJD continues to occur, with about 20 cases reported annually.

Reservoir

Prion disease is present in cattle (BSE), sheep, goats, mink, mule deer and elk, cats and exotic zoo animals. Transmission of variant CJD in humans has only been linked to the consumption of meat and meat products from cattle with BSE.

Infected humans with variant CJD and classical CJD are potential sources of infection for other humans by iatrogenic means.

Mode of transmission

The majority of cases of cCJD appear to occur spontaneously with no source identified. In very rare cases transmission of cCJD has occurred through iatrogenic means. This has included direct or indirect contact with brain tissue and cerebrospinal fluid.

For example, corneal or dural grafts or injections of contaminated pituitary hormone obtained from cadavers. Growth hormone is now made artificially. There is no evidence of risk to people in close casual contact with a person infected with CJD.

Variant CJD is believed to be transmitted to humans through consumption of cattle infected with BSE.

There have been no cases of vCJD linked to the receipt of infected blood products. As there is a theoretical risk of infection from blood products, blood donors are screened with respect to their possible exposure in areas affected by vCJD, particularly the United Kingdom.

Period of communicability

The central nervous system tissues are infectious during symptomatic illness of CJD.

Animal studies suggest that the lymphoid and other organs are probably infectious long before symptoms develop.

Susceptibility and resistance

Genetic mutations have been found in familial CJD. Genetic susceptibility also occurs for vCJD for humans who are homozygous for methionine at codon129 of the prion gene.

Control measures

Preventive measures

Precautionary measures instituted in Australia to reduce the risk of vCJD importation include:

- a ban on the importation of beef and beef products from the UK since 1996. Extended to other affected European countries
- monitoring and restriction by the Therapeutic Goods Administration (TGA) of the source of materials of animal origin used in the manufacture of medicines and medical devices
- a ban on blood products from people who have lived in the UK for six months or more since 1980 until 1996, commenced in 2000
- surveillance for vCJD by the Australian National CJD Registry based at the University of Melbourne
- active surveillance of cattle for BSE by the Australian Government Department of Agriculture, Fisheries and Forestry.

Control of case

There is no specific treatment except for supportive care.

Hospitalised patients should be managed using standard precautions. Tissues, surgical instruments and all wound drainage should be considered contaminated and must be inactivated. The PrP is very resistant to destruction by normal methods including standard sterilisation and because of this instruments used on CJD patients, particularly in surgery involving the brain, spine or eye, may need to be destroyed.

It is important to obtain an accurate history of travel, any previous surgical or dental procedures, and any history of exposure to human growth hormone or transplanted tissue.

If there is no travel history, obtain details of any past procedure or surgery.

Inform the Australian Government Department of Agriculture, Fisheries and Forestry to monitor Australian cattle if de novo vCJD occurs.

Control of contacts

Individuals who have may have shared a common exposure with a case or who may have been exposed to infected material from a case, such as transplanted tissue, should be counselled by a specialist infectious diseases physician.

Control of environment

All wound drainage, tissues and surgical equipment should be considered to be contaminated. See also Control of case, above. These should be terminally disposed of or inactivated (see Appendix 3).

The World Health Organization has advised that no part or product of any animal which has shown signs of a TSE should enter a human or animal food chain.

Outbreak measures

In the event of cases of vCJD being detected in Australia the Australian Government Department of Health and Ageing with the Chief Medical Officer will coordinate the national response in consultation with the State and Territory health departments, and in consultation with animal health authorities.

International measures

See Outbreak measures, above. International advisories and human and animal quarantine issues are the responsibility of various Australian and State Government departments.

Additional sources of information

- Australian Government Department of Health and Ageing 2003, '*How Australia will respond to our first case of vCJD. A guide for the public'*, www.health.gov.au
- Australian Government Department of Agriculture, Forestry and Fisheries, www.affa.gov.au
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- Creutzfeld-Jakob Disease Foundation Inc, www.cjdfoundation.org
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- Health Canada Online 2003, *Classical Creutzfeld-Jakob disease*, www.hc-sc.gc.ca
- National Institute of Neurological Disorders and Stroke 2003, *Creutzfeldt-Jakob disease fact sheet*, Bethesda USA, www.ninds.nih.gov
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- Therapeutic Goods Administration (for specific TGA measures to minimise the risk of exposure to TSEs through medicines and medical devices), www.tga.health.gov.au
- Turner, M 2001, 'Variant Creutzfeldt-Jakob disease and blood transfusion', *Current Opinion in Hematology*, vol. 8, no. 6, pp. 372–379.
- World Health Organization 2002, *Variant Creutzfeld-Jakob Disease - fact sheet 180*, www.who.int

Croup or bronchiolitis

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
Respiratory syncytial virus (RSV), parainfluenza viruses and adenoviruses are the causative agents.

Parainfluenza type 1 virus is the most common cause of croup and RSV the most common cause of bronchiolitis.

Identification
Clinical features
Disease is generally characterised by fever and one or more systemic reactions such as chills, headaches, generalised aches, malaise and anorexia. Gastrointestinal disturbances may also occur. In babies and young children general features are often not apparent and disease presents with localising signs at various sites in the respiratory tract.

Croup
Croup (laryngotracheobronchitis) has a prodrome of fever, runny nose and sore throat. Cough is also common. Inflammation at the subglottic level produces a classic high-pitched inspiratory stridor and a hoarse voice. The larger airways are narrowed by inflammation resulting in various degrees of shortness of breath and increased respiratory rate. Airway obstruction can progress with in-drawing of the intercostal spaces and the soft tissues of the neck, cyanosis and death without urgent treatment.

Bronchiolitis
A one to seven day prodrome of mild fever, coryza and cough is common with bronchiolitis. Disease can rapidly progress to deepening cough, tachypnoea, restlessness, chest wall retraction, nasal flaring and grunting. Audible wheezing is a characteristic feature. It can be accompanied by paroxysms of coughing, vomiting, dehydration, otitis media and diarrhoea.

Method of diagnosis
The diagnosis of croup and bronchiolitis is usually based on characteristic clinical findings. Serologic diagnosis can be unreliable. Identification of the specific viral agent may be accomplished by isolation in tissue culture from throat, tracheal and nasal wash specimens, or by multiplex PCR.

Incubation period
The incubation period varies from one to ten days.

Public health significance and occurrence
There is limited data on the epidemiology of croup and bronchiolitis in Australia. Croup is more common in autumn and affects children aged three months to three years. It peaks in the second year of life. Bronchiolitis is more common in winter and predominantly affects children in the first year of life.

Lower respiratory tract infections due to viral agents are significant causes of infant and childhood morbidity and mortality worldwide. Persons with underlying cardiac or pulmonary disease or compromised immune systems are at increased risk for serious complications of RSV infection, such as pneumonia and death. RSV infection among recipients of bone marrow transplants has resulted in high mortality rates. Symptomatic RSV disease can recur throughout life because of limited protective immunity induced by natural infection.

Reservoir
Humans.

Mode of transmission
RSV is transmitted via oral contact, droplet spread or by contact with hands or fomites soiled by respiratory discharges from an infected person.

Period of communicability
RSV is communicable shortly prior to and for the duration of active disease. Prolonged shedding of RSV has been documented.

Susceptibility and resistance
Everyone is susceptible to infection. Reinfection with the agents that cause croup is common but the infection is generally milder.

Control measures
Preventive measures
There is no vaccine available. Basic hygiene can help limit the spread of many diseases including croup and bronchiolitis.

Control of case

Children with these diseases should not attend school or child care centres while unwell. Investigations are generally not indicated but may be useful in outbreak situations.

Control of contacts

Investigation of contacts is not necessary but the diagnosis in other family or close contacts should be considered if they are symptomatic.

Control of environment

Not applicable.

Outbreak measures

Public health action is dependant on the setting in which the case has occurred and is based on an assessment of ongoing risk. The risk for nosocomial transmission of RSV increases during community outbreaks. Nosocomial outbreaks of RSV can be controlled by adhering to contact and respiratory precautions.

Additional sources of information

- Centers for Disease Control and Prevention, Atlanta USA, *Respiratory syncytial virus infectio*n, http://www.cdc.gov/ncidod

Cryptococcal infection (cryptococcosis)

Victorian statutory requirement

Notification and school exclusion are not required.

Infectious agent

Cryptococcus neoformans, an encapsulated yeast-like fungus. There are two principal variants:
C. neoformans var. neoformans (serotypes A & D) and *C. neoformans var. gattii* (serotypes B & C).

Identification

Clinical features

Cryptococcal infection usually presents as sub-acute or chronic meningoencephalitis with headache and altered mental state. Lung involvement may cause symptoms of lower respiratory tract infection or may be asymptomatic. Skin, bone and other organs are less frequently infected.

Method of diagnosis

Encapsulated budding forms of the fungus may be seen in the CSF, urine or pus using Indian ink staining. Cryptococcal antigens may also be detected in the CSF and serum.

The diagnosis is confirmed by culture (CSF, blood, sputum or andurine) or by histopathology (Mayer's mucicarmine staining).

Pulmonary cryptococcosis in non-HIV infected persons usually manifests as a nodule which must be distinguished from a malignancy. Malignancies may co-exist.

Incubation period

The incubation period is unknown. Pulmonary infection may precede infection in other sites by months or years.

Public health significance and occurrence

Human infection is rare in the absence of immunosuppression. Persons at increased risk of infection include patients with impaired immunity due to HIV/AIDS infection, corticosteroid therapy, lymphoma or sarcoidosis.

Cryptococcal infections occur sporadically in all parts of the world. Adults are more commonly infected with males more commonly infected than females.

Reservoir

Cryptococcus has saprophytic growth in the external environment.
C. neoformans var. neoformans occurs worldwide, frequently in association with pigeon or other bird droppings.
C. neoformans var. gattii occurs in endemic foci in the tropics and sub-tropics where certain eucalypts provide an ecological niche.

Mode of transmission

Transmission is presumed to be by inhalation.

Period of communicability

Not spread directly from person to person, nor spread between animals and people.

Susceptibility and resistance

Human resistance is presumed to be considerable given the widespread distribution of the organism and the rarity of infection. It is not known whether infection confers immunity.

Susceptibility is increased during corticosteroid therapy, immune deficiency disorders (especially AIDS), and disorders of the reticuloendothelial system, particularly Hodgkin's disease and sarcoidosis.

Control measures

Preventive measures

No vaccine is available. Some patients may require maintenance antibiotics to prevent repeat infections (see below).

Control of case

Clinicians should consider referral to a specialist centre for treatment. Typical treatment often involves amphotericin or flucytosine.

Patients with HIV/AIDS may require continuing maintenance therapy (secondary prophylaxis), typically fluconazole orally daily.

Control of contacts

No action required.

Control of environment

Large accumulations of bird droppings should be removed after first being wetted or chemically disinfected to reduce aerosolisation.

Outbreak measures

Case clusters are rare. Environmental investigations focus on potential reservoirs of infection such as bird droppings, although a definitive source is rarely found.

Special settings

Where nosocomial transmission is suspected in single cases or clusters, the risk of further infections should be reduced through appropriate control of the external environment, with investigation of the internal environment within facilities as appropriate.

Cryptosporidiosis

Victorian statutory requirement

Cryptosporidial infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude cases from child care and school until diarrhoea has ceased or until a medical certificate of recovery is produced.

Notification is required if *Cryptosporidium* spp are isolated from water supplies.

Infectious agent

Cryptosporidium parvum is a coccidian protozoon.

Identification

Clinical features

Cryptosporidiosis is a parasitic infection that commonly presents as gastroenteritis. Enteric symptoms usually include watery diarrhoea associated with cramping abdominal pain, bloating, vomiting and fever. The disease is usually mild and self-limiting. In persons with impaired immunity, particularly those with AIDS, it may be prolonged and life-threatening. Cryptosporidiosis infection may less commonly involve the lungs (bronchitis or pneumonia), gall bladder (cholecystitis) or pancreas (pancreatitis). Symptoms usually last four to twenty-one days.

Method of diagnosis

As tests for *Cryptosporidium* are not routinely conducted in some facilities laboratories should be informed if cryptosporidiosis is suspected.

Oocysts may be identified by microscopy of faecal smears treated with a modified acid-fast stain. A monoclonal antibody test is useful for detecting oocysts in faecal and environmental samples.

Oocyst excretion is most intense during the first days of illness. Oocysts are rarely recovered from solid faeces.

ELISA assays have been developed for the detection of antibodies but these are not in routine use.

Incubation period

The incubation period is estimated to be one to twelve days, with an average of seven days.

Public health significance and occurrence

Cryptosporidiosis occurs worldwide. Young children, the families of infected persons, men who have sex with men, travelers, health care workers and people in close contact with farm animals comprise most reported cases. Substantial outbreaks linked to public water supplies have been reported in the United States. Multiple outbreaks associated with public swimming pools and spas have been reported in Australia and worldwide. The risk of infection for Melbourne residents has been greater for people exposed to public swimming pools and household contacts of infected persons.

Reservoir

Reservoirs include humans, cattle and other domestic animals.

Mode of transmission

Transmission occurs by the faecal-oral route (person to person and animal to person), and via ingestion of contaminated foods and water.

Period of communicability

Cases may be infectious for as long as oocysts are excreted in the stool. Asymptomatic excretion may persist for several weeks after symptoms resolve.

Under suitable conditions oocysts may survive in soil and be infective for up to six months.

Susceptibility and resistance

Everyone is susceptible to infection. People with normal immune systems usually have asymptomatic or self-limited gastrointestinal disease.

People with impaired immunity may experience prolonged illness.

Control measures

Preventive measures

Encourage good personal hygiene, particularly following contact with animals or infected persons. Particular attention to hand washing is required during calving seasons on cattle properties.

Filter or boil contaminated drinking water, as chemical disinfectants such as chlorine are not effective against oocysts at the concentrations used in water treatment.

Control of case

Treatment is symptomatic and particularly involves rehydration. Antibiotics are not indicated.

Exclude symptomatic persons from food handling, direct care of hospitalised and institutionalised patients and care of children in child care centres until asymptomatic.

Disinfect soiled articles.

As oocyst excretion may persist for extended periods it is not advisable for adults to swim in public pools for a period of seven days and children for a period of four weeks after their diarrhoea has ceased. Showering before swimming is recommended at all times.

Control of contacts

The diagnosis should be considered in symptomatic contacts.

Control of environment

Faecal contamination of pools requires prompt action by the pool operator including disinfection, but oocysts resist standard chlorination. Refer to the Department of Human Services' *Pool operators' handbook*.

Outbreak measures

An outbreak investigation is required if two or more cases are clustered in a geographic area or institution. Investigate potential common sources such as contact with farm animals, consumption of contaminated water or unpasteurised milk or exposure to a common recreational swimming area.

The Department of Human Services considers cases may be linked to a public swimming facility if two or more people with Cryptosporidium infection (confirmed by a pathology laboratory) have used the same pool within two weeks of their illness. In this situation pool owners may need to close the affected swimming pool until it has been treated and superchlorinated with at least 14 mg/L free chlorine for at least 12 hours.

It is important to ensure that the total chlorine level in a treated pool is less than 8 mg/L before re-opening it to the public. If an outbreak is particularly large, the Department may request additional steps to be undertaken.

Additional sources of information

- Victorian Department of Human Services 2000, *Pool operators' handbook*, http://www.health.vic.gov.au/environment

Cytomegalovirus infection

Victorian statutory requirement

Notification and school exclusion are not required.

Infectious agent

Cytomegalovirus (CMV) also designated as Human herpesvirus 5, is a member of the subfamily betaherpesvirus of the family herpesviridae. Other members of the herpesvirus group include herpes simplex virus types 1 and 2, varicella zoster virus (which causes chickenpox), and Epstein-Barr virus (which causes infectious mononucleosis/glandular fever). These viruses share a characteristic ability to remain dormant within the body over a long period.

Identification

Clinical features

Primary CMV infection of children and adults may cause a mononucleosis syndrome clinically indistinguishable from that caused by the Epstein-Barr virus (glandular fever). Features include fever, lymphadenopathy and mild hepatitis. More rare features include anaemia, thrombocytopaenia, pneumonitis, meningoencephalitis and Guillain-Barrè syndrome. Many infections are asymptomatic, particularly those in young children.

Pregnancy

Healthy pregnant women are not at special risk for disease from CMV infection but between 1% and 5% are infected for the first time during their pregnancy. Many women will already have been exposed to CMV and so are not at risk of a new infection during their pregnancy.

When infected with CMV, most pregnant women have no symptoms while a very few have a disease resembling mononucleosis. However, for those women who are infected for the first time during their pregnancy, one in three will pass the CMV infection on to their developing unborn child.

CMV remains the most important cause of congenital viral infections in Australia. For infants who are infected by their mothers before birth, two potential problems exist:

Generalised infection may occur with symptoms ranging from moderate enlargement of the liver and spleen with jaundice, to a fatal illness. With supportive treatment most infants with CMV disease survive. However 80% to 90% will have complications within the first few years of life that may include hearing loss, vision impairment and varying degrees of mental retardation.

Another 5% to 10% of infants who are infected but without symptoms at birth will subsequently have varying degrees of hearing and mental or coordination problems.

Immunosuppression

Other people at increased risk of severe infection include patients with impaired immunity due to HIV/AIDS infection, corticosteroid therapy, lymphoma or sarcoidosis. In these people, disease is usually due to reactivation of previous infection. Manifestations include sight-threatening retinitis, pneumonitis, gastrointestinal ulceration and inflammation, and neurological disease particularly affecting the brain and spinal cord.

Method of diagnosis

CMV may be detected by virus isolation or PCR, usually from urine. Virus may also be detected in saliva, breast milk, semen and cervical secretions during primary and reactivated infection.

CMV causes typical 'cytomegalic' cells in tissue culture and characteristic histologic features in affected tissues. CMV antigens may be detected using rapid antigen tests. Serology is also available where recent infection is suggested by the identification of CMV IgM or a fourfold or greater rise in serum IgG titres to CMV from paired sera.

Isolation of CMV from culture does not necessarily imply recent infection as CMV may be excreted for months to years following infection. Positive results for CMV from laboratory investigations should always be considered with clinical findings.

Incubation period

The incubation period of sporadic cases of CMV usually cannot be determined. Perinatal infection develops three to twelve weeks after delivery. In adults, illness usually occurs three to eight weeks after blood transfusion and between four weeks and four months after organ transplantation.

Public health significance and occurrence

Although infection with CMV is very common around the world, symptomatic disease is rare. The risk of severe or complicated CMV infection is increased in some groups including:

- the developing infant during pregnancy
- people with immunosuppression such as organ transplant recipients, people infected with human immunodeficiency virus (HIV) and those being treated for cancer.

Serosurveys of adult populations worldwide have shown wide-spread evidence of previous CMV infection with seropositivity rates ranging from 40% in highly developed countries to 100% in developing countries.

The incidence peaks during the perinatal period with a secondary peak among young adults in areas where perinatal infection is less common.

Reservoir

Humans.

Mode of transmission

CMV is excreted in urine, saliva, breast milk, cervical secretions and semen during primary and reactivated infections. CMV may be transmitted by:

- transplacental infection of the foetus of a mother with primary or reactivated infection
- perinatal infection of neonates via infective maternal cervical secretions or breast milk
- blood transfusion or organ transplantation
- intimate exposure by mucosal contact with infective tissues, secretions or excretions.

CMV is not readily spread by casual contact but requires prolonged, intimate exposure for transmission. This can occur in settings such as child care centres where toddlers shed the virus in saliva and urine and thereby spread the infection among them.

Period of communicability

CMV may be shed in the bodily fluids of any previously infected person, and thus may be found in urine, saliva, blood, tears, semen and breast milk. The shedding of virus may take place intermittently for months to years after primary infection without any detectable signs, and without causing symptoms.

Neonatal infection in particular is associated with prolonged excretion. The period of excretion seems to be shorter in adults.

Susceptibility and resistance

Once a person becomes infected, the virus may remain dormant in their body for life (latent infection). Recurrent disease rarely occurs unless the person's immune system is suppressed due to therapeutic drugs or disease.

Control measures

Preventive measures

There is no vaccine available to protect against CMV infection.

Public health measures focus on reducing the risk of CMV transmission to pregnant women, women of childbearing age and other people at risk of more serious infections.

Women of childbearing age working in hospitals (especially obstetric and paediatric wards), child care centres and preschools should practice strict infection control precautions and regard all body fluids as potentially infectious.

The CMV status of blood and organ donors should be matched to that of recipients wherever possible. If CMV seropositive donors must be used for CMV seronegative recipients, prophylactic use of hyperimmune immunoglobulin or antiviral drugs may be considered.

Control of case

There is no specific treatment recommended for primary CMV infection of normal hosts.

Immunosuppressed persons with CMV retinitis or pneumonitis are usually treated in specialist centres with ganciclovir, foscarnet, or cidofovir/probenecid. These drugs may also be of benefit for other complications of CMV infection.

Control of contacts

None required because of the high prevalence of asymptomatic virus shedders in the community.

Control of environment

Not applicable.

Outbreak measures

Not applicable.

Additional sources of information

- Centers for Disease Control and Prevention, Atlanta USA, *Cytomegalovirus infection*, http://www.cdc.gov/ncidod

Dengue virus disease

Victorian statutory requirement

Dengue virus infection (Group B disease) requires notification within five days of diagnosis.

School exclusion: case should be isolated until the fever subsides to prevent further mosquito bites.

Infectious agent

Dengue virus (DENV) has four related but distinct serotypes: 1, 2, 3 and 4.

Dengue virus has been recognised since the latter part of the 18th century as causing epidemics in tropical and subtropical parts throughout the world. Dengue was first recognised in Townsville late in the 19th century and early in the 20th century. Outbreaks occurred in an area from the coast of Western Australia to the Northern Territory and down through high rainfall areas of Queensland and New South Wales. At that time *Aedes aegypti* mosquitoes were widely distributed in northern Australia and occurred as far south as the Victorian border in eastern Australia and south of Perth in Western Australia. By the 1970s *Aedes aegypti* were restricted to a small area of northern Queensland. Epidemic dengue returned to north Queensland in 1981–82. Other outbreaks occurred there in the 1990s, a time when *Aedes aegypti* mosquitoes were spreading westwards from Queensland to the Northern Territory border and towards the New South Wales border.

Identification

Clinical features

Dengue fever (break bone fever)

Dengue fever classically presents as an acute febrile illness of sudden onset. It is extremely debilitating with fever lasting three to five days, myalgia (particularly backache), arthralgia, retro-orbital pain, anorexia, gastrointestinal disturbance, rash and increased vascular permeability. There is a high subclinical rate of milder disease in children compared to adults and a low fatality rate. Recovery from infection with one serotype of the dengue virus results in homologous immunity but does not provide protection against infection with other serotypes.

Dengue haemorrhagic fever

Dengue haemorrhagic fever (DHF) is a severe complication of dengue virus infection. It occurs mainly in children and is characterised by abrupt onset of fever, haemorrhagic phenomena and thrombocytopaenia. In its severest form it may result in shock (dengue shock syndrome [DSS]), which has a high fatality rate. The rate of death from DHF without DSS is usually quoted at 1–5%. This is believed to be caused by immune enhancement when a person with dengue antibodies due to a previous infection is subsequently infected by a dengue virus of a different serotype.

Method of diagnosis

Dengue virus infection is diagnosed by a significant rise in antibodies to the dengue virus serotype.

Laboratory evidence requires one of the following:

- isolation of dengue virus from clinical material
- detection of dengue viral RNA in clinical material
- a significant rise in the level of dengue virus specific IgG proven by neutralisation or another specific test
- dengue virus specific IgM in the CSF in the absence of IgM to Murray Valley encephalitis, Kunjin or Japanese encephalitis viruses
- dengue virus specific IgM in serum, except in north Queensland. In north Queensland dengue virus specific IgM in serum is acceptable evidence only when this occurs during a proven outbreak.

Confirmation of laboratory results by a second arbovirus reference laboratory is required if the case occurs in previously unaffected areas of Australia. North Queensland is currently the only area with the potential for indigenous (epidemic) dengue fever in Australia.

Incubation period

The incubation period is usually short but varies from three to fourteen days.

Public health significance and occurrence

Dengue is not an endemic disease in Australia and the outbreaks which have occurred have been due to importations of the virus by a viraemic tourist or returning resident. It is important to

rapidly diagnose the disease in returning residents and tourists to prevent local spread in receptive areas. Spread or introduction of *Aedes aegypti* from its present distribution in Queensland must be closely monitored.

Of great concern has been the repeated detection of imported *Aedes albopictus* mosquitoes into various parts of Australia dating from 1975 in Townsville. Since then it has been detected at various times and in various carriers on ships, in machinery and in car tyres in South Australia, Perth and Darwin. In 1998 it was trapped on a wharf in Cairns and similarly at West Melbourne in 2002. Preventing the introduction and establishment of *Aedes albopictus* remains a high priority because this mosquito has the potential to spread widely over Australia including southern areas. It can also transmit dengue and other arboviruses.

Reservoir

Humans are the only vertebrate hosts of the virus. There is a jungle cycle between monkeys and mosquitoes, but this plays no role in human disease.

Mode of transmission

Dengue is transmitted by the bite of an infected mosquito, particularly *Aedes aegypti*. This was first recognised by workers in Queensland early in the 20th century. *Aedes aegypti* breeds in fresh water and particularly in man made containers such as old tyres, pot plant holders, buckets and tree hollows in urban areas. *Aedes albopictus* is a mosquito common in South East Asia and Papua New Guinea and can also be an important vector. Other *Aedes* species are involved in the enzootic monkey cycle.

Period of communicability

There is no evidence of person to person transmission.

Susceptibility and resistance

Infection with a serotype of dengue virus does not necessarily confer immunity.

Control measures

Preventive measures

There are effective vaccines available against a number of the dengue virus serotypes.

Dengue fever can be prevented by:

- mosquito control measures
- personal protection measures such as long sleeves and mosquito repellents
- avoidance of mosquito-prone areas.

Control of case

Isolate the patient and prevent mosquito access until fever subsides.

Investigate the source of infection.

Control of contacts

Not applicable.

Control of environment

- Search for and eliminate breeding sites of *Aedes aegypti* in the urban area.
- Use mosquito repellents, mosquito nets and other methods of personal protection.
- Control *Aedes aegypti* near airports.
- Prevent importation of new vectors, for example *Aedes albopictus.*

Outbreak measures

Not applicable.

Diphtheria

Victorian statutory requirement

Diphtheria (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion is relevant for cases and contacts:

- Cases should be excluded until a medical certificate of recovery is received following at least two negative throat swabs. The first should be 24 hours or more after finishing a course of antibiotics and the second 48 hours later.
- Contacts should be excluded until cleared to return by the Department of Human Services.

Infectious agent

Corynebacterium diphtheriae of the *gravis*, *mitis* or *intermedius* biotypes is an aerobic gram-positive bacillus. Toxin production results when the bacteria are infected by a bacteriophage containing the diphtheria toxin gene *tox*.

Identification

Clinical features

Diphtheria is an acute bacterial infection caused by toxigenic strains of *Corynebacterium diphtheriae*. It primarily affects the tonsils, pharynx, nose and larynx. Other mucous membranes, skin, and rarely the vagina or conjunctivae can also be involved. The toxin causes local tissue destruction and membrane formation.

The characteristic lesion in the throat is an adherent greyish-white membrane that first occurs on the tonsils, but may spread up onto the palate and involve the pharynx and result in respiratory obstruction.

The onset is insidious with early symptoms of malaise, sore throat, anorexia and low-grade fever. Patients with severe pharyngeal disease may develop neck swelling giving a characteristic 'bull neck appearance'. Systemic absorption of the toxin can result in neuropathy and cardiomyopathy, resulting in early death or later neurological complications.

Laryngeal diphtheria can present as a slowly progressive croup which can result in death if the airway obstruction is not relieved.

Non-toxigenic strains of *C. diphtheriae* rarely cause local lesions but may cause infective endocarditis.

Cutaneous diphtheria presents with lesions of variable appearance but which may resemble impetigo.

Non-cutaneous diphtheria has a case fatality rate of 5–10% with higher rates in children under five years and adults over 40 years of age.

Method of diagnosis

Diagnosis is usually based on observation of the classical greyish-white membrane overlying the tonsils or pharynx.

Specimens for *C. diphtheriae* culture should be obtained from the nose and throat and from any other suspicious lesions. Swabs should be obtained from the pharyngeal membrane, or a portion of the membrane itself could be submitted for culture.

Selective medium is required to culture *C. diphtheriae* so the testing laboratory should be notified that the disease is clinically suspected. All isolates should be sent to a public health reference laboratory for *C. diphtheriae* toxin detection by polymerase chain reaction (PCR).

Incubation period

The incubation period is two to five days but occasionally longer.

Public health significance and occurrence

Diphtheria occurs worldwide and is more prevalent in winter months in temperate zones. Illness is now rare in highly immunised communities.

An epidemic began in the Russian Federation in 1990 involving all of the countries of the former Soviet Union and Mongolia. The epidemic has declined after a peak in 1995 but was responsible for over 140 000 cases and over 4000 deaths. Over 70% of cases were aged 15 years or older.

Diphtheria is now a very rare infection in Australia but may occur in unimmunised people who are recent travellers, or their contacts. The last reported case in

Victoria occurred in 1991. Importation of the infection from other affected countries remains a concern in Australia with the potential to affect unimmunised children and adults as well as adults with waning immunity post-vaccination.

Reservoir

Humans are the usual reservoir and carriers are usually asymptomatic.

Mode of transmission

Transmission is droplet spread from the respiratory tract. More rarely transmission can occur from contact with articles soiled with discharges from infected lesions.

Period of communicability

Transmission may occur as long as virulent bacilli are present in discharges and lesions. The time is variable but is usually two weeks or less and seldom more than four weeks without antibiotics. Appropriate antibiotic therapy promptly terminates shedding. The rare chronic carrier may shed organisms for six months or more.

Susceptibility and resistance

Infants born of immune mothers are relatively immune, but passive immunity is usually lost by six months of age.

Lifelong immunity is usually, but not always, acquired after disease or inapparent infection.

A primary course of toxoid vaccination provides long lasting but not lifelong immunity. Vaccinated individuals may become colonised by *C. diphtheriae* in the nasopharynx while still being protected from clinical disease.

Control measures

Preventive measures

Diphtheria vaccination is part of the Australian Standard Vaccination Schedule. Primary vaccination is achieved with three doses of a diphtheria-toxoid containing vaccine at two, four and six months of age. Booster doses are currently recommended. They are given as DTPa at four years of age and as adult/adolescent formulation DTPa at 15 to 17 years of age. Prior to the 8th birthday, DTP-containing vaccines should be used. After the eighth birthday, smaller doses of toxoid (adult/adolescent formulation DTPa or DT-containing vaccines) should be given. Refer to the current edition of *The Australian immunisation handbook* (National Health and Medical Research Council).

Adults who have been fully vaccinated in the past should receive a booster dose of adult tetanus-diphtheria vaccine (Td, 'ADT') at the age of 50 years unless a booster dose has been documented in the previous ten years.

For 'catch-up' diphtheria immunisation schedules for children and adults refer to the current edition of *The Australian immunisation handbook* (National Health and Medical Research Council).

Control of case

The management of cases involves diphtheria antitoxin, antibiotic therapy and infection control. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). Specialist infectious diseases advice should always be sought on clinical suspicion of a case of diphtheria.

Diphtheria antitoxin should be given if there is strong clinical suspicion, immediately after specimens are taken and without waiting for laboratory confirmation. The dosage will depend on severity which is assessed by the extent of the pharyngeal membrane and duration of disease. Antitoxin can be obtained from CSL Limited. Patients must be tested for hypersensitivity prior to administration. Co-administration of antitoxin with corticosteroids may be recommended for patients with hypersensitivity to antitoxin.

Parenteral antibiotic treatment is usually required initially and can be either erythromycin or benzathine penicillin. These can be substituted for by equivalent oral formulations once the patient can swallow comfortably. These should be continued to complete a total of 14 days of treatment.

Natural infection with diphtheria does not guarantee ongoing immunity. The patient should begin or complete active immunisation with an age-appropriate diphtheria toxoid-containing vaccine during convalescence.

Use standard precautions with additional respiratory precautions for pharyngeal diphtheria and standard precautions with additional contact precautions for cutaneous diphtheria, until the case is shown to be clear of carriage.

The disease is usually not highly contagious after 48 hours of antibiotic therapy.

Control of contacts

All close contacts of the case including all household contacts and other persons directly exposed to oral secretions from the case, should have swabs taken for culture from their throat and nose.

A prophylactic course of seven days of oral erythromycin or a single dose of procaine penicillin IM is recommended for close contacts. Such contacts should also be kept under surveillance for seven days, regardless of their immunisation status.

Contacts are excluded from childcare and school until cleared by the Department of Human Services. Other contacts are advised to exclude themselves from work and particularly food handling, until bacteriologic examination shows they are not carriers of the organism.

Unvaccinated contacts should be commenced on their primary course of vaccine.

Vaccinated contacts should be given a booster injection of vaccine if more than five years have elapsed since their last dose.

For close contacts identified as diphtheria carriers:

- ensure that prophylactic antibiotic therapy has been given (as above for close contacts)
- exclude until two negative swabs, the first not less than 24 hours after finishing the antibiotics and the other 48 hours later.

If either of the repeat cultures is positive then an additional ten day course of erythromycin or penicillin is recommended, followed by two repeat cultures.

Extensive swabbing to detect diphtheria carriers apart from close contacts is not recommended.

Outbreak measures

Outbreaks of diphtheria require immunising the largest possible proportion of the population involved, emphasising the need for protection of infants and preschool children. In outbreaks amongst adults immunise groups that are most affected and at high risk. Repeat immunisations may be recommended after one month.

Outbreak investigations involve enhanced case surveillance with laboratory confirmation of all suspected cases, as well as the identification and appropriate management of close contacts and asymptomatic carriers (see Control of contacts, above).

Additional sources of information

- Gidding, HF, Burgess, MA, Gilbert, GL 2000, 'Diphtheria in Australia, recent trends and future prevention strategies', *Communicable Diseases Intelligence*, vol. 24, no. 6.

Donovanosis

Victorian statutory requirement

Donovanosis (Group C disease) requires written notification within five days of the initial diagnosis.

Specific information must be notified under the Health (Infectious Diseases) Regulations 2001. To maintain confidentiality, only the name code (first two letters of the surname followed by the first two letters of the first name) is required.

Medical practitioners have a statutory obligation under the *Children and Young Person's Act 1989* to notify the Department of Human Services Child Protection Service if they believe that a child is in need of protection on the basis of sexual abuse.

Infectious agent

Previously known as *Calymmatobacterium granulomatis*, a gram-negative bacillus, the causative agents is now named *Klebsiella granulomatis*.

Identification

Clinical features

Donovanosis is a chronic, progressively destructive infection which affects the skin and mucous membranes of the external genitalia, inguinal and anal regions. Disseminated disease is uncommon but may be life threatening and so should be considered in patients from endemic areas. It presents initially as raised, 'beefy' nodules or sores. Lesions may extend peripherally with characteristic rolled edges. Local spread to pelvic and abdominal structures occurs and dissemination to distant sites can also occur.

Method of diagnosis

The diagnosis is confirmed by demonstrating 'Donovan bodies' in Wright or Giemsa-stained smears of granulation tissue or by histological examination of biopsy specimens.

Incubation period

The incubation period is weeks to months.

Public health significance and occurrence

Donovanosis is rare in industrialised countries but endemic in some tropical and subtropical countries and areas including northern Australia.

There have been no cases of donovanosis notified in Victoria since at least 1992.

Reservoir

Humans.

Mode of transmission

Transmission is primarily sexual. It is possible that some cases are transmitted non-sexually.

Period of communicability

The period of communicability is unknown but may be months to years.

Susceptibility and resistance

Everyone is susceptible to infection.

Control measures

Preventive measures

Preventative measures include education about safe sex practices including use of condoms and early detection of infection by testing of people at risk.

Control of case

First-line treatment for donovanosis is azithromycin. Treatment should be directly observed. Follow-up is important as resolution may be slow and recurrence may occur.

Control of contacts

Sexual contacts should be examined for possible infection. The likelihood of transmission per act of unprotected intercourse is considered to be low and the likelihood of a long term partner being infected is low to moderate. Contacts dating back weeks or months should be traced according to the sexual history.

Control of environment

Not applicable.

Outbreak measures

Not applicable.

Additional sources of information

- Australian Government Department of Health and Family Services 1998, *Contact tracing manual – a practical handbook for health care providers managing people with HIV, viral hepatitis, other STDs and HIV-related tuberculosis*.
- Carter, JS, Bowden, FJ, Bastian, I, Myers, GM, Sriprakash, KS, Kemp, DJ 1999, 'Phylogenetic evidence for reclassification of *Calymmatobacterium granulomatis* as *Klebsiella granulomatis*', *International Journal of Systemic Bacteriology*, vol. 49, pp. 1695–700.
- Venereology Society of Victoria 2002, *National management guidelines for sexually transmissible infections*, Venereology Society of Victoria, http://www.msch.org.au

Erythema infectiosum (human parvovirus infection or slapped cheek disease)

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
The causative agent is human parvovirus B19.

Identification
Clinical features
Asymptomatic infection with human parvovirus B19 is common.

In children it causes a mild illness with little or no fever but a striking redness of the cheeks, hence the alternative name of 'slapped cheek disease'. There may also be a lacy pink rash on the trunk and limbs that fades within a week, but which may recur over several weeks on exposure to heat or sunlight. Headache, itch or common cold-type symptoms may also occur. In adults the rash is often absent or atypical. They may have cold-type symptoms and sometimes painful or swollen joints lasting two or three days.

Parvovirus affects the development of red blood cells. As a result several groups of people are at increased risk of developing complications:

- infection in the first half of pregnancy can cause foetal anaemia with hydrops foetalis. Foetal death occurs in less than ten per cent of these cases
- persons with haemolytic anaemia may develop transient aplastic crises, often in the absence of a rash
- immunosuppressed persons may develop severe chronic anaemia.

Method of diagnosis
Diagnosis can be suspected on clinical grounds, particularly during outbreaks. However, confirmation depends on demonstrating the presence of specific IgM antibodies or seroconversion to specific IgG antibodies. Comparison of the current antibody status against pre-natal screening serology for parvovirus is often useful in pregnancy.

Specific IgM antibody titres decline two to three months after infection while IgG levels, which appear two weeks after infection, can persist indefinitely.

Nucleic acid (PCR) testing and electron microscopy can also be used to confirm foetal infection.

Incubation period
The incubation period varies from four to twenty days.

Public health significance and occurrence
Human parvovirus infection occurs worldwide and is a common childhood disease. Outbreaks occur during winter and spring with epidemics occurring every three to four years.

Up to 50% of susceptible household contacts and 10–60% of child care or school contacts may be infected during outbreaks.

Reservoir
Humans.

Mode of transmission
The virus is transmitted by contact with infected respiratory secretions. It may be spread vertically from mother to foetus and rarely by transfusion of blood products.

Period of communicability
Children with erythema infectiosum are most infectious before the onset of the rash and are probably not infectious after the rash appears.

Patients with an aplastic crisis are infectious for a week after the onset of symptoms.

Immunosuppressed persons with chronic anaemia due to infection may excrete virus for years.

Susceptibility and resistance
Infection generally confers immunity. Serological surveys suggest 5–15% of preschool children and 50–60% of all adults are immune.

Control measures
Preventive measures
There is no vaccine available.

All people who are non-immune to parvovirus, immunosuppressed, have chronic haemolytic disorders, or who are pregnant are at increased risk of complications.

These people should be advised of the risk that parvovirus infection may pose to them. They should avoid close contact with children or adults in settings where parvovirus infection may occur such as schools, child care centres and health care facilities.

Strict hand washing and separate eating utensils are also advised for these people.

Control of case

There is no specific treatment required for uncomplicated infection.

Specialist advice should be sought if a patient with immunodeficiency or a blood disorder contracts parvovirus infection.

Control of contacts

Intrauterine infection may rarely result in foetal hydrops or death if infection occurs within the first 20 weeks of pregnancy. Medical advice should be sought for pregnant women who have been in close contact with a case of parvovirus infection. Specific antibody testing should be performed to determine the woman's immune status to parvovirus.

Control of environment

Not applicable.

Special settings

Patients and health care workers with acute parvovirus infection should not have contact with high risk hospitalised patients such as pregnant women, the immunosuppressed and those with a chronic haemolytic anaemia.

Outbreak measures

General public health measures include:

- advising high risk persons of relevant outbreaks
- advising patients and contacts to observe strict hand washing after coughing, sneezing and before eating.

Slapped cheek infection information sheet for pregnant women

Also known as human parvovirus infection or erythema infectiosum

What is slapped cheek infection?

Erythema infectiosum is also known as 'slapped cheek disease' or 'fifth disease' and is a common childhood viral infection caused by human parvovirus B19. Fifty to sixty percent of women are immune to the virus by the time they reach childbearing age. Occasionally, an unborn baby of a non-immune mother can develop problems if infected before the 20th week of pregnancy.

What are the symptoms?

Most cases experience no symptoms at all.

In children the infection causes a mild illness with little or no fever but a striking redness of the cheeks (hence 'slapped cheek disease'). This may be accompanied by a lacy looking rash that fades within a week but can re-occur over several weeks on exposure to heat or sunlight.

Adults often do not have a rash but may have cold-type symptoms and/or painful or swollen joints over two to three days.

How is it spread?

Parvovirus infection is spread by infected respiratory secretions through coughing, sneezing or touching something that has been coughed or sneezed on. About 50% of non-immune people will become infected if there is a case in their household, less if the case is at school or child care. Cases are infectious before the onset of the rash and are probably not infectious after the rash appears.

The incubation period of the infection is 1 to 2 weeks.

How can it affect my baby?

The risk to unborn babies is low. Spread from mother to baby can only occur if the mother is not immune. Even if the mother is affected only one-third of babies will develop the infection (generally about a month after the mother's illness).

Infection during the first 20 weeks of pregnancy can rarely cause a form of anaemia (low blood count) in the baby. In many cases this resolves by itself but in some instances it may require treatment. Very rarely it can be fatal. Parvovirus infection does not cause congenital abnormalities.

How can I protect myself and my baby?

Washing hands before eating or touching your face can help prevent infection. Avoid sharing cutlery, cups and plates.

What action should I take if I think I have been exposed?

A pregnant woman who believes she has been in contact with a case of parvovirus infection should consult the doctor supervising her pregnancy even if she has no symptoms. Blood testing can assist doctors advising women who are pregnant of the risk, if any, which parvovirus infection poses. There is no risk to a woman (or her baby) that already has immunity.

If active infection is diagnosed, ultrasound is performed every 1–2 weeks to monitor the health of the baby. If there are signs the baby is having difficulty with severe anemia a blood transfusion while the baby is still in the womb may be considered.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Department of Human Services
www.health.vic.gov.au/ideas

Slapped cheek infection information sheet

Also known as human parvovirus infection or erythema infectiosum

What is slapped cheek infection?

Erythema infectiosum, also known as 'slapped cheek disease' or 'fifth disease' is a common childhood viral infection caused by human parvovirus B19. Five to 15% of preschool children and 50–60% of all adults are immune.

What are the symptoms?

Most cases experience no symptoms at all.

In children the infection causes a mild illness with little or no fever but a striking redness of the cheeks (hence 'slapped cheek disease'). This may be accompanied by a lacy looking rash that fades within a week but can re-occur over several weeks on exposure to heat or sunlight. Adults often do not have a rash but may have cold-type symptoms and sometimes painful or swollen joints over two to three days.

How is it spread?

Parvovirus infection is spread by infected respiratory secretions (coughing, sneezing, or touching something that has been coughed or sneezed on). About 50% of non-immune people will become infected if there is a case in their household, less if the case is at school or child care. The incubation period of the infection is one to two weeks. Cases are infectious before the onset of the rash and are probably not infectious after the rash appears.

Who is at risk?

Several groups of people are at risk from the effects of parvovirus infection on developing red blood cells:

- people with chronic blood disorders (for example, sickle cell disease) may develop severe anaemia (low blood count)
- immunosuppressed people (for example those on chemotherapy, organ transplant recipients) may develop chronic anaemia
- occasionally, an unborn baby of a non-immune mother can develop problems if infected before the 20th week of pregnancy.

How can I protect myself?

Washing hands before eating or touching your face can help prevent infection. Avoid sharing cutlery or cups and plates with others.

What action should I take if I think I have been exposed?

There is no specific treatment required for uncomplicated infection. Specialist advice should be sought if a person with immunodeficiency or a blood disorder suffers parvovirus infection. A pregnant woman who believes she has been in contact with a case of parvovirus infection should consult the doctor supervising her pregnancy even if she has no symptoms.

Can my child attend day care or school?

Yes. As cases are infectious before the onset of the rash and are probably not infectious after the rash occurs there is no reason to exclude the child from school or day care once the rash appears.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Department of Human Services
www.health.vic.gov.au/ideas

Victoria
The Place To Be

Food or water-borne illness

Victorian statutory requirement

Two or more related cases of suspected food or water-borne illness must be notified within 24 hours of diagnosis.

School exclusion: for most gastrointestinal illnesses children should be excluded from school or childcare until at least the diarrhoea has ceased.

Infectious and other causative agents

The most frequent causes of food or water-borne illnesses are various bacteria, viruses and parasites. Refer to specific sections for detail on the more common agents.

Non-infective agents:

- heavy metal poisoning, including, cadmium, copper, lead, tin and zinc
- fish toxins that are present in some shellfish or fish like paralytic shellfish poisoning or ciguatera
- plant toxins which occur naturally in some foods such as toxic fungi and green potato skins
- toxic cyanobacteria (blue green algae) overgrowth in water.

Bacteria:

- toxin produced in food:
  - *Staphylococcus aureus*
  - *Clostridium botulinum*
  - *Bacillus cereus*
- damage to gut wall and/or systemic infection:
  - *Salmonella* spp.
  - *Shigella* spp.
  - *Clostridium perfringens*
  - *Campylobacter* spp.
  - *E. coli*.
  - *Helicobacter pylori*
  - *Vibrio cholerae/V. parahaemolyticus*
  - *Yersinia enterocolitica*
  - *S.typhi/Paratyphi*
  - *Brucella* spp.
  - *Listeria monocytogenes*

Viruses:

- Hepatitis A and E viruses
- Noroviruses and other small round structured viruses (SRSV)
- Rotavirus

Parasites:

- *Cryptosporidium* spp.
- *Entamoeba histolytica*
- *Giardia lamblia*

Identification

Clinical features

Symptoms vary with the causative agent and range from slight abdominal pain and nausea to retching, vomiting, abdominal cramps, fever and diarrhoea. Fever, chills, headache, malaise and muscular pains may accompany gastrointestinal symptoms. Vomiting, with or without diarrhoea, abdominal cramps and fever are common symptoms of viral disease or staphylococcal intoxication. Certain food-borne illnesses can present with meningitis or septicaemia (listeriosis) or with neurological symptoms (paralytic shellfish poisoning, botulism).

Severity depends on host and agent characteristics and the infectious dose. Hospitalisation and death may occur due to acute dehydration, metabolic acidosis and subsequent organ failure.

The duration of illness varies from hours (24–48 hours in viral and staphylococcal infections) to days and even weeks in salmonellosis and campylobacteriosis.

Method of diagnosis

Diagnostic methods vary according to the type of infective agent:

- bacteria can be isolated from faeces or blood or by detection of toxin
- parasites can be isolated by microscopy of fresh or appropriately preserved faeces
- viruses can be isolated by stool electron microscopy (EM), immune EM or paired sera from patients to detect seroconversion to a virus
- chemicals can be isolated by serological detection of implicated compounds.

Advice regarding specific tests should be sought from laboratories with expertise in the identification of gastrointestinal pathogens and chemical agents.

Incubation period

Incubation periods are typically short for toxin-producing bacteria and longer for others.

Public health significance and occurrence

Food and water-borne diseases are thought to be the most common of all acute illnesses. However a large proportion of disease is not detected, as many people will not seek health care with mild illness. Occurrence is worldwide and the incidence varies from country to country. In recent years the detection of outbreaks of viral origin, especially noroviruses, has been increasing.

Reservoir

- Soil, dust, cereals
- Bacteria and parasites: fish, birds, reptiles, wild and domestic animals
- Viruses: humans

Mode of transmission

Transmission is predominantly via the faecal-oral route or ingestion of contaminated food and water sources. Transmission via aerosols (produced during profuse vomiting) has been implicated in outbreaks involving viral pathogens.

Period of communicability

Communicable periods for food and water-borne illnesses depend on the causative agents. Viruses are generally communicable during the acute phase and up to two days after recovery while bacteria are generally communicable during the acute diarrhoeal stage. For parasites refer to relevant sections in this book.

Susceptibility and resistance

With most infections everyone is susceptible, however sporadic disease is more often detected in young children, the elderly or immunocompromised people. This is in some part due to the health care seeking behaviours of those caring for patients in these categories.

Control measures

Preventive measures

Prevention of the contamination of potable water is very important. Contaminated water should be treated by adequate filtration and disinfection or by boiling.

Avoiding contamination of food is also important. This can be achieved by:

- providing raw materials of better microbiological quality
- educating food handlers about proper food processing, preparation, storage and in personal hygiene
- adopting the following 'Ten golden rules for safe food preparation' developed by WHO:
  - choose food processed for safety
  - cook food thoroughly
  - eat cooked food immediately
  - store cooked food carefully
  - reheat cooked food thoroughly
  - avoid contact between raw foods and cooked foods
  - wash hands repeatedly
  - keep all kitchen surfaces meticulously clean
  - protect food from insects, rodents, and other animals
  - use pure water

Incorporation of HACCP (Hazard Analysis Critical Control Point) systems is important for good manufacturing practices for food industries.

Vaccines are currently available for cholera and hepatitis A (refer to relevant sections).

Control of case

Control of the case ranges from supportive treatment and rehydration to hospitalisation.

Cases due to infection need exclusion from food handling, schools and children's services centres until after the diarrhoea has ceased.

Health care workers need exclusion if employed in an area with high risk patients, such as special care nurseries or nursing homes, until after the diarrhoea has ceased.

Control of contacts

Control of contacts includes:

- prevention of further ingestion of contaminated food or water
- surveillance of contacts who are food handlers if required
- withdrawal of implicated food (if in retail outlets) from sale.

Control of environment

Investigate water sources or place of manufacture or preparation of incriminated food and institute corrective action.

Outbreak measures

Food and water-borne outbreaks are usually detected following the onset of illness in a group of people who have shared a common meal. The primary objectives of outbreak control are the rapid identification of the causative agent through epidemiological, environmental and laboratory investigations and prevention of further disease by destruction or denaturation of the source.

International measures

International outbreaks are increasingly being recognised, primarily due to the increased trade in food and agricultural products worldwide. Food and water-borne pathogens and contaminants have been identified as potential biological terrorism agents. Cases of rare diseases like botulism should be investigated immediately. Some diseases require notification to the World Health Organization, like cholera.

Quarantine of suspected sources or halting international trade should be coordinated through Food Standards Australia New Zealand.

Additional sources of information

Food Standards Australia New Zealand, http://www.foodstandards.gov.au

Common food- or water-borne pathogens

Causative agent	Incubation period	Duration of illness	Predominant symptoms	Foods commonly implicated
Bacteria				
Campylobacter jejuni	1–10 days (usually 2–5 days)	2–5 days occasionally >10 days	Sudden onset of diarrhoea, abdominal pain, nausea, vomiting	Raw or undercooked poultry, raw milk, raw or undercooked meat, untreated water
E. coli enterohaemorrhagic (STEC, VTEC)	2–10 days	5–10 days	Severe colic, mild to profuse bloody diarrhoea can lead to haemolytic uraemic syndrome	Many raw foods (especially minced beef), unpasteurised milk, contaminated water
E. coli enteropathogenic enterotoxigenic enteroinvasive	12–72 hrs (enterotoxigenic)	3–14 days	Severe colic, watery to profuse diarrhoea, sometimes bloody	Many raw foods, food contaminated by faecal matter, contaminated water
Salmonella serovars (non-typhoid) products	6–72 hrs	3–5 days	Abdominal pain, diarrhoea, chills, fever, malaise	Raw or undercooked meat and chicken, raw or undercooked eggs and egg
Salmonella Typhi/ paratyphi	Typhoid 8–14 days Paratyphoid 1–10 days	Days-weeks (chronic asymptomatic carriers can occur)	Systemic illness - sustained fever, headache and constipation rather than diarrhoea	Raw shellfish, salads, contaminated water
Shigella spp.	12–96 hrs	4–7 days	Malaise, fever, vomiting, diarrhoea (blood & mucus)	Foods contaminated by infected food handlers and untreated water contaminated by human faeces
Yersinia enterocolitica	3–7 days	1–21 days	Acute diarrhoea sometimes bloody, fever, vomiting	Raw meat especially pork, raw or undercooked poultry, milk and milk products
Vibrio cholerae	A few hours to 5 days	3–4 days	Asymptomatic to profuse painless watery diarrhoea, dehydration	Raw seafood, contaminated water
Vibrio parahaemolyticus	4–30 hours (usually 12–24 hrs)	1–7 days	Abdominal pain, diarrhoea, vomiting and sometimes fever. Illness of moderate severity	Raw and lightly cooked fish, shellfish, other seafoods
Listeria monocytogenes	3–70 days	Varies	Gastrointestinal symptoms rare; flu-like symptoms to meningitis/ septicaemia; infection in pregnancy can result in abortions, neonatal infection	Unpasteurised milk, soft cheese, pate, coleslaw, salads, ready to eat seafood, cold meats, fresh fruit drinks
Viruses				
Norovirus (and other viral gastroenteritis)	24–48 hrs	12–60 hrs	Severe vomiting, diarrhoea	Oysters, clams, foods contaminated by infected food handlers and untreated water contaminated by human faeces
Rotaviruses	24–72 hrs	Up to 7 days	Malaise, headache, fever, vomiting, diarrhoea	Foods contaminated by infected food handlers and untreated water contaminated by human faeces
Hepatitis A	15–50 days	Usually 1–2 weeks	Fever, nausea, abdominal discomfort, possibly jaundice	Shellfish, foods contaminated by infected food handlers and untreated water contaminated by human faeces

Common food- or water-borne pathogens continued

Causative agent	Incubation period	Duration of illness	Predominant symptoms	Foods commonly implicated
Parasites				
Cryptosporidium	1–12 days	4–21 days	Profuse watery diarrhoea, abdominal pain	Foods contaminated by infected food handlers and untreated water contaminated by human faeces
Giardia lamblia	1–3 weeks	1–2 weeks to months	Loose pale greasy stools, abdominal pain	Foods contaminated by infected food handlers and untreated water contaminated by human faeces
Entamoeba histolytica	2–4 weeks	Weeks to months	Colic, mucous or bloody diarrhoea	Foods contaminated by infected food handlers and untreated water contaminated by human faeces
Toxin producing bacteria				
B. cereus (toxin in food)	1–6 hrs (vomiting) or 6–24 hrs (diarrhoea)	< 24 hrs	Two known toxins causing nausea and vomiting or diarrhoea and cramps	Cereals, rice, meat products, soups, vegetables
Clostridium botulinum	12–36 hrs	Variable	(Neurotoxin) Blurred or double vision, difficulty swallowing, respiratory paralysis, muscle weakness and lethargy	Canned food, often home canned food (low acid)
C. perfringens (toxin in gut)	6–24 hrs	24 hrs	Sudden onset colic, diarrhoea	Meats, poultry, stews, gravies, (often inadequately reheated or held warm)
Staphylococcus aureus (toxin in food)	30 min – 8 hrs	24 hrs	Acute vomiting, and cramps, may lead to collapse	Cold foods (much handled during preparation) milk products, salted meats
Fish / shellfish toxins				
Scombroid fish poisoning (histamine poisoning)	Few hours	Up to 12 hours	Tingling and burning around mouth, sweating, diarrhoea, vomiting, headache, dizziness	Fish such as tuna, mackerel, skipjack, bonito, herring and sardines. Fish stored at >5°C for extended periods
Ciguatera poisoning	Less than 24 hours	Weeks to months	Numbness and tingling around mouth, diarrhoea, vomiting and nausea followed by neurological symptoms such as dizziness, blurred vision and temperature reversal.	Large tropical reef fish
Paralytic shellfish poisoning (PSP)	Minutes to several hours	Several days	Burning and tingling around the mouth and extremities, nausea dizziness, potentially muscle and respiratory paralysis	Bivalve molluscs
Diarrhetic shellfish poisoning (DSP)	30 mins – 2 hrs	Hours to 3 days	Diarrhoea, nausea, vomiting and abdominal pain	Mussels, scallops and clams

Giardiasis

Victorian statutory requirement

Giardiasis (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude cases from child care and school until diarrhoea has ceased or until a medical certificate of recovery is produced.

Infectious agent

Giardia lamblia is a flagellate protozoan which lives in the duodenum and jejunum.

Identification

Clinical features

Giardia infection is usually asymptomatic but may present as acute or chronic diarrhoea associated with abdominal cramps, bloating, nausea, vomiting, fever, fatigue and weight loss. Fat malabsorption may lead to steatorrhoea. Symptoms usually last one to two weeks or months. The rate of asymptomatic carriage may be high.

Method of diagnosis

Stool microscopy for cysts or trophozoites can be used for diagnosis of *Giardia* however a negative test does not preclude infection.

Incubation period

The incubation period is usually one to three weeks but it can be longer. It is on average seven to ten days.

Public health significance and occurrence

Occurrence is worldwide and endemic in most regions. Over 800 cases are reported in Victoria each year. Infection is detected more frequently in children than adults. It is readily transmitted in institutions such as day care centres among children who are not toilet trained. Other risk factors for infection include travel to high risk areas, immunosuppression, male to male sexual intercourse and achlorhydria.

Reservoir

Reservoirs include humans and animals as well as contaminated waters.

Mode of transmission

Transmission occurs person to person and animal to person via hand to mouth transfer of cysts from infected faeces or faecally contaminated surfaces. Water-borne outbreaks may occur due to faecal contamination of public water supplies or recreational swimming areas.

Period of communicability

It is communicable for the entire period of cyst excretion.

Susceptibility and resistance

Everyone is susceptible to infection. Relapses may occur.

Control measures

Preventive measures

Preventative measures include:

- educating families and personnel of day care centres in personal hygiene such as the need for hand washing before meals, after toilet use and changing nappies
- protecting public water supplies against faecal contamination
- educating travellers about the need for safe food and water consumption.

Control of case

Symptomatic cases are usually treated with metronidazole or tinidazole. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). Treatment of asymptomatic carriers is rarely warranted.

Dispose of faeces in a sanitary and hygienic manner and disinfect soiled clothing and other articles concurrently.

School exclusion criteria apply until diarrhoea has ceased or until a medical certificate of recovery is produced. Food handlers should not attend work until diarrhoea has ceased and strict hygienic food preparation practices should be maintained.

It is also recommended that health care workers or child care workers do not attend work until diarrhoea has ceased.

Control of contacts

The diagnosis should be considered in symptomatic contacts. Active case finding among contacts is rarely indicated.

Control of environment

Disinfection of contaminated areas or water sources is required. Particular attention should be paid to potentially contaminated surfaces in child care centres.

Outbreak measures

Two or more related cases may indicate an outbreak and requires prompt reporting to the Department of Human Services. Attempt to identify a potentially common exposure such as child care attendance or exposure to farm animals and recreational swimming areas. Epidemiological, environmental and laboratory investigations may be warranted as per the Department's *Guidelines for the investigation of gastrointestinal illness*.

Gonorrhoea

Victorian statutory requirement

Gonorrhoea (Group C disease) must be notified in writing within five days of diagnosis.

Specific information must be notified under the Health (Infectious Diseases) Regulations 2001. To maintain confidentiality, only the name code (first two letters of the surname followed by the first two letters of the first name) is required. A questionnaire is sent to the diagnosing doctor to collect additional information on the case that is essential for detecting disease trends and informing policy development.

Medical practitioners have a statutory obligation under the *Children and Young Person's Act 1989* to notify the Department of Human Services Child Protection Service if they believe that a child is in need of protection on the basis of sexual abuse.

Infectious agent

Neisseria gonorrhoeae

Identification

Clinical features

Infections with *N. gonorrhoeae* may present with a number of clinical syndromes.

The most common presenting symptom in males is a painful purulent urethral discharge. If left untreated, complications may include epididymitis, prostatitis and urethral stricture. Anorectal infection is more common in homosexual males and is usually asymptomatic. It may cause pruritis, tenesmus and discharge. Pharyngeal infection is usually asymptomatic.

In females, an initial urethritis or cervicitis occurs a few days after exposure. It is frequently mild and passes unnoticed. Females may have abnormal vaginal discharge and post-coital bleeding. Later, pelvic inflammatory disease may develop. Pelvic inflammatory disease may cause ectopic pregnancy, infertility or chronic pelvic pain.

Conjunctivitis can occur in neonates and rarely in adults. It may cause blindness if not rapidly and adequately treated.

Septicaemia and septic arthritis are rare complications.

Method of diagnosis

Swabs taken from the urethra, cervix, pharynx, rectum or other site should be rolled onto a slide first and then sent to the laboratory in an appropriate transport medium.

The following tests can be performed on swabs and smears taken from the site of infection:

- Gram stain on discharges smeared on the slide.
- culture on both selective and non-selective media should be used. Culture of *N. gonorrhoeae* provides definitive diagnosis, and isolates provide valuable information on patterns of antibiotic resistance and other epidemiological markers.
- nucleic acid testing can be performed on cervical and urethral swabs and urine. In women, PCR testing of urine is less sensitive than PCR testing on endocervical swab specimens. In cases diagnosed by PCR, further specimens should be obtained if possible for culture to allow monitoring of antibiotic resistance.

Co-infection with *Chlamydia trachomatis* sometimes occurs, particularly in imported cases. Screening for other sexually transmissible infections such as chlamydia should be considered when testing for *N. gonorrhoeae*.

Incubation period

The incubation period is usually two to seven days.

Public health significance and occurrence

Gonorrhoea is common worldwide and affects both sexes. Infection may be symptomatic or asymptomatic. Infections of the cervix, anus and throat usually cause no symptoms. Gonorrhoea can have acute and chronic sequelae.

Strains of gonococci resistant to penicillin are common and widespread. Resistance to fluoroquinolone antibiotics such as ciprofloxacin is common among isolates from infections acquired in Asia. Ciprofloxacin resistance in gonococcal isolates in Victoria is increasing.

Gonorrhoea may increase susceptibility to the sexual acquisition of HIV infection and increase HIV infectiousness.

Other serious complications such as blindness from neonatal conjunctival infection and the various complications of pelvic inflammatory disease are currently rare in Victoria. The rate of notified cases of gonorrhoea increased in Victoria in the late 1990s to a level not seen since the mid 1980s. The increase involved men who have sex with men who comprised approximately two thirds of cases, and also heterosexual men. A similar phenomenon was noted elsewhere in Australia and overseas. This increase has been sustained in Victoria.

Reservoir
Humans.

Mode of transmission
Gonorrhoea is transmitted by contact with exudates from mucous membranes of infected people, almost always as the result of sexual activity.

Gonococcal conjunctivitis can occur in neonates who have had contact with the mother's infected birth canal during childbirth.

Period of communicability
Communicability may extend for months in untreated individuals.

Susceptibility and resistance
Everyone is susceptible to infection.

Control measures
Preventive measures
Preventative measures include education about safe sex practices including use of condoms and early detection of infection by testing of those at risk.

Control of case
Ceftriaxone plus azithromycin or doxycycline (to cover co-existing chlamydial infection), are used to treat gonorrhoea. Ciprofloxacin can be used as an alternative to ceftriaxone when a sensitive strain has been identified. Advice on the clinical management of patients with gonococcal infection can be found in *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and the *National management guidelines for sexually transmissible infections* (Venereology Society of Victoria, 2002).

Specialist consultation should be sought for complicated or disseminated infections and for infection during pregnancy.

Control of contacts
Sexual partners of individuals with gonorrhoea should be examined and investigated then treated empirically.

Contact tracing assistance can be provided by the Department's partner notification officers (03) 9347 1899.

Control of environment
Not applicable.

Outbreak measures
Not applicable.

Additional sources of information

- Australian Government Department of Health and Family Services 1997, *Contact tracing manual – a practical handbook for health care providers managing people with HIV, viral hepatitis, other STDs and HIV-related tuberculosis*.
- Centers for Disease Control and Prevention 2002, 'Sexually transmitted diseases treatment guidelines 2002', *Morbidity and Mortality Weekly Report*, vol. 51 (RR06), pp.1–80, http://www.cdc.gov/mmwr
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- Fleming, DT & Wasserheit, JN 1999, 'From epidemiological synergy to public health policy and practice: the contribution of other sexually transmitted diseases to sexual transmission of HIV infection,' *Sexually Transmissible Infections*, vol. 73, pp. 3–17.
- Venereology Society of Victoria 2002, *National management guidelines for sexually transmissible infections*, Venereology Society of Victoria, http://www.msch.org.au

Haemophilus influenzae infections

Victorian statutory requirement

Suspected and confirmed *Haemophilus influenzae* type B (Hib) infections (Group A disease) must be notified immediately by telephone followed by written notification within five days.

Infectious agent

Haemophilus influenzae is a gram-negative coccobacillus. Invasive infections are commonly caused by serotype B.

Identification

Clinical features

Meningitis

The onset can be sub-acute or sudden with fever, vomiting, lethargy and meningeal irritation with a bulging fontanelle in infants and stiff neck and back in older children.

Epiglottitis

The patient is usually a child and presents with signs of upper respiratory tract obstruction and a characteristic expiratory snore, difficulty in swallowing with drooling of saliva, irritability, restlessness and fever. Progression of the infection can lead to complete respiratory obstruction.

H. influenzae type b infection may also cause other diseases such as pneumonia, septic arthritis, cellulitis and osteomyelitis.

Method of diagnosis

Clinical diagnosis is confirmed in the laboratory by:

- isolation of Hib from a normally sterile site such as blood or cerebrospinal fluid, typing should be confirmed by an approved reference laboratory
- detection of Hib antigen in CSF when other laboratory parameters are consistent with bacterial meningitis, and when there has been no Hib vaccination within 21 days of onset.
- Body fluids and urine may give positive antigen reactions for Hib for up to 21 days after vaccination.

Incubation period

The incubation period is uncertain. It is probably two to four days.

Public health significance and occurrence

Prior to the introduction of Hib vaccine to the routine immunisation schedule in 1993, Hib disease was the most common serious invasive bacterial infection in children. At this time at least 500 cases of Hib disease annually in Australian in children less than six years of age. There were 10–15 deaths per year and 20–40% of survivors were left with permanent neurological damage.

Indigenous children were at five to six times greater risk of developing Hib disease and acquired it at a much younger age than non-indigenous children.

By 1998 the number of notified cases in Australia had reduced by more than 90%. The number of notified cases has continued to fall. Invasive Hib disease is now only very rarely seen in Victorian children. There has been no evidence of a shift in Hib cases to older age groups.

Hib epiglottitis may still occur, particularly in adults and unimmunised children. This diagnosis should still be considered when a person presents with fever and signs of upper respiratory obstruction resembling croup. Other previously rare bacterial causes of epiglottitis may now be more likely diagnoses.

Immunosuppressed individuals of any age remain at risk from Hib infection. Asplenic patients are at greater risk of infection if they have not been appropriately immunised.

Reservoir

Humans.

Mode of transmission

Hib is transmitted person to person through respiratory droplet spread and may also be rarely acquired through contact with infected respiratory discharges.

Period of communicability

Hib is communicable for as long as the organisms are present in the nasopharynx. Patients are no longer infectious once they have received 24 to 48 hours of appropriate antibiotic therapy.

Susceptibility and resistance

Sustained immunity is conferred through immunisation or prior infection. Maternal antibody provides passive immunity for a variable time period after birth.

Infection does not always result in immunity. This is particularly evident in children less than two years of age who are unable to mount an antibody response to the type b capsular polysaccharide, even following invasive disease.

Most secondary cases among close contacts occur within the first week after exposure, though late secondary cases have been reported.

Control measures

Preventive measures

Routine childhood immunisation remains the most important preventive measure against Hib disease. Hib vaccine is recommended as part of the Australian Standard Vaccination Schedule for all children at two, four and 12 months of age and for older persons with asplenia. Refer to the current edition of the *Australian immunisation handbook* (National Health and Medical Research Council) for further details.

Before and after splenectomy

Hib is an uncommon cause of post-splenectomy sepsis in adults and children. Children over two years of age who are fully immunised do not require a Hib booster following splenectomy. A single dose is recommended for any other individuals (regardless of age) if incompletely or unvaccinated who have close contact with children less than five years. No booster doses are required.

If possible the vaccine should be given at least two weeks before splenectomy.

Control of case

Intravenous cefotaxime or ceftriaxone may be used for empirical therapy until antibiotic sensitivities are known. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and seek expert infectious disease advice.

These antibiotics do not clear Hib from the nasopharynx. Rifampicin should be given to cases prior to discharge from hospital to ensure clearance of the organism. If the treated patient is less than two years of age and has not been immunised, a course of Hib vaccine should still be given after discharge from hospital.

Respiratory isolation procedures are recommended for 24 hours after the start of treatment.

Concurrent or terminal disinfection of possibly contaminated items is not generally required.

Control of contacts

Unvaccinated contacts less than five years of age should be immunised as soon as possible.

Household contacts

Parents of confirmed cases should be educated about the risks of secondary cases in siblings and other close contacts under five years of age, and seek early medical review if any close contacts develop symptoms consistent with Hib disease.

Chemoprophylaxis (see below) is indicated for household contacts only if:

- the household of a case contains one or more infants under seven months of age regardless of vaccination status, or
- the household of a case contains one or more children aged seven months to five years who are not age-appropriately immunised against Hib according to the current Australian Standard Vaccination Schedule.

In either setting, all persons in the same household should receive chemoprophylaxis and inadequately vaccinated children should receive age appropriate Hib vaccination.

Child care

If the case attends a child care facility for more than 18 hours a week and other children less than two years of age in this facility are in close contact, chemoprophylaxis should be given to all contacts including staff **if** any of the close contacts are inadequately vaccinated.

Chemoprophylaxis does not eliminate the need for surveillance and parents of contacts should be advised of the risk of late secondary cases despite prophylaxis.

Chemoprophylaxis

Decisions about the use and advice about contra-indications, dosing and supply of rifampicin for chemoprophylaxis should always be made in consultation with the Department of Human Services. For adverse effects and contra-indications to rifampicin, see section on meningococcal disease.

Control of environment

See Outbreak measures, below.

Outbreak measures

Outbreaks of Hib are now rare. The public health response to a cluster of Hib cases is based on the control principles outlined above in Control of contacts, which may require expansion in the extent of contact surveillance, chemoprophylaxis and vaccination.

Hand, foot and mouth disease

Victorian statutory requirement

Notification is not required.

School exclusion is required until all blisters have dried.

Infectious agent

Coxsackievirus group A, mainly type 16, is the infectious agent.

Human hand, foot and mouth disease is unrelated to the foot and mouth disease of animals (caused by members of the family *Picornaviridae*).

Identification

Clinical features

Hand, foot and mouth disease (HFMD) occurs mainly in children under ten years of age and in young adults. Symptoms and lesions usually persist for seven to ten days.

The clinical picture consists of sore throat, fever and vesicular lesions on the buccal surfaces of the cheeks, gums and sides of the tongue.

Papulovesicular lesions of the palms, fingers and soles commonly occur. Occasionally maculopapular lesions appear on the buttocks.

Method of diagnosis

Diagnosis of HFMD is usually clinical. Viral isolation from nasopharyngeal or stool specimens is possible but rarely indicated.

Incubation period

The incubation period is from three to seven days.

Public health significance and occurrence

Hand, foot and mouth disease occurs worldwide sporadically and in epidemics. The greatest incidence is in summer and early autumn. Outbreaks occur frequently among groups of children in child care centres and schools.

Reservoir

Humans.

Mode of transmission

HFMD is transmitted by direct contact with fluid from the vesicular lesions, direct contact with nose and throat discharges and faeces of an infected person, and aerosol droplet spread.

Period of communicability

It is communicable during the acute stage of disease from nose and throat secretions and as long as there is fluid in the lesions. Viruses persist in the stools for several weeks.

Susceptibility and resistance

Everyone is susceptible to infection. Immunity to the specific virus may be acquired due to previous infection. Second attacks may occur with group A coxsackievirus of a different serotype.

Control measures

Preventive measures

Not applicable.

Control of case

Control of the case includes:

- exclusion from school of children with hand, foot and mouth disease until all blisters have dried
- covering lesions on hands and feet if possible and allowing to dry naturally
- avoiding piercing lesions as the fluid within the blisters is infectious
- good hand washing, cleaning and disposal of soiled articles.

Control of contacts

Not applicable.

Outbreak measures

Not applicable.

Hand, foot and mouth disease information sheet

What is hand food and mouth disease?

Hand foot and mouth disease is caused by a virus (usually coxsackie virus A16).

It causes blisters on the hands and feet, in the mouth and often in the 'nappy' area.

It is generally only a mild disease that lasts seven to ten days.

It is more common during warmer weather and tends to spread easily between children.

This infection is spread by direct contact with fluid from the skin blisters, nose and throat discharges, droplets (sneezing, coughing) and faeces (stools). Good personal hygiene is important to prevent spread of the infection to others.

There is no connection between this disease and the foot and mouth disease that affects cattle and some other animals.

Who gets hand, foot and mouth disease?

Most people have been infected with the virus which causes this disease by the time they are adults. So it is generally just a small percentage of children who get features of disease after infection.

Signs and symptoms

People usually develop symptoms between three to seven days after being infected.

The most common signs and symptoms are:

- a high temperature (fever)
- a sore throat
- small, blister-like lesions that may occur on the inside of the mouth, sides of the tongue, palms of the hands, fingers, soles of the feet and 'nappy' area.

How long is it infectious?

The skin blisters of hand, foot and mouth disease are infectious until they become crusty and there is no fluid in the blisters. The virus may also be shed in the faeces for several weeks after the blisters resolve.

Good personal hygiene is essential to prevent the spread of hand, foot and mouth disease to others, both for those infected and their carers. This includes:

- washing hands carefully after contact with the blister-like lesions, after handling nose and throat discharges, and after contact with faeces such as with nappy changing
- allowing blisters to dry naturally. Do not pierce blisters, as the fluid within is infectious
- using separate eating and drinking utensils.

Children with hand, foot and mouth disease should be excluded from school and child care centres until all the blisters have dried.

How do you treat hand foot and mouth disease?

There is no specific treatment for hand, foot and mouth disease.

Use paracetamol (not aspirin) as directed for fever and any discomfort.

The disease itself is generally mild. If a child with hand, foot and mouth disease complains of severe headache, if fever persists, or if there are any other worrying symptoms consult your local doctor immediately.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Department of Human Services
www.health.vic.gov.au/ideas

Victoria
The Place To Be

Hendra and Nipah viruses

Victorian statutory requirement

Notification is not required however any new case of these emerging infections should be discussed with the Department of Human Services as a matter of urgency.

School exclusion is not required.

Infectious agent

Two distinct, but closely related RNA viruses of the family *Paramyxoviridae*: Hendra virus which has so far only been detected in Queensland, and Nipah virus which has been confined to the Malay Peninsula.

Identification

Clinical features

Both viral diseases may cause acute infection with a variety of symptoms including fever, headache, shortness of breath, dizziness, drowsiness and confusion. Two of the three recorded cases of Hendra virus in humans were fatal. One death was due to septic pneumonia while the other was due to severe encephalitis.

In clinical cases of Nipah virus infection encephalitis is the major manifestation, often leading to coma and death in three to thirty days. The case fatality rate for clinical cases approaches 50%. The frequency of subclinical infections is unknown.

Method of diagnosis

The diagnosis can be made by the detection of specific neutralising IgM and IgG antibodies to either virus. Testing is available through the CSIRO Australian Animal Health Laboratory at Geelong. The diagnosis can also be confirmed by virus isolation from infected tissues.

Incubation period

The incubation period varies from four to18 days and rarely up to three months (Hendra virus).

Public health significance and occurrence

Hendra and Nipah viruses are recently recognised zoonotic viral diseases. Hendra virus appears to have emerged from fruit bats in Australia. Horses are the intermediary host most commonly associated with human infection. The first described outbreak of Hendra virus infection occurred in the Brisbane suburb of Hendra in 1994, involving 21 horses (14 fatal cases) and two of their human handlers (one fatal case). A smaller outbreak occurred in 1995 involving two horses and a farmer from the northern Queensland town of Mackay.

The presumed reservoir for Hendra virus is the fruit bat, the Australian flying fox (Pteropus spp.), which appears to be an asymptomatic host. A 20% seropositive rate to Hendra virus has been found among *Pteropus* bats in Queensland. Wildlife workers who frequently come in to contact with Australian bats have very low seropositive rates.

Nipah virus (named after the Baru Sungai Nipa village in Malaysia) is closely related to Hendra virus. Nipah virus may also have emerged from fruit bats. Pigs are the most common intermediary host associated with human infection. The virus was first identified in 1999 during the investigation of an outbreak in several pig-farming provinces on the Malay Peninsula. The outbreak began in 1998 and resulted in 265 confirmed human cases with 105 deaths in Malaysia and Singapore.

No human or animal cases of Hendra or Nipah viral disease have been detected in Victoria.

Reservoir

Fruit bats (Pteropus spp.) are the primary reservoir for both viruses and appear to be asymptomatic carriers. Horses are the most likely intermediary host in human infection for Hendra virus although other species such as cats show serological evidence of exposure.

Pigs are the most likely intermediary host for Nipah virus although seropositive horses and dogs have been identified.

Mode of transmission

The mode of transmission is unknown although a respiratory route is suspected. Human infection has been most commonly associated with direct contact with infected horses (Hendra) or infected pigs (Nipah). Symptomatic infections also occur in cats and other animals. Both viruses have been isolated from the urine of infected bats and other infected animals.

Period of communicability

There is no evidence of person to person transmission.

Susceptibility and resistance

Unknown.

Control measures

Preventive measures

In an outbreak setting the public should be advised to avoid contact with possible animal sources, particularly bats.

Control of case

Treatment is primarily supportive as there is no proven specific treatment. An

uncontrolled trial of the antiviral drug ribavirin has suggested it may reduce the mortality in Nipah virus encephalitis. Expert treatment advice should be sought from an infectious diseases physician.

Control of contacts
No person to person transmission has been observed.

Control of environment
See Outbreak measures, below.

Outbreak measures

A single confirmed case of either of these emerging viral infections would constitute an outbreak.

Case investigation determines the likely source of infection through a detailed history of the patient's work and travel history.

Further cases may be identified through active case finding amongst close contacts with similar exposure, as well as animal source detection if the infection was acquired in Australia.

In the event of a case being linked to exposure in Victoria or elsewhere in Australia, the Department would work closely with relevant animal health authorities and scientists to control possible sources of infection.

Measures could include:

- appropriate protective equipment and hygiene practices for animal handlers and investigators on implicated farms or properties
- slaughter of infected horses, pigs, or other animals with burial or incineration of carcasses

restriction of movement of horses or pigs from infected farms or designated areas.

International measures

Prohibition of export of animal products from affected areas.

Additional sources of information

- McCormack, JG, Allworth, MA 2002, 'Emerging viral infections in Australia,' *Med J Aust*, vol. 177, pp. 45–49.
- Patterson, DL, Murray, PK, McCormack, JG 2000, 'Zoonotic disease in Australia caused by a novel member of the Paramyxoviridae', *Clin Infect Dis*, vol. 27, pp. 112–8.

Hepatitis A

Victorian statutory requirement

Hepatitis A infection (Group B disease) must be notified in writing within five days of diagnosis.

School and child care exclusion are outlined below (see Control measures).

Infectious agent

Hepatitis A virus (HAV) is the causative agent.

Identification

Clinical features

Illness due to hepatitis A typically causes acute fever, malaise, anorexia, nausea and abdominal discomfort. This is followed a few days later by dark urine and jaundice. Symptoms usually last several weeks although convalescence may sometimes be prolonged. Severe illness may rarely occur when hepatitis A infection complicates pre-existing liver disease. Infants and young children infected with HAV may have a mild illness with few or no symptoms, with jaundice often being absent.

Method of diagnosis

A blood test indicating IgM anti-HAV antibodies confirms recent infection. These antibodies are present for two to four months after infection. IgG antibodies alone are evidence of past infection.

In the acute stage of the illness, blood biochemistry shows elevated transaminase levels indicating hepatocellular damage. The pattern of liver function tests may be non-specific in later illness.

Incubation period

The incubation period is fifteen to fifty days, with an average of 28–30 days.

Public health significance and occurrence

Hepatitis A occurs worldwide. In developing countries most people are infected during childhood. With good sanitation and hygiene in the developed world, most people now reach adulthood without experiencing infection. There are about 70–200 cases per year in Victoria. Notifications have been declining nationally since the late 1990s. Infection is more common in travelers to endemic areas, injecting drug users, children in childcare and men who have sex with men.

Common source outbreaks due to contaminated food are rare.

Reservoir

Humans.

Mode of transmission

Infection is transmitted by the faecal-oral route from person to person or via fomites. Infectious food handlers may contaminate non-cooked foods such as salads.

Infection can also occur through ingestion of contaminated food or water. Filter-feeding shellfish such as oysters raised in contaminated waters may harbour the virus.

The precise timing and mode of transmission are often difficult to define.

Period of communicability

Cases are most infectious from the latter half of the incubation period until a few days after the onset of jaundice, corresponding to a peak in transaminase levels in cases without jaundice. Most cases are not infectious after the first week of jaundice. Long term carriage or excretion of the virus does not occur.

Susceptibility and resistance

All non immune people are susceptible to infection. Immunity after infection is probably lifelong.

Control measures

Preventive measures

Education about good hygiene is important, particularly hand washing before handling food and eating and after using the toilet. Inadequate sanitation and housing may contribute to endemic illness.

Inactivated hepatitis A vaccines are available for use in persons two years of age and over. Protection begins within 14–21 days after the first dose. A second dose is required for long term protection. The vaccine is recommended for travellers to high risk areas, persons in high risk occupations such as childcare workers and emergency services personnel, injecting drug users and men who have sex with men.

Control of case

Treatment is generally supportive.

Exclude from childcare, school or work for at least one week after the onset of illness or jaundice and until they are well.

Children must have a medical certificate of recovery before returning to school or child care.

Educate the patient and their family on the need for strict hygiene practices.

Infected persons should not prepare meals for others while infectious, nor share utensils, toothbrushes, towels and face washers.

Dispose of or thoroughly wash nappies of infants that have hepatitis A.

Control of contacts

Normal immunoglobulin (IG) 0.02 mL/kg body weight intramuscularly is recommended for:

- household and sexual contacts of the case
- staff and children in close contact with a case in a childcare centre.

IG is not recommended for usual office, school or factory contacts. IG must be given within seven to ten days of exposure to be effective. IG is rarely given to persons exposed to a potential common source of hepatitis A such as food or water because cases related to such a source are usually recognised too long after the exposure for IG to be effective. Timely administration of IG will prevent or modify clinical illness for approximately six weeks after the dose. However, people exposed and infected before the administration of IG may still experience a mild infection, and may have the potential to infect others if strict personal hygiene is not maintained.

Surveillance of contacts in a household or workplace should be maintained.

Live vaccines such as Measles Mumps Rubella (MMR) should not be administered for three months after a dose of IG, and may also be ineffective if given in the 14 days prior to IG. Reschedule such routine vaccinations.

When the case is a food handler:

- consider serological testing of co-workers to determine whether they have been infected or are susceptible
- place uninfected susceptible co-workers under surveillance and give them IG prophylaxis. These persons remain at a risk of developing mild illness modified by IG but can generally continue to work provided good personal hygiene and food handling practices are maintained
- undertake surveillance for hepatitis A in patrons by seeking a history of exposure to the food premises from cases notified over the next two to three months
- carefully consider the role of the infected food handler. If transmission to patrons appears likely, consider urgent follow-up of exposed patrons to offer them IG prophylaxis. Note that when the index case is a patron, it is usually too late to offer IG prophylaxis to other diners, although personal contacts of the patron case should be offered IG according to the usual protocol.

When the case is a health care worker, the role of the case should be assessed and consideration given to the provision of IG prophylaxis for co-workers and patients in their direct care whilst infectious. Surveillance of contacts in the health care facility should be maintained.

Control of environment

A source of infection should always be sought. For apparently sporadic cases, consider contact with another known case and recent travel to an area where the disease is endemic. Acquisition of infection from young children, particularly those in childcare should be considered.

Special attention should be given to toilet hygiene in schools and childcare centres. Ensure that soap and water are available and are used regularly to wash hands.

Food premises, health care facilities or child care centres where a case has worked whilst potentially infective should be requested to carry out a clean up in accordance with the Department's *Guidelines for the investigation of gastrointestinal illness*.

Outbreak measures

Clusters of cases possibly related to a single source will require epidemiological and environmental investigation, including case finding and surveillance and public health measures to prevent further cases.

Use of the hepatitis A vaccine in an outbreak setting is dependent on rapid identification of the outbreak and persons at risk, and the ability to achieve high vaccine coverage levels.

Hepatitis B

Victorian statutory requirement

Hepatitis B infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

Hepatitis B virus (HBV) is the causative agent.

Identification

Clinical features

Less than 10% of children and only 30–50% of adults with acute HBV infections will have icteric disease. The onset is usually insidious with anorexia, abdominal discomfort, nausea, vomiting, lethargy and occasional rash and arthralgia. It often progresses to dark urine and jaundice. The severity of infection ranges from asymptomatic cases detected only after further investigation of abnormal liver function tests, to fulminating and often fatal case with extensive acute hepatic necrosis.

Method of diagnosis

HBV infection is confirmed by the detection of hepatitis B surface antigen (HBsAg) or of HBV DNA in serum. Serology determines whether infections are newly acquired or reflect chronic carriage.

Serology for newly acquired infections requires one of the following:

- detection of HBsAg in a patient shown to be negative within the last 24 months
- detection of HBsAg and high levels of specific IgM to hepatitis B core antigen (IgM HBcAg) in the absence of prior evidence of HBV infection
- detection of HBV DNA and high levels of specific IgM to hepatitis B core antigen (IgM HBcAg) in the absence of prior evidence of HBV infection.

Serology for chronic carriers requires:

- detection of HBsAg or HBV DNA in the serum of a patient on two occasions at least six months apart.

The following table summarises the interpretation of hepatitis B virus serology.

Tests	Results	Interpretation
HbsAg anti-HBc anti-HBs	negative negative negative	susceptible – discuss vaccination
HbsAg anti-HBc anti-HBs	negative positive positive	immune due to infection
HbsAg anti-HBc anti-HBs	negative negative positive	immune due to hepatitis B vaccination
HbsAg anti-HBc IgM anti-HBc anti-HBs	positive positive positive negative	acutely infected
HbsAg anti-HBc IgM anti-HBc anti-HBs	positive positive negative negative	chronic carrier
HbsAg anti-HBc anti-HBs	negative positive negative	* five interpretations possible (see below)

Source: Centers for Disease Control and Prevention (CDC).

*1. May be recovering from acute HBV infection
2. May be distantly immune and test not sensitive enough to detect very low level of anti-HBs in serum
3. May be susceptible with a false positive anti-HBc
4. May be undetectable level of HbsAg present in the serum and the person is actually a carrier
5. Maternal antibody

Incubation period

The incubation period is 45–180 days with an average of 60–90 days.

Public health significance and occurrence

Hepatitis B virus is a major cause of chronic hepatitis, cirrhosis and hepatocellular carcinoma.

An estimated two billion people have been infected with HBV worldwide, 350 million of whom are chronic carriers. Each year an estimated one million people die as a result of HBV infections and over four million new acute clinical cases occur.

In countries with low endemicity (HBsAg prevalence less than 2%) most infections occur in young adults and especially among persons who belong to known high risk groups. Risk of HBV infection increases with:

- unprotected sexual contact
- injecting drug use
- household and sexual contact with known HBV infected persons
- incarceration
- workplace exposure to blood.

In higher endemicity areas (HBsAg prevalence 2%) most infections occur as a result of perinatal transmission from HBsAg-positive mothers or early horizontal transmission via close contact in the household family setting.

Reservoir

Humans.

Mode of transmission

Although hepatitis B surface antigen (HBsAg) has been found in virtually all body secretions and excretions, only blood (serum or plasma), semen and vaginal fluids have been shown to be infectious.

Transmission occurs via percutaneous and permucosal exposure to contaminated blood and body fluids. This may occur during:

- sexual contact
- birth
- injecting drug use
- some household activities such as sharing razors or toothbrushes
- invasive procedures in the community such as tattooing or body-piercing, if there has been inadequate infection control
- invasive medical or dental procedures if there has been inadequate infection control.

All blood and blood products produced for medical purposes in Australia are carefully screened for HBV and other blood-borne viruses using nucleic acid testing.

Period of communicability

The blood of infected persons is infective many weeks before the onset of symptoms and remains infective through the acute clinical course of the disease and during the chronic carrier state, which may persist for life. The proportion of infected individuals who become carriers is inversely related to their age at infection. Persons who are HBV DNA positive are highly infectious.

Susceptibility and resistance

All non immune people are susceptible to infection. Immunity conferred through infection confers lifelong immunity in those who do not become chronic carriers.

Control measures

Preventive measures

Universal vaccination for hepatitis B is part of the Australian Standard Vaccination Schedule. All children are offered a birth dose which should be given within the first seven days after birth and thereafter the infant should receive hepatitis B vaccine at 2, 4 and 12 months of age.

For those not immunised in childhood, the ASVS recommends all pre-adolescent children aged 10–13 years receive hepatitis B vaccine. This is carried out in Year 7 of school. These children receive two doses of adult formulation hepatitis B vaccine.

Health care workers should ascertain their HBV immune status, particularly those engaging in invasive procedures. If infected, health care workers should consult with a medical practitioner to review and consider modifications to their work practices to reduce the risk of transmission to others in accordance with the guidelines of their relevant professional registration boards. Non-immune health care workers should be vaccinated against HBV.

Control of case

All newly acquired cases should be interviewed to identify likely risk factors for their infection and to identify others who may be at risk of infection. If the patient's history suggests nosocomial transmission such as a surgical procedure, or other possible source of infection that may put the general public at risk such as a commercial tattoo, the Department of Human Services should be contacted for further advice and investigation.

Isolation of HBV positive patients is not required. The infected person should be educated about transmission routes, safe injecting and sexual practises, blood and body fluid precautions, and not donating organs or blood.

Control of contacts

Non-immune sexual contacts should be offered hepatitis B immunoglobulin (HBIG) 400 IU IM within 14 days of contact and commence hepatitis B vaccination.

Household contacts should be tested for HBsAg and anti-HBc and offered vaccination if susceptible.

Infants born to HBsAg positive mothers should be given a single dose of HBIG and vaccine within 12 hours of birth, at different sites. The remaining doses of vaccine should be given at 2, 4 and 12 months of age.

Recipients of needle-stick injuries should be considered for hepatitis B immunoglobulin (see Appendix 4).

Control of environment

Appendix 5 outlines procedures for dealing with spills of blood and body fluids.

Outbreak measures

Special settings

Health care workers

Registration boards should be consulted in relation to their policies regarding health care workers with blood-borne viruses. For example, the Medical Practitioners Board of Victoria has a policy on medical practitioners and medical students who carry a blood-borne virus which is available at http://medicalboardvic.org.au. Recommendations are also included in *Infection control guidelines for the prevention of transmission of infectious diseases in the health care setting*, http://www.icg.health.gov.au

Hepatitis C

Victorian statutory requirement

Hepatitis C infection (Group B disease) must be notified in writing within five days of diagnosis.

Specific information is required to be notified under the Health (Infectious Diseases) Regulations 2001. In certain circumstances the attending doctor and patient may be asked to complete a questionnaire to collect additional information. All information collected by the Department of Human Services is treated as confidential. It is used for reasons such as detecting disease trends and to inform public health action and policy development.

School exclusion is not required.

Infectious agent

Hepatitis C virus (HCV) is a small RNA virus that is closely related to the flaviviruses and animal pestiviruses.

Identification

Clinical features

Most infections with HCV are asymptomatic and acute infection may only be detected in patients by the development of elevated serum alanine aminotransferase (ALT) levels. When symptoms and signs do occur, they are similar to other forms of viral hepatitis but usually milder. Estimates vary but between 10% and 50% of people infected with HCV completely recover and are clear of the virus in the subsequent few years. Community based studies report a greater likelihood of viral clearance compared with hospital-based studies. If symptoms of ongoing disease occur they may be non-specific and include fatigue, headaches and nausea.

Method of diagnosis

HCV infection is confirmed by using the combination of a HCV antibody test and PCR to detect HCV RNA. A positive antibody test implies previous infection by the virus and a positive HCV RNA implies ongoing infection.

Antibodies are directed against the products of expressed clones or peptides of the HCV. First generation enzyme immunoassay (EIA) for antibody detection became available in Australia in 1990 and since then second and third generation EIA tests with improved sensitivity and specificity have been developed.

Supplemental tests are also available in the form of recombinant immunoblot assays (RIBA). The significance of equivocal reactivity detected by EIA tests and indeterminate reactivity detected by RIBA testing remains problematic in low risk groups.

A positive HCV RNA test is a marker for viraemia and ongoing infection. A single negative PCR does not exclude infection as viraemia may be intermittent. The patient should be retested in six to 12 months time.

Current EIA tests cannot distinguish between patients who are currently infectious and those who have recovered from infection and developed immunity.

Incubation period

The incubation period ranges from two weeks to six months. It is most commonly six to nine weeks after which serum ALT levels rise. Current HCV antibody tests become positive two to three months after exposure.

Public health significance and occurrence

Hepatitis C occurs worldwide. Current estimates suggest that more than 200 000 Australians have been infected with this virus and that 11 000 new infections are occurring each year. Specific groups such as injecting drug users are at greater risk of HCV infection.

Three quarters of people infected with HCV become chronic carriers of the virus. Of those chronically infected, approximately 10–20% will develop liver cirrhosis over a period of 15–40 years and an estimated 5% will develop hepatocellular carcinoma after 40 years of infection.

There are at least six major genotypes of HCV. At present the main genotypes found in the Australian population are 1 (54%), 3 (36%) and 2 (6%).

Reservoir

Humans.

Mode of transmission

Hepatitis C is primarily transmitted by blood-to-blood contact.

In Australia and other Western countries the sharing of injecting equipment by intravenous drug users is the most common mode of transmission. Tattooing, ear piercing and body piercing using unsterile equipment are other potential sources. There is a high prevalence of HCV in people who have been in prison because of the high likelihood of injecting drug use and tattooing.

Health care and laboratory staff who handle blood and blood products are at increased risk. The Centers for Disease Control and Prevention report that the risk of contracting hepatitis C after percutaneous exposure such as needle stick or sharps injury from the blood of a person with hepatitis C antibody has been estimated at 0–7% (average 1.8%). The risk of transmission is negligible if the source is HCV RNA-negative.

Sexual transmission rates of HCV infection are very low. The risk is increased if the HCV positive partner is immunocompromised as the viral blood titre may be increased, or when there is the possibility of blood-to-blood contact for example sex during menstruation and traumatic sexual practices.

Mother to baby transmission is approximately 5–6% and is thought to occur only when the mother is HCV RNA positive. The likelihood of transmission is increased if the mother is also infected with HIV. Although HCV has occasionally been detected in breast milk there is no evidence that HCV is transmitted from mother to child by breast feeding.

Community or household transmission of HCV is considered rare.

A proportion of HCV positive individuals do not fall into any known risk subgroup. They may have forgotten that they had exposure to injecting drugs many years ago or they may be unwilling to discuss the possibility.

Re-use of poorly cleaned needles by medical practitioners and others in some countries and cultural practices that involve skin piercing are other potential sources of infection.

Period of communicability

Communicability occurs during the acute clinical stage of HCV infection and indefinitely in the chronic carrier stage. All HCV positive individuals should be considered potentially infectious although the risk is minimal in the non-viraemic (PCR negative) individual.

Susceptibility and resistance

All non immune people are susceptible to infection. The degree of immunity following infection is uncertain. If infection resolves and the virus is cleared, the person can be re-infected with the same and other genotypes. However there is some evidence from cohort studies that the likelihood of reinfection is reduced after the first HCV infection.

Control measures

Preventive measures

All health care providers with potential contact with blood or body fluids should use standard precautions.

Use single-use equipment for all skin penetration procedures or use appropriate cleaning, disinfection or sterilisation methods when reusable instruments are used for any procedure. This includes needles.

Control of case

All people diagnosed with HCV infection should be reviewed by a hepatitis specialist (either a gastroenterologist or an infectious diseases physician) and an assessment made of the likelihood of disease progression. Treatment is offered based on the presence of liver fibrosis.

Length of treatment and type of treatment depends mainly on the genotype and sometimes whether previous treatment has failed. Combined therapy with alpha interferon and ribavirin or pegylated interferon and ribavirin are possible treatment regimens.

Counselling of the patient is a very important part of the management. This counselling should include:

- exploring the likely source of the infection
- current knowledge of the natural history
- possible symptoms
- advice on prevention of further transmission of infection
- lifestyle issues such as immunisation against hepatitis A and B, minimisation of alcohol intake, cessation of smoking and healthy diet.

The patient should be advised not to:

- donate blood or body organs
- share injecting equipment
- share personal items such as toothbrushes or razors.

They should also be advised to:

- consider discussing their condition with their health care provider when undergoing any dental or medical procedure
- wipe up any blood spills with single use disposable paper towels and clean area with detergent and warm water
- cover any cuts or wounds with an occlusive waterproof dressing
- place blood-stained paper tissues, sanitary towels or dressings in a plastic bag before disposal

- use safer sex practices. People in long term stable relationships will need to discuss condom use with their health care provider. Safe sex is not routinely recommended among long term monogamous couples.

Control of contacts

There is no vaccine available for the prevention of hepatitis C.

Prophylactic immunoglobulin for contacts has no role.

Control of environment

Not applicable.

Special settings

Health care workers

Registration boards should be consulted in relation to their policies regarding health care workers with blood-borne viruses. For example, the Medical Practitioners Board of Victoria has a policy on medical practitioners and medical students who carry a blood-borne virus which is available at http://medicalboardvic.org.au. Recommendations are also included in *Infection control guidelines for the prevention of transmission of infectious diseases in the health care setting*, http://www.icg.health.gov.au

Antenatal care

Antenatal care should include a comprehensive assessment of hepatitis C risk factors. Women found to be at higher risk of hepatitis C infection or exposure should be encouraged to undergo hepatitis C antibody screening.

Other settings

All workplaces should have policies and procedures in place regarding action to be taken in the event of a blood spill or sharps injury. Further information can be found in *Infection control guidelines for the prevention of transmission of infectious diseases in the health care* http://www.icg.health.gov.au/

Additional sources of information

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Hepatitis D
(delta hepatitis)

Victorian statutory requirement
Hepatitis D infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent
Hepatitis D virus (HDV) is a virus-like particle consisting of a coat of hepatitis B virus (HBV) surface antigen and a unique internal antigen, the delta antigen.

Identification
Clinical features
Onset of disease is usually abrupt, with signs and symptoms resembling those of hepatitis B infection. It may be severe and is always associated with a coexistent HBV infection. Co-infection (simultaneous infection with HBV and HDV) or superinfection (infection with HDV in a person who already has HBV) with HDV is usually more severe than HBV infection alone and more likely to result in fulminant disease. Co-infection has a lower risk of severe chronic disease than does superinfection.

Method of diagnosis
Serological diagnosis is made by:

- detection of total antibody to HDV (anti-HDV). A positive HDV IgM result indicates ongoing replication
- detection of HDV-specific RNA by polymerase chain reaction (PCR) testing. PCR is the most sensitive assay for assessing HDV viraemia.

Incubation period
Approximately two to eight weeks.

Public health significance and occurrence
Hepatitis D occurs worldwide and is most prevalent in countries that have a high incidence of hepatitis B. The highest incidence occurs in parts of Russia, Romania, southern Italy, Africa, pockets of South America and the islands of the Western Pacific.

Despite high rates of hepatitis B in Asian countries the incidence of hepatitis D is lower. Hepatitis D is uncommon in Australia. Three to twelve cases are reported per year in Victoria.

Reservoir
HDV is unable to infect a cell by itself and requires co-infection with HBV to undergo complete replication. Therefore humans with HBV infection act as reservoirs.

Mode of transmission
This virus is transmitted by the same methods as HBV: exposure to infected blood and serous body fluids, contaminated needles, syringes or blood and plasma product transfusions. Sexual transmission may also occur but is less common than with hepatitis B. Perinatal infection is rare. Infection may occur at the same time as a new HBV infection (co-infection) or after someone has been infected with HBV and become a chronic HBV carrier (super-infection).

Period of communicability
This is similar to that of HBV. Persons infected with the HDV are thought to be most infectious before the onset of symptoms. All persons with asymptomatic infection, persons with acute disease and those with chronic carriage of the virus are infectious to others.

Susceptibility and resistance
All people susceptible to hepatitis B infection or those who have chronic hepatitis B can be infected with HDV.

Control measures
Preventive measures
Prevention of hepatitis B infection with hepatitis B vaccine prevents infection with HDV. For persons with chronic hepatitis B infections, the only preventive measure is avoidance of exposure to potential sources of HDV. This means always using a new needle and syringe when injecting drugs and practising safe sex.

Control of case
There is no specific treatment for hepatitis D, although alpha-interferon has been shown to be of some benefit. Expert advice for ongoing management should be sought.

Isolation is not required.

Educate patient about safe injecting, safe sex and blood and body fluid precautions.

Control of contacts
Initiate contact tracing with patient.

Susceptible sexual, injecting and household contacts should be offered hepatitis B vaccine.

Vaccination against hepatitis B prevents HDV infection.

Control of environment
Not applicable.

Outbreak measures
Not applicable.

Hepatitis E

Victorian statutory requirement

Hepatitis E infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

Hepatitis E virus (HEV) is the causative agent.

Identification

Clinical features

The clinical course of disease due to HEV is similar to that of hepatitis A. It is a self-limiting disease of adults aged 15–40 years. A high case fatality rate (up to 20%) has been described in pregnant women affected in their third trimester of pregnancy.

Method of diagnosis

Exclusion of other causes of acute hepatitis, particularly hepatitis A is important. HEV may be detected by immune electron microscopy of faeces collected during the acute phase. Serological tests to confirm HEV infection are available through the Victorian Infectious Diseases Reference Laboratory.

Incubation period

The incubation period varies from two weeks to two months. In different epidemics the average incubation period has varied from 26 to 42 days.

Public health significance and occurrence

Sporadic cases and epidemics in adults have occurred in India, areas of the former Soviet Union, some African countries, Mexico and parts of Asia. The disease is not endemic in Australia and cases reported to date have occurred in travellers, with the exception of one patient with no history of overseas travel who was diagnosed in the Northern Territory in 1995.

Reservoir

Humans and some primates act as reservoirs.

Mode of transmission

Hepatitis E is transmitted via contaminated water and possibly through person to person transmission via the faecal-oral route. Evidence of infection in rats and other rodents in some endemic countries suggests other mechanisms of transmission are likely.

Period of communicability

The period of communicability is unknown. HEV has been detected in stools 14 days after the onset of jaundice.

Susceptibility and resistance

Susceptibility is unknown, however disease tends to occur in adults and pregnant women are at particular risk of fulminating disease.

Control measures

Preventive measures

Good personal hygiene is important, particularly after defecation. Travellers to endemic areas should be advised of the risk and avoid ingestion of potentially contaminated water. There are no vaccines against HEV.

Control of case

Treatment is supportive only, particularly the maintenance of hydration.

Food handlers must not work for at least seven days after the onset of jaundice and until well.

It is recommended that health care workers and child care workers remain away from work for at least seven days after the onset of illness and until well. Children should not attend school or child care for seven days after the onset of symtoms.

Control of contacts

Consider the diagnosis in symptomatic contacts. Immunoglobulin prepared from donors in non-endemic countries will not prevent infection or disease.

If the case has worked as a food handler, child care worker or health care worker, surveillance for further cases in the work place should be carried out.

Control of environment

Infected persons should be advised to maintain strict personal hygiene and avoid preparing meals for others unless adequate food safety can be guaranteed.

Food premises, child care centres or health care facilities where a case has worked whilst potentially infective should be requested to complete a clean up in accordance with the Department's *Guidelines for the Investigation of gastrointestinal illness*.

Outbreak measures

A case with no history of overseas travel would constitute an outbreak in Victoria. Immediate notification is critical to identify the source and prevent further disease. A detailed epidemiological, environmental and laboratory investigation of common exposures, particularly water, amongst cases is necessary.

Additional sources of information

- Heath, TC, Burrow, JN, Currie, BJ, Bowden, FJ, Fishe,r DA, Demediuk, BH, Locarnini, SA, Anderson, DA 1995, 'Locally acquired hepatitis E in the Northern Territory of Australia', *Med J Aust*., vol. 162, no. 6, pp. 318–9.
- World Health Organization, http://www.who.int/csr

Herpes simplex infections

Victorian statutory requirement

Notification is not required.

School exclusion: young children with cold sores who are unable to comply with good hygiene practices should be excluded while the lesion is weeping. Lesions should be covered by a dressing where possible.

Infectious agent

Human herpes simplex virus (HSV) types 1 and 2 cause disease.

Identification

Clinical features

Cold sores are the most common manifestation of herpetic infection and are characterised by a localised primary lesion, latency and a tendency to local recurrence.

In children with atopic dermatitis and immunosuppressed patients, herpes simplex virus may disseminate causing a generalised eruption that requires hospitalisation for intravenous antiviral therapy. Herpes simplex may become chronic in patients with HIV infection with recalcitrant crusted lesions and ulceration. Herpes simplex may be complicated by erythema multiforme which is often more disabling than the infection itself. Herpes simplex virus infection may cause severe extensive disease in immunosuppressed individuals.

HSV types 1 and 2 generally produce distinct clinical syndromes depending on the portal of entry.

HSV type 1

The primary infection may be mild and generally occurs in early childhood before the age of five years. About ten per cent of primary infections cause a more severe form of disease manifested by fever and malaise. This may last a week or more and can be associated with vesicular lesions leading to ulcers in and around the mouth (gingivostomatitis), eye infection (keratoconjunctivitis), a generalised vesicular skin eruption complicating chronic eczema or more rarely encephalitis.

Features of gingivostomatitis include ulceration of the tongue, gums, lips and anterior buccal mucosa, severe systemic toxicity and lymphadenopathy.

Reactivation of latent viral infection in the dorsal root ganglia results in cold sores appearing as clear vesicles on an erythematous base. These usually occur on the face and lips and crust and heal in a few days. This reactivation may be precipitated by trauma, fever, environmental conditions such as windy days, sunburn or intercurrent disease.

HSV type 2

This virus is the usual cause of genital herpes although this can also be caused by type 1 virus. Genital herpes occurs mainly in adults and is sexually transmitted. Primary and recurrent infections occur, with or without symptoms.

The principal sites of primary disease in women are the cervix and vulva. Recurrent disease generally involves the vulva, perineal skin, legs and buttocks. In men, lesions appear on the glans penis or prepuce, and in the anus or rectum of those engaging in anal sex. Other genital or perineal sites as well as the mouth may also be involved in either gender depending on sexual practices.

HSV 2 infections are rarely associated with aseptic meningitis and radiculitis.

Method of diagnosis

The diagnosis may be suggested by cytologic changes in tissue scrapings or biopsy. Confirmation is made by direct fluorescent antibody tests, by isolation of the virus from oral or genital lesions or other sites, or by detection of HSV DNA by nucleic acid testing in lesion or spinal fluid. Techniques are also available to differentiate type 1 from type 2 antibody if required.

Incubation period

The incubation period varies from two to twelve days.

Public health significance and occurrence

Asymptomatic infections with HSV type 1 virus are common. Seventy to ninety per cent of adults have circulating antibodies to HSV type 1 virus indicating previous infection.

HSV type 1 is a common cause of meningoencephalitis. Vaginal delivery in pregnant women with active genital infection carries a high risk of disseminated visceral infection, encephalitis and death to the newborn.

HSV type 2 is frequently associated with sexually transmitted infections and 20–30% of adults have antibody evidence of exposure. The prevalence is greater in socio-economically disadvantaged groups and those with multiple sexual partners.

Reservoir

Humans.

Mode of transmission

Contact with HSV type 1 in the saliva of carriers is the most important mode of spread. Contact of health care workers with patients who are shedding HSV may result in an infection of the tip of the finger (herpetic whitlow). It begins with intense itching and pain and is followed by vesicle formation and then ulceration.

Transmission of HSV type 2 to non-immune adults is usually by sexual contact.

Period of communicability

Secretion of virus in the saliva may occur up to seven weeks after recovery from stomatitis.

Patients with primary genital lesions are infective for seven to ten days. Those with recurrent disease are infectious for four to seven days with each episode.

Susceptibility and resistance

Everyone is susceptible to infection. The disease does not usually confer protective immunity because the virus tends to become latent in dorsal root ganglia of the spine where it may become reactivated at a later date.

Control measures

Preventive measures

No vaccine is currently available.

Health education and personal hygiene should be directed toward minimising transfer of infectious material and reducing the risk of exposure to high risk groups.

Emphasise personal hygiene to minimise the transfer of infectious material. Wear gloves when in direct contact with infectious lesions and wash hands with soap and water afterwards.

Use of latex condoms during sexual intercourse decreases the risk of infection.

Control of case

Non-genital herpes

For symptomatic treatment of minor attacks, use povidone iodine 10% paint applied three times daily. Also consider topical antiviral therapy. Therapy should be self-initiated and commenced at the earliest sign of onset. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Sun protection is important in preventing recurrences of facial herpes simplex.

Specialist advice on systemic antiviral treatment should be sought for:

- severe primary or severe recurrent attacks
- attacks complicated by erythema multiforme
- primary or recurrent attacks in HIV-infected patients or the immunosuppressed.

Patients with active lesions should have no contact with newborns, children with burns or eczema and immunosuppressed patients. Consider caesarean section before the membranes rupture when primary or recurrent genital infections occur in late pregnancy to minimise the risk of neonatal infection.

Contact isolation is required for disseminated severe infections and for infected neonates because of the risk to other neonates or pregnant women.

Anogenital herpes

Patients should be fully screened for other STIs, including HIV infection, on their first presentation.

For initial attack or infrequent recurrent attacks treatment usually consists of valaciclovir, famciclovir or aciclovir. For suppression of frequent recurrent attacks aciclovir or valaciclovir are generally used. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

If there is breakthrough during prophylaxis, higher doses may be successful. Relapse may occur at the cessation of prophylaxis.

Control of contacts

None applicable.

Control of environment

None applicable.

Outbreak measures

None applicable.

Human immunodeficiency virus or acquired immunodeficiency syndrome

Victorian statutory requirement

Both HIV infection and AIDS are Group D notifications. A separate notification form is required for HIV and AIDS diagnoses. Written notification is required within five days of the initial diagnosis.

School exclusion is not required unless the child has a secondary infection.

Infectious agent

Human immunodeficiency virus (HIV) types 1 and 2 are a member of family retroviridae. A number of subtypes exist within HIV–1 and HIV–2.

Identification

Clinical features

AIDS is a severe, life-threatening disease that represents the late clinical stage of infection with the HIV. Several weeks after infection with HIV, a number of infected individuals will develop a self-limited glandular fever-like illness lasting for a week or two. Infected persons may then be free of clinical signs or symptoms for months or years.

Treatment with antiretroviral medication has resulted in fewer cases of AIDS. The burden of illness is now increasingly due to non-AIDS infections, toxicities related to antiretroviral therapy including changes in body shape and metabolic markers such as diabetes and high cholesterol, and neurological and psychiatric manifestations of HIV.

Untreated individuals are at risk of specific opportunistic infections and malignancies and a range of other AIDS indicative diseases. Major diseases that may be indicative of AIDS include:

- *Pneumocystis carinii* pneumonia
- oesophageal candidiasis
- Kaposi's sarcoma
- chronic herpes simplex infection
- cryptococcosis
- cryptosporidiosis
- toxoplasmosis
- cytomegalovirus infection
- mycobacteriosis
- lymphoma
- HIV encephalopathy
- HIV wasting disease

Method of diagnosis

Careful history and physical examination looking for risk factors and clinical manifestations of immunodeficiency are necessary.

Diagnostic testing includes:

- detection of HIV antibody by the ELISA screening test and confirmation by Western blot analysis
- detection of the viral p24 antigen in serum
- PCR tests to detect pro-viral DNA sequences
- HIV culture, although this is only performed in certain special clinical situations.

Incubation period

The period from infection to the primary seroconversion illness is three to eight weeks. The period from infection to development of anti-HIV antibodies is three weeks to three months.

The interval from HIV infection to the diagnosis of AIDS ranges from about nine months to 20 years or longer, with a median of 12 years. There is a group of people with a more rapid onset of disease who develop AIDS within three to five years of infection. Treatment with antiretroviral drugs and disease-specific prophylaxis has resulted in an 80% reduction in AIDS-associated illnesses.

Public health significance and occurrence

Occurrence is worldwide. There were 40 million people living with HIV/AIDS by the end of 2001 and in 2000 three million people died from HIV-related illnesses. The vast majority of HIV infections occur in developing countries.

For the period 1983 to 2003 there was a cumulative total of 4680 HIV diagnoses in Victoria. This represents about 21% of Australia's total. Males accounted for 94% of the diagnoses. Male to male sexual contact including homosexual and bisexual contact accounts for the majority of new diagnoses in men. In females, heterosexual contact and injecting drug use are the most common risk factors.

Reservoir

Humans.

Mode of transmission

HIV can be transmitted from an infected person by:

- Sexual exposure to infected semen, vaginal fluids and other infected body fluids during unprotected sexual intercourse with an infected person. This includes oral sex.

- Inoculation with infected blood, blood products and through transplantation of infected organs such as bone grafts or other tissues, or by artificial insemination with infected semen.
- Breastfeeding of an uninfected infant by an HIV-positive mother. Interventions that decrease the risk of vertical transmission from an infected woman to her child include antiretroviral therapy during pregnancy and caesarean section. Avoiding breastfeeding also decreases transmission. With these interventions the risk of mother to child transmission is less than 5%. If there is no intervention, the risk of mother to child HIV transmission has been estimated to be 20–45%.
- Sharps injuries including needle stick injuries or other exposure to blood and body fluids. The rate of seroconversion following a needle stick injury involving HIV infected blood is said to be less than 0.5%, but this is dependent on the type of needle stick injury (deep versus shallow) and the viral load of the infected person.

Period of communicability

All antibody positive persons carry the HIV virus.

Infectivity is presumed to be life long, although successful therapy with antiretroviral drugs can lower the viral load in blood and semen to undetectable levels.

Susceptibility and resistance

Everyone is susceptible to infection.

The presence of other sexually transmitted infections, especially those with skin or mucosal ulceration, may increase susceptibility.

Control measures

Preventive measures

Preventive measures for HIV centre on personal and institutional factors.

Personal factors include:

- public education on the use of condoms and safer sex practices
- public education should stress that having unprotected sex with unknown or multiple sexual partners and sharing needles (drug users) increases the risk of infection with HIV
- unprotected sexual intercourse with persons with known or suspected HIV infection should be avoided
- HIV-infected persons should be offered confidential counselling and access to screening and treatment for sexually transmissible infections and appropriate antiviral therapy for HIV
- care should be taken when handling, using and disposing of needles or other sharp items
- use of needle exchange programs by injecting drug users should be facilitated.

Institutional factors include:

- use of appropriate infection control measures by all health care and emergency workers
- use of appropriate infection control measures in all premises where skin penetration is carried out, for example electrolysis, tattooing or body piercing
- blood and blood products for transfusion and the donors of tissues and body fluids such as semen should be tested for the presence of markers of HIV
- sharps injuries, including needle stick injuries, and parenteral exposure to laboratory specimens containing HIV should be dealt with according to *Infection control guidelines for the prevention of transmission of infectious diseases in the health care setting* http://www.icg.health.gov.au/
- non-occupational exposure to infected blood or body fluids should be assessed and managed according to Australian National Council on AIDS, *Hepatitis C and related diseases guidelines*, http://www.ancahrd.org/pubs/

Control of case

Standard precautions (see Appendix 3) apply to all patients.

Additional transmission-based precautions apply for specific infections that occur in AIDS patients such as tuberculosis. Equipment contaminated with blood or body fluids should be cleaned and then disinfected or sterilised as appropriate.

Patients and their sexual partners should not donate blood, organs or other human tissue.

All HIV positive persons should be evaluated for the presence of tuberculosis.

Treatment

Anti-retroviral drug therapy is used to treat established HIV infection. As such treatment is specialised and constantly changing, only those doctors experienced in HIV management should prescribe antiretroviral therapy. For further information, see the current edition of the *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). Other treatment includes specific treatment or prophylaxis for the opportunistic infectious diseases that result from HIV infection.

Control of contacts

If a person is diagnosed as having HIV infection, the diagnosing practitioner has a responsibility to ensure that sexual and needle-sharing contacts are followed up where possible.

Assistance with partner notification may be provided by Department of Human Services through its partner notification officers.

Pre and post-test counselling must be provided for all contacts seeking HIV testing.

Control of environment

The procedure for dealing with spills of blood and body fluids is in Appendix 5.

Outbreak measures

The epidemiology of HIV is closely monitored in Victoria and public health action is informed by enhanced epidemiological information notified to the Department.

Special settings

Health care workers

Registration boards should be consulted in relation to their policies regarding health care workers with blood-borne viruses. For example, the Medical Practitioners Board of Victoria has a policy on medical practitioners and medical students who carry a blood-borne virus, which is available at http://medicalboardvic.org.au. Recommendations are also included in *Infection control guidelines for the prevention of transmission of infectious diseases in the health care setting*, http://www.icg.health.gov.au

Antenatal care

Antenatal care should include a comprehensive assessment of HIV risk factors. Women found to be at higher risk of HIV infection or exposure should be encouraged to undergo HIV antibody screening.

Other settings

All workplaces should have policies and procedures in place regarding action to be taken in the event of a blood spill or sharps injury. Further information can be found in *Infection Control Guidelines: for the prevention of transmission of infectious diseases in the health care setting*. http://www.icg.health.gov.au

International measures

WHO initiated a global prevention and control program in 1987. Since 1995, the global AIDS program has been coordinated by UNAIDS. Nearly all countries have developed an AIDS prevention and care program.

Additional sources of information

- Australian Government Department of Health and Family Services 1997, *Contact tracing manual – A practical handbook for health care providers managing people with HIV, viral hepatitis, other STDs and HIV-related tuberculosis*, Australian Government Department of Health and Family Services.
- Australian Government Department of Health and Aged Care 2000, *National HIV/AIDS strategy 1999–2000 to 2003–2004 – changes and challenges,* Australian Government Department of Health and Aged Care, http://www.health.gov.au
- Australian National Council on AIDS, Hepatitis C and Related Diseases, www.ancahrd.org
- Australasian Society for HIV Medicine Inc 2001, *HIV/Viral hepatitis – A guide for primary care*, http://www.ashm.org.au/
- Centers for Disease Control 2001, 'Updated U.S. Public health service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for postexposure prophylaxis', *MMWR*, vol. 50, RR11, pp. 1–42, http://www.cdc.gov/mmwr
- Fleming, DT & Wasserheit, JN 1999, 'From epidemiological synergy to public health policy and practice: the contribution of other sexually transmitted diseases to sexual transmission of HIV infection', *Sexually transmissible infections*, vol. 73, pp. 3–17.

- Venereology Society of Victoria 2002, *National management guidelines for sexually transmissible infections*, Venereology Society of Victoria, http://www.mshc.org.au
- Victorian Department of Human Services 2002, *Victorian HIV/AIDS strategy 2002–2004*, http://www.health.vic.gov.au/ideas
- Working Group of the UK Chief Medical Officer's Expert Advisory Group on AIDS 2000, *Review of the evidence on risk of HIV transmission associated with oral sex – report of a working group of the UK Chief Medical Officer's Expert Advisory Group on AIDS*, Department of Health, London.

Hydatid disease
(echinococcosis)

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
Echinococcus granulosus (dog tapeworm) is the causative agent.

Identification
Clinical features
Hydatid disease in humans is produced by cysts that are the larval stages of the tapeworm *Echinococcus*. Brood capsules are formed within cysts,cysts containing 30–40 protoscoleces. Each of these is capable of developing into a single tapeworm. Symptoms depend on the location of the cyst within the body and develop as a result of pressure, leakage or rupture. The most common site for the cysts is the liver. Less commonly brain, lungs and kidneys are affected. The heart, thyroid and bone are uncommonly affected.

Cysts in the body may remain viable or die and calcify. They may be detected on routine X-rays. The prognosis is generally good and depends on the site and potential for rupture and spread. Sudden rupture of the brood capsules and liberation of the daughter cysts may cause fatal anaphylaxis. Persons who have a calcified cyst detected on X-ray may still have active infection.

Method of diagnosis
Diagnosis may be made plain X-ray, ultrasound or CT scan. If a cyst ruptures, appropriate examination for protoscoleces, brood capsules and cyst wall in sputum, vomitus, faeces or urine should be undertaken.

The Casoni skin test has now been replaced by serological tests for hydatid disease. These include fluorescent antibody (FA) and indirect haemagglutination antibody testing.

Incubation period
The incubation period varies from months to years.

Public health significance and occurrence
Hydatid disease occurs worldwide and is mainly associated with sheep farming.

Notification of hydatid infection ceased in Victoria earlyVictoria early in 2001. In the decade prior to 2001 there was an average of 16 notifications per year. Most of these represented infections acquired overseas. Occasional cases of recently acquired hydatid infection have been identified in visitors to rural areas in Victoria where there are infected sheep or dingoes. Urban dogs which accompany travellers are often suspected of being an intermediary of the cycle of transmission to humans. People who trap wild dogs are similarly at risk.

Reservoir
The domestic dog and other canids, definitive hosts for *E. granulosus*, may harbour thousands of adult tapeworms without being symptomatic.

Felines and most other carnivores are normally not suitable hosts for the parasite.

Intermediate hosts include herbivores, sheep, cattle, goats, pigs, horses, kangaroos, wallabies and camels. Sheep are the major intermediate hosts. Sheep eat the worm eggs from pasture contaminated with dog faeces. These hatch inside the sheep, forming cysts. The life cycle is completed when dogs are infected through eating the offal of infected livestock or wild animals, particularly the liver and lung.

Mode of transmission
Human infection occurs by hand-to-mouth transfer of tapeworm eggs from dog faeces. The larvae penetrate the intestinal mucosa, enter the portal system and are carried to various organs where they produce cysts in which infectious protoscoleces develop.

The important life cycle is dog-sheep-dog. A dingo-wallaby-dingo (or wild dog) sylvatic cycle also occurs. A dog-wild pig-dog cycle has been recognised and poses a special risk for wild pig-hunters.

Period of communicability
Hydatid disease is not transmitted from person to person.

Dogs pass eggs approximately seven weeks after infection. In the absence of reinfection this ends within one year.

Susceptibility and resistance
Young children are more likely to be infected as they are more likely to have closer contact withcontact with infected dogs and they are less likely to have appropriate hygiene habits. There is no evidence to suggest children are more susceptible to infection than adults.

Control measures

Preventive measures

Basic hygiene such as washing hands with soap after gardening or touching the dog and washing vegetables that may have been contaminated by dog faeces, are are important in prevention of this disease.

Control of case

Surgery is often the treatment of choice for infection with Echinococcus granulosus, sometimes combined with prolonged high-doses of the drug albendazole. Percutaneous drainage with ultrasound guidance plus prolonged high-dose albendazole theraphytherapy has been effective for liver cysts. Praziquantel followed by prolonged high-dose albendazole theraphytherapy is used if there is cyst spillage from trauma or surgery. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). Specialist infectious disease advice should be sought.

Control of contacts

Persons carrying the infection are not contagious to others. Encourage contacts to practice appropriate hygiene and to report early any compatible symptoms.

Control of environment

Dogs kept in and around the case's house may require veterinary screening for hydatid infection.

In general, dogs should be treated with an anti-tapeworm medication such as praziquantel every six weeks in rotation with a broad spectrumbroad-spectrum de-worming preparation to prevent disease in dogs and break the life cycle of the parasite.

Review practices that may have led to infection. In particular, restrict dog access to raw offal from infected sheep or kangaroos to prevent the life cycle continuing. Incinerate or deeply bury infected organs from dead intermediate animal hosts.

Outbreak measures

Not applicable.

Additional sources of information

- Victorian Department of Primary Industries, www.dpi.vic.gov.au
- Victorian Department of Sustainability and Environment, www.dse.vic.gov.au

Impetigo (school sores)

Victorian statutory requirement

Notification is not required.

School exclusion: exclude until appropriate treatment has commenced. Sores on exposed surfaces must be covered with a watertight dressing.

Infectious agent

Various strains of *Streptococcus pyogenes*, group A streptococci (GAS) and *Staphylococcus aureus* cause disease.

Identification

Clinical features

Impetigo is a contagious superficial skin infection seen mainly in children but it may occur at any age. The infection may present with mildly irritating blisters that become pustular and erode rapidly leaving a honey-coloured crust. It often appears around the nose and mouth.

Local lymph nodes may be enlarged and the affected child may occasionally be acutely ill.

Impetigo due to *S. pyogenes* is not generally associated with scarlet fever but may rarely cause a glomerulonephritis. This usually occurs three to eight weeks after the skin infection. Skin GAS infections may be an important risk factor for rheumatic heart disease, independent of throat GAS carriage.

Impetigo in the neonate often follows *S. aureus* colonisation of the nose, umbilicus, rectum or conjunctivae. The lesions are initially vesicular and become seropustular and may develop bullae (bullous impetigo). Lesions are most common in the nappy area. Complications are rare.

Staphylococcal skin infections rarely result in the more severe 'scalded skin syndrome' which varies from a diffuse scarlatiniform erythema to a generalised bullous desquamation of the skin.

Method of diagnosis

Diagnosis should be confirmed by isolation of the organism from skin swabs. This also allows confirmation of antibiotic susceptibility.

Incubation period

The incubation period is one to three days for *S. pyogenes* and four to ten days for *S. aureus*.

Public health significance and occurrence

Occurrence is worldwide. Impetigo is a rapidly spreading, highly contagious skin infection that frequently occurs in children's settings such as day care centres, kindergartens and schools.

Reservoir

Humans.

Mode of transmission

The organisms enter through damaged skin and are transmitted through direct contact with patients or asymptomatic carriers. Nasal carriers are particularly likely to transmit disease. It is rarely transmitted by indirect contact with objects.

Period of communicability

If untreated, purulent discharges may remain infectious for weeks to months. Most cases are no longer infectious after 24 hours of appropriate antibiotic therapy.

Susceptibility and resistance

Everyone is susceptible to streptococcal and staphylococcal skin infection.

Persons suffering from chronic conditions producing breaks in the skin, such as eczema or atopic dermatitis, may be at greater risk of impetigo.

Control measures

Preventive measures

Good personal hygiene practices including a daily bath or shower. Emphasise the importance of not sharing toilet articles and of suitably covering cuts and abrasions.

Educate on modes of transmission and possible complications of impetigo and reinforce the importance of treating cases promptly.

Control of case

General therapy may consist of saline or soap and water or aluminium acetate solution or potassium permanganate solution to remove crusts.

For cases where *Streptococcus pyogenes* is suspected or confirmed treatment is generally phenoxymethylpenicillin or benzathine penicillin.

Patients with penicillin hypersensitivity are generally given roxithromycin.

For cases where *Staphylococcal aureus* is suspected or confirmed mupirocin ointment is the usual treatment.

For severe, widespread or longstanding infections flucloxacillin, cephalexin or roxithromycin may be used as each of these drugs is active against both S. aureus and S. pyogenes.

In all cases see the current edition of the *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

General advice for patients with impetigo includes:

- consider using anti-bacterial soap for bathing for two to three weeks
- dispose of soiled dressings appropriately
- emphasise the need for hand washing, especially after changing dressings, and the importance of avoiding sharing toilet articles, towels, clothing or bed linen
- avoid scratching or touching the lesions to prevent spread to other areas of the body
- advise on the importance of completing the recommended antibiotic course.

Patients must be excluded from school or child care services until antibiotic treatment has commenced. Sores on exposed surfaces such as scalp, face, hands or legs must be covered with a watertight dressing.

Control of contacts

Advice to household members should include:

- education about the mode of transmission
- avoiding direct contact with lesions on the affected person if possible
- remembering to wash hands regularly particularly after touching the lesions or scabs of the infected person and use gloves where possible
- refer symptomatic contacts for appropriate treatment.

Control of environment

See Control of contacts, above.

Outbreak measures

Child care settings and schools

- Exclude all confirmed cases and refer suspected cases for appropriate treatment and management.
- Emphasise the need for good hand washing procedures for all staff and children.
- Advise parents of other children and staff who may have had contact with the cases to remain vigilant for signs of impetigo and seek treatment if symptoms develop.
- Ensure that sores on exposed skin surfaces of confirmed cases are covered with a watertight dressing while at school.

Hospital nursery or maternity ward

- Cohort cases and contacts until all have been discharged. Staff working with colonised infants should not work with non-colonised newborns.
- Obtain swabs from discharging lesions to determine organism.

Treat confirmed cases with appropriate antibiotics.

- Draining lesions should be covered at all times with a dressing.
- Trace and determine source of infection. Consider:
  - examining staff for active lesions anywhere on the body
  - obtaining nasal swabs from staff to detect asymptomatic carriers and treating accordingly.
- Promote the need for good hand washing and hygiene practices among staff and visitors to the unit where the outbreak has occurred.
- Investigate adequacy of infection control procedures and the availability of hand washing facilities including antiseptic hand solutions.

Additional sources of information

- Centers for Disease Control and Prevention, *Group A Streptococcal (GAS) Disease*, www.cdc.gov/ncidod
- Shelby-James, TM, Leach, AJ, Carapetis, JR, Currie, BJ, Mathews, JD 2002, 'Impact of single dose azithromycin on group A streptococci in the upper respiratory tract and skin of Aboriginal children', *Pediatr Infect Dis J*, vol. 21, no. 5, pp. 375–80.

Impetigo (school sores) information sheet

What is impetigo?

Impetigo is a contagious skin infection usually caused by either Staphylococcus or Streptococcus bacteria. It is most commonly found in children although it may also occur in adults.

Impetigo may affect skin anywhere on the body but commonly occurs in the area around the nose and mouth. It first appears as a small itchy, inflamed area of skin which blisters. The blisters rupture, release a yellow fluid and develop honey-coloured crusts and form scabs. New blisters develop in the same area or in different parts of the body and may ooze fluid which is highly contagious.

Impetigo is easily diagnosed by the doctor. Occasionally a skin swab may be taken to identify the bacteria responsible for the infection.

How is impetigo spread?

Impetigo is extremely contagious. It can be spread from one person to another through touch or shared items such as clothes and towels. However, a person can also spread it to another part of their own body through scratching or picking at the blisters and scabs.

Who is most at risk of developing impetigo?

Children are most at risk of developing impetigo. Children and adolescents may be more likely to develop impetigo if the skin has already been irritated or injured by other skin problems such as eczema, insect bites, skin allergy or recent cuts or abrasions.

How long does it take until symptoms start?

The incubation period will vary depending on the particular bacteria.

It is usually 1–3 days for streptococcal and 4–10 days for staphylococcal infections.

How is impetigo treated?

- Impetigo is most often treated with antibiotics, either orally or with bactericidal ointment. It is important to follow the recommended treatment and complete the course of antibiotics.
- Treatment involves washing the sores and crusts every 12 hours or as directed with the prescribed soap or lotion. After each wash pat dry.
- Healing should begin within 3 days and the infection eliminated in 7–10 days.
- If the sores spread and get worse despite treatment or the child becomes unwell with fever, see your doctor.
- Cover the sores with an airtight dressing if the child is returning to school in order to reduce the risk of spreading the infection.
- The child's clothes, towels and bedclothes should be changed at least once a day.
- Always remember to wash your hands after touching scabs or sores or handling infected clothing.

How long does impetigo remain infectious?

If untreated, oozing sores remain infectious for as long as they persist.

When can children return to school or child care?

Children can return to school or child care after treatment has started and the sores are completely covered with a watertight dressing.

How can impetigo be prevented?

- Encourage children to wash their hands regularly and always use their own towel and facecloth.
- Cut your child's nails short and encourage them not to scratch scabs or pick their nose.
- Keep injured areas of skin clean and covered to minimise the chance of any bacterial infection, including impetigo.
- Always wash your hands after touching sores or scabs and use gloves if possible when treating infected children.
- Keep children with impetigo away from other children for the period of exclusion. This is until antibiotic treatment has commenced and the sores are covered with a watertight dressing.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Department of Human Services
www.health.vic.gov.au/ideas

Infectious mononucleosis (glandular fever)

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
Epstein-Barr virus (EBV) is the causative agent.

Identification
Clinical features
EBV is an acute viral infection affecting mainly young adults. Clinical features include fever, generalised lymphadenopathy and a sore throat that usually is an exudative pharyngotonsillitis.

Splenomegaly occurs in 50% of patients and jaundice in 4%. In young children the disease is mild or asymptomatic. The duration of symptoms varies from one to several weeks. A variety of uncommon complications have been described and fatalities are exceedingly rare. A chronic form of the disease is suggested as one of the causes of the chronic fatigue syndrome.

Herpes virus 6, cytomegalovirus or toxoplasmosis may cause a syndrome resembling glandular fever, both both clinically and haematologically.

Method of diagnosis
Diagnostic ELISA IgG and IgM antibody testing can be conducted on sera. A full blood examination characteristically shows mononucleosis and a lymphocyte count of 50% or more. PCR testing is available foravailable for CSF or tissue specimens through VIDRL. Virus can also be isolatedbe isolated from throatfrom throat swabs or nasopharyngealor nasopharyngeal aspirates.

ELISA IgG for nuclear antigen,antigen takes two to three months to become positive.

Incubation period
The incubation period is from four to six weeks.

Public health significance and occurrence
Occurrence is worldwide and widespread in early childhood in developing countries. In developed countries the age of infection is delayed until older childhood or young adulthood and is most commonly seen in high school and university students. Only 50% of those infected will develop clinical disease.

Epstein-Barr virus appears to play a causative rolecausative role in:

- Burkett's lymphoma which is a monoclonal tumor of B cells hyperendemic in highly malarious zones of the world
- nasopharyngeal carcinoma, particularly among groups from China and Taiwan
- hairy cell leukemia.

Reservoir
Humans.

Mode of transmission
EBV is transmitted by person to person spread by the oropharyngeal route via saliva, classically by 'tongue-kissing'. Young children may be infected by saliva on the hands of attendants or on toys.

Period of communicability
The period of communicability is prolonged. Pharyngeal excretion may persist for a year or more after infection. Twenty per cent or more of EBV antibody positive healthy adults are long term oropharyngeal carriers.

Susceptibility and resistance
Everyone is susceptible to infection. Infection confers a high degree of resistance. Reactivation of EBV may occur in immunosuppressed individuals.

Control measures
Preventive measures
There is no vaccine available. Basic hygiene can help prevent many diseases including glandular fever. Teach children not to share spoons, forks, cups, soft drink cans or sports water bottles. Adults should not share personal items such as glasses, cigarettes, lipstick or other items that may be covered in saliva.

Control of case
Isolation is not necessary. There is no treatment and antibiotics are not indicated.

Control of contacts
Not applicable.

Control of environment
Not applicable.

Special settings
People with active EBV infection should not visit people receiving organ transplants including bone marrow.

Outbreak measures
Not applicable.

Influenza

Victorian statutory requirement

Influenza (Group B disease) must be notified in writing within five days of laboratory confirmation.

School exclusion: exclude until well.

Infectious agent

Influenza virus (types A, B and C) is the causative agent.

Identification

Clinical features

Influenza is an acute respiratory disease. Symptoms include fever, headache, myalgia, lethargy, coryza, sore throat and cough. Infections in children, particularly type A and B (H1N1) may also be associated with gastrointestinal symptoms such as nausea, vomiting and diarrhoea. Croup is a common presentation in children.

Most symptoms resolve within two to seven days although the cough may persist longer. Complications of influenza include middle ear infections, secondary bacterial pneumonia and exacerbation of underlying chronic health conditions.

During influenza epidemics, patients with early influenza symptoms (fever >38°C, plus at least one systemic symptom such as myalgia, and one respiratory symptom) have a 60–70% chance of having influenza infection.

Method of diagnosis

A clinical diagnosis can be confirmed by culture or antigen testing of appropriate respiratory specimens such as nasopharyngeal aspirate or nose and throat swabs, taken within five days of onset. Or it can be confirmed by serology performed on blood specimens taken during the acute and convalescent stages.

The diagnosis can be confirmed in the laboratory by one or more of the following:

- detection of influenza virus by culture or nucleic acid testing, most commonly polymerase chain reaction (PCR) testing
- demonstration of a significant rise, i.e. fourfold increase in the influenza-specific antibody titre between a serum sample collected in the acute phase and another sample collected in the convalescent phase two to three weeks after onset of symptoms
- a single high influenza-specific antibody titre of five dilutions or greater. This means a titre of 160 or greater, or 128 or greater, depending upon the titration method.

Incubation period

The incubation period is one to four days.

Public health significance and occurrence

Influenza occurs as pandemics, epidemics, outbreaks and as sporadic cases.

Severe disease and complications such as viral and bacterial pneumonia occur primarily among the elderly and those debilitated by a chronic disease.

In temperate zones outbreaks tend to occur in winter. In the tropics they often occur in the rainy season but outbreaks or sporadic cases may occur at any time.

Most human infections are caused by either type A or B influenza viruses. Type A has been associated with widespread epidemics and pandemics, while type B has been infrequently associated with regional epidemics, and type C is only rarely associated with human infection.

Influenza A is sub-typed further. It has two surface antigens (proteins) that are used for sub-typing: haemagglutinin (H) and neuraminidase (N). Since 1918 the only three influenza A sub-types known to usually cause human disease are H1N1, H2N2 and H3N2. Other subtypes such as HSN1 are very rare.

Influenza viruses are formally named according to their type (A, B or C), their sub-type antigenic characterisation and location of first isolation; for example, influenza A (H1N1) or New Caledonia.

The emergence of completely new sub-types of type A virus (antigenic shift) occurs at irregular intervals and is responsible for pandemics. Minor antigenic changes (antigenic drift) are responsible for annual epidemics and regional outbreaks.

Reservoir

Humans are the primary reservoir. Animal reservoirs are suspected as sources of new human subtypes and may occur particularly when people and livestock (for example pigs and poultry) live closely together. In 2004 an outbreak of avian influenza (influenza A H5N1) caused a number of human infections in South East Asia.

Mode of transmission

Influenza viruses are predominately transmitted by airborne spread in aerosols but can also be transferred by direct contact with droplets. Nasal inoculation after hand contamination with the virus is also an important mode of transmission.

Direct contact is important, as the virus will survive some hours in dried mucus particularly in cold and dry environments.

Period of communicability

It is probably communicable for three to five days from clinical onset in adults and up to seven days and occasionally longer in young children.

Susceptibility and resistance

When a new subtype appears, all people are susceptible except those who have lived through earlier epidemics caused by a related subtype.

Infection produces immunity to the specific infecting virus, but the duration and breadth of immunity varies widely. This is partly dependent on host factors, the degree of antigenic drift in the virus and the period of time since the previous infection.

Control measures

Preventive measures

The influenza vaccine in Australia is developed in time for the annual winter rise in 'flu' activity. The strains that are contained in the vaccine are based on the circulating strains in the previous couple of years as well as those circulating in the previous Northern Hemisphere winter. It normally includes representatives of both major influenza A subtypes (H1N1, H3N2) and B strain.

Influenza vaccine is recommended on the Australian Standard Vaccination Schedule annually for all persons 65 years and older.

Free annual influenza vaccine is provided and recommended for the following groups in Victoria:

- all people aged 65 years and older
- all Aboriginal and Torres Strait Islanders aged 50 years and older, and those aged 15–49 years who are at high risk for the complications of influenza including those with:
  - chronic disease such as diabetes, heart, lung, kidney or liver disease
  - decreased immunity
  - living in chronic care facilities
- all public hospital staff in both outpatient and ward settings who provide direct care to patients, to protect themselves and their patients.

Annual influenza vaccination is also recommended for staff working in nursing homes and other chronic care facilities to protect themselves and their patients.

Control of case

Symptomatic treatment alone or with the addition of a neuraminidase inhibitor, if commenced within the first 36 hours of the onset of the illness, can shorten the duration by two to three days. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

For sporadic cases isolation is often unrealistic due to the delay in diagnosis. If cases are still symptomatic they should be advised to remain at home until well and to avoid contact with high risk persons.

Control of contacts

Control of contacts may be of benefit in high risk populations who should be advised to seek medical advice on prophylaxis and to seek early medical review if symptoms develop.

Chemoprophylaxis with amantadine or a neuraminidase inhibitor may be considered in special circumstances against influenza A strains, for example in residential institutions. The potential value of chemoprophylactic drugs must be assessed against their side effects.

Control of environment

Cases and carers should be advised about the importance of hand washing, covering the mouth when coughing, sneezing into disposable tissues, and the appropriate cleaning or disposal of contaminated objects.

Outbreak measures

The most important control measure to prevent serious morbidity and mortality from influenza epidemics is appropriate immunisation. Investigations are generally restricted to outbreaks in groups at higher risk of complications (see Special settings, below).

An influenza pandemic results when antigenic shift leads to a new highly virulent influenza subtype for which there is little or no immunity in the population. Public health action in this setting may

involve a variety of measures to control spread in the community.

Special settings

Aged care facilities, health care facilities and child care centres are all special areas at high risk of influenza outbreaks.

Aged care facilities

Specific infection control measures should be implemented in the event of:

- a laboratory-confirmed case of influenza
- two or more cases of an acute respiratory illness consistent with influenza ('influenza-like illness').

Infection control measures include vaccination of any unvaccinated staff and residents, exclusion of sick staff members, cohorting of resident cases, active case finding and, in some settings, the use of antiviral treatment and prophylaxis.

Health care facilities

Outbreaks of an unidentified respiratory illness in a hospital setting including outbreaks of influenza-like illness are investigated jointly by the Department of Human Services and the hospital's infection control unit.

Child care centres

Outbreaks of influenza or influenza-like illness in child care require exclusion of cases and may warrant prophylaxis for high risk contacts. The Department of Human Services can advise on prophylaxis and infection control procedures.

Additional sources of information

Victorian Infectious Disease Reference Laboratory, *The flu report*, http://www.vidrl.org.au (during flu season).

Invasive pneumococcal disease

Victorian statutory requirement

Invasive pneumococcal disease (Group B disease) requires notification in writing within five days of diagnosis.

Infectious agent

Streptococcus pneumoniae is a gram-positive *Streptococcus* of which 90 serotypes are known to cause disease. Worldwide, approximately 23 serotypes account for the majority of infections.

Identification

Clinical features

Invasive pneumococcal disease commonly presents as septicaemia, meningitis and pneumonia. Septicaemia and meningitis are more common in children (with the exception of Aboriginal children who present most commonly with pneumonia), while pneumonia is more frequent in adults. Other clinical presentations include septic arthritis, peritonitis, pleurisy and pericardial abscess.

Method of diagnosis

Identification of the organism by culturing it from a normally sterile site like blood or cerebrospinal fluid or by nucleic acid tests such as PCR. Rapid antigen detection tests are available but they are of limited use in the diagnosis of invasive disease in children due to the frequency of pharyngeal pneumococcal carriage.

Incubation period

The incubation period is one to three days.

Public health significance and occurrence

S. pneumoniae is one of the most common causes of bacterial meningitis, septicaemia and pneumonia worldwide. Indigenous children in central Australia have the highest reported rates of invasive pneumococcal disease worldwide. The overall incidence rate in Victoria is approximately 9 per 100 000 population per year, with an overall case fatality rate approaching eight per cent. Rates of disease are highest in children aged less than two years and persons aged 65 years and over.

The prevalence of antibiotic resistance is increasing. Approximately 12% of isolates in 2001 in Australia were resistant to penicillin and five per cent were resistant to third generation cephalosporins. The prevalence of antibiotic resistant differs by State and Territory.

Reservoir

S. pneumoniae are commonly found in the upper respiratory tract of humans.

Mode of transmission

Respiratory droplets, direct oral contact or indirect contact through articles freshly soiled with respiratory discharges.

Period of communicability

Bacteria are communicable in respiratory infections, until discharge from the mouth and nose no longer contain virulent pneumococci in significant numbers. Penicillin renders patients with susceptible strains non-infectious within 24–48 hours.

Susceptibility and resistance

Everyone is susceptible to infection, however the risk of invasive disease is highest for those aged less than two years and the elderly. Other risk factors include prematurity and low birth weight, immunosuppressive therapy and exposure to tobacco smoke. Chronic illness such as asplenia, sickle cell disease, cardiovascular disease, diabetes mellitus, cirrhosis, Hodgkin's disease, lymphoma, multiple myeloma, renal failure, nephrotic syndrome, HIV infection and recent organ transplant are also risk factors for infection. Immunity is thought to be serotype specific.

Control measures

Preventive measures

A 7-valent pneumococcal conjugate vaccine (7vPCV) and a 23-valent polysaccharide pneumococcal (23vPPV) vaccine are both available in Australia. Approximately 86% of serotypes causing disease in non-Indigenous children in Australia (55% in indigenous children) are contained in the 7vPCV, and 93% of those causing adult disease are contained in the 23vPPV.

In Australia the 23vPCV is recommended on the Australian Standard Vaccnation Schedule for all persons aged 65 years and over, those in high risk groups and all Indigenous persons aged 50 years or more. The vaccine is funded for all persons aged 65 years and over and indigenous persons aged 50 years or more, and those aged 15–49 years in certain high risk groups.

The 7vPCV is recommended on the ASVS for all children at two, four and six months of age. From 1 January 2005 this vaccine will be funded under the National Immunisation Program. Refer to *The Australian immunisation handbook* (National Health and Medical Research Council).

Control of case

Penicillin remains the treatment of choice until antibiotic sensitivities are obtained. Patients who are allergic to penicillin may be given cephalosporins or erythromycin for pneumonia and chloramphenicol for meningitis. Attempt to obtain blood or CSF specimens prior to commencing therapy however treatment should not be delayed in children and infants, particularly if the clinical presentation suggests septicaemia or meningitis. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Respiratory isolation may be warranted in hospitals for patients with infection due to an antibiotic resistant strain to reduce the risk of transmitting it to other patients at high risk of pneumococcal disease.

Control of contacts

Investigation of contacts is of no practical value.

Control of environment

Disinfect or destroy articles contaminated with discharges from the nose and throat or from other infected sites.

Outbreak measures

In outbreaks in institutions or in other closed population groups immunisation could be considered.

Additional sources of information

- Australian Government Department of Health and Ageing, National Indigenous Pneumococcal and Influenza Program, http://www.health.gov.au
- MJA 2000 'Pneumococcal disease in Australia', *Medical Journal of Australia*, vol. 173, Supplement.
- Roche P & Krause V. 'Invasive pneumococcal disease in Australia' *Comm Dis Intell*, 2001, 24 (4); 505–519.
- Victorian Department of Human Services, Victorian Pneumococcal and Influenza Program, http://www.health.vic.gov.au/immunisation

Japanese encephalitis

Victorian statutory requirement

Japanese encephalitis (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion: case should be isolated until the fever subsides to prevent further mosquito bites.

Infectious agent

Japanese encephalitis virus (JEV) was first isolated in Japan in 1935. However, the disease Japanese encephalitis had been first described in Japan as early as 1871, and since then has been found in Russia, most of the Far East and South East Asia, and more recently it has spread to the Indian subcontinent and Nepal. It is the principal cause of epidemic viral encephalitis in the world, resulting in of the order of 50 000 clinical cases annually.

Of great concern to Australia was the introduction of the JEV into the Torres Strait islands (1995) with two fatal cases of encephalitis and on to the mainland of Australia (Cape York) in 1998. Seropositive pigs were also detected on the mainland. The most likely source of the outbreak in the Torres Strait islands was Papua New Guinea, where the first human cases were detected in 1997.

Identification

Clinical features

Over 90% of Japanese encephalitis virus infections are subclinical. Encephalitis is its serious manifestation. This is clinically indistinguishable from other viral encephalilitides and has a mortality of 20–50%. Up to 50% of patients have serious sequelae.

Method of diagnosis

Confirmation of JEV infection is made by either isolating the virus or by a rising antibody titre.

Laboratory evidence requires one of the following:

- isolation of JEV from clinical material
- detection of JEV viral RNA in clinical material
- IgG seroconversion or a significant increase in antibody level or a fourfold rise in titre of JEV specific IgG proven by neutralisation or another specific test, with no history of recent JE or yellow fever vaccination
- JEV specific IgM in the CSF, in the absence of IgM to Murray Valley encephalitis, Kunjin and dengue viruses
- JEV specific IgM detected in serum in the absence of IgM to Murray Valley encephalitis, Kunjin and dengue viruses, with no history of recent JEV or yellow fever vaccination.

Confirmation by a second arbovirus reference laboratory is required if the case appears to have been acquired in Australia.

Clinical evidence

Febrile illness of variable severity associated with neurological symptoms ranging from headache to meningitis or encephalitis. Symptoms may include headache, fever, meningeal signs, stupor, disorientation, coma, tremors, generalised paresis, hypertonia and loss of coordination. The encephalitis cannot be distinguished clinically from other central nervous system infections.

Incubation period

The incubation period is usually six to sixteen days.

Public health significance and occurrence

The occurrence of JEV disease in Papua New Guinea and probable spread from there to cause disease in the Torres Strait Islands poses a significant threat to Australia. Suitable vector mosquitoes such as Culex annulirostris and vertebrate hosts in the form of water birds are widespread across the mainland. There are also many wild pigs in north eastern Australia to act as amplifiers for the virus. There is a theoretical concern that migratory birds could carry the virus southwards in Australia, even as far as Victoria.

Reservoir

Infection is maintained in enzootic cycles between birds and pigs: water birds (herons and egrets) are the main reservoir for disseminating the virus whilst pigs are important amplifier hosts. Pigs do not show signs of infection other than abortion and stillbirth, but have continuing viremia allowing transmission to man via mosquitoes. Humans and other large vertebrates such as horses are not efficient amplifying hosts, and are therefore ‘dead-end’ hosts for the JEV.

Mode of transmission

In Asia the rice field breeding mosquitoes, mainly Culex tritaeniorhynchus, usually transmit JEV. In the Torres Strait Islands outbreak virus was isolated from Culex annulirostris mosquitoes which were considered to be the main vector involved. Culex gelidus is a new potential vector in Australia if introduced from Asia.

Period of communicability

There is no evidence of transmission from person to person.

Susceptibility and resistance

Infection with JEV confers lifelong immunity.

Control measures

Preventive measures

There is an effective vaccine available. It requires three doses on days zero, seven and 28 with a booster every three years.

Control of case

- Isolate patient and prevent mosquito access until fever subsides.
- Investigate source of infection.

Control of contacts

Not applicable.

Control of environment

Search for and eliminate breeding sites of mosquito vectors in the urban area.

Use mosquito repellents, mosquito nets and other methods of personal protection.

Outbreak measures

Not applicable.

Kunjin virus disease

Arboviruses are viruses which are spread by the bite of arthropods, particularly mosquitoes. They are divided into alphaviruses and flaviviruses.

Victorian statutory requirement

Kunjin virus infection (Group B disease) requires notification within five days of diagnosis.

School exclusion is not required.

Infectious agent

Kunjin virus (KUNV) is a flavivirus and was first isolated from *Culex annulirostris* mosquitoes collected in north Queensland in 1960 and given the name of a nearby aboriginal clan living on the Mitchell River. It is closely related to the West Nile virus which was probably exported from the Middle East to New York in 1999 where it caused thousands of deaths in birds and horses and human disease including fatal encephalitis.

Identification

Clinical features

Serological surveys indicate that subclinical infection is common. Two main clinical forms of disease have been reported: mild disease and encephalitis. Mild disease consisting of lymphadenopathy, fever, lethargy and rash was first noted when two laboratory workers acquired the infection in Queensland in 1963. A few other similar cases have been described in Australia including some with additional muscle weakness and fatigue. There has been a comparatively small number (about six) of reported cases of encephalitis due to Kunjin virus but one source quoted that a total of 15 cases occurred prior to 2000.

Fatalities are rare or absent. Very few epidemiological studies have been carried out to determine the life cycle, nature and frequency of Kunjin virus infection in Australia.

Method of diagnosis

Infection is confirmed by a significant rise in antibody titre to the virus in two blood specimens taken seven to ten days apart.

Laboratory evidence requires one of the following:

- isolation of Kunjin virus from clinical material
- detection of Kunjin virus RNA in clinical material
- IgG seroconversion or a significant increase in antibody level or a fourfold rise in titre of Kunjin virus specific IgG proven by neutralisation or another specific test
- Kunjin virus specific IgM detected in the CSF
- Kunjin virus specific IgM detected in serum in the absence of IgM to Murray Valley encephalitis, Japanese encephalitis or dengue viruses. This is only accepted as laboratory evidence for encephalitic illnesses.

Confirmation of laboratory results by a second arbovirus reference laboratory is required if the case occurs in areas of Australia not known to have established enzootic, endemic or regular epidemic activity.

Clinical evidence

Clinical evidence may present as non-encephalitic, encephalitic and asymptomatic disease.

Non-encephalitic illness

Acute febrile illness with headache, myalgia and/or rash.

Encephalitic disease

Acute febrile meningoencephalitis characterised by one or more of the following:

- focal neurological disease or clearly impaired level of consciousness
- abnormal CT, MRI scan or EEG
- presence of pleocytosis in the CSF.

Incubation period

The incubation period is probably similar to Murray Valley encephalitis virus (MVEV) disease.

Public health significance and occurrence

Kunjin virus has many similarities to MVE virus and disease due to these two viruses can only be distinguished by virological tests. This distinction is important in periods when weather patterns and other portends suggest that an outbreak of MVE virus may be imminent in southeast Australia. This has a higher mortality rate and can be more prevalent.

Serological surveys have shown that Kunjin virus infection has occurred over wide areas of Australia infecting humans, and wild and domestic animals including cattle, sheep and horses. Similarly to MVE virus, Kunjin virus occasionally spreads southward from the tropical north to central and southeastern Australia after heavy rains. Kunjin virus has been detected in Victoria on several occasions since 1974, most recently in 2001.

Reservoir
The virus is endemic in the tropical north of Australia and Sarawak where it has cycles of infection between birds and mosquitoes in enzootic foci.

Mode of transmission
Transmission occurs via mosquitoes, particularly Culex annulirostris.

Period of communicability
There is no evidence of person to person transmission.

Susceptibility and resistance
Infection confers lifelong immunity.

Control measures
Preventive measures
There is no vaccine available.

Kunjin virus infection can be prevented by:

- mosquito control measures
- personal protection measures such as long sleeves and mosquito repellents
- avoidance of mosquito-prone areas and vector biting times at dusk and dawn.

Control of case
Investigate the source of infection. Search for unreported or undiagnosed cases of encephalitis from the Murray-Darling drainage basin.

The patient with suspected infection or friend or relative, should be asked to recall if in the month prior to onset of symptoms he or she had:

- been bitten by mosquitoes
- visited regions where arboviruses are endemic
- participated in recreational or other activities involving exposure to bushland or other mosquito habitat such as gardening, bushwalking, camping and picnicking.

Control of contacts
Not applicable.

Control of environment
To reduce or prevent virus transmission, interruption of human-mosquito contact is required by:

- suppression of the vector mosquito population
- avoidance of vector contact and biting times at dusk and dawn
- applying mosquito control measures in local municipalities
- using personal protection measures such as long sleeves, long trousers, mosquito repellents
- avoiding mosquito-prone areas.

Outbreak measures
Search for unreported or undiagnosed cases of encephalitis from the Murray-Darling drainage basin.

Legionellosis
(Legionnaires' disease)

Victorian statutory requirement
Legionellosis (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion is not required.

Infectious agent
Legionellae are gram-negative bacilli. There are currently more than 45 known species of Legionellae. Those that are known to cause disease in Australia include *L. pneumophila, L. longbeachae, L. micdadei* and *L. bozemanii. L. pneumophila* has 16 identified serogroups. *L. pneumophila* serogroup 1 has been identified as the cause of over 80% of cases in Victoria.

Identification
Clinical features
Legionellosis has two recognised presentations: Legionnaires' disease and Pontiac fever. Only Legionnaires' disease has been reported in Australia.

Legionnaires' disease
This is the pneumonic form of the illness. There is often a severe flu-like prodrome with anorexia, malaise, myalgia and fever. Upper respiratory tract symptoms such as runny nose and sore throat are rare.

Patients may present with any form of pneumonia. As a group they are more likely than other community acquired pneumonias to fulfill criteria for severe disease. There are nearly always radiographic changes on CXR at the time of presentation.

Other features commonly include hyponatraemia, fever greater than 40˚C, renal impairment, diarrhoea and confusion.

Pontiac fever
A non-pneumonic form of the infection has been reported in other countries, presenting as a flu-like illness with fever and malaise lasting two to three days. Although there is said to be a high attack rate (95%), recovery is rapid with no reported deaths.

Method of diagnosis
Various methods of diagnosis for *Legionellae* infection include urinary antigen testing, serology, culture and nucleic acid testing.

Urinary antigen testing
The *Legionella* urinary antigen test is the most rapid and sensitive test currently available but will only detect the most common serogroup, *L. pneumophila* serogroup 1. The antigen test may not become positive for up to five days into the illness and should be repeated if the specimen was taken early in the illness and legionellosis is still strongly suspected.

Serology
Positive *Legionella* antibody results (both IgG and IgM) are common in healthy adult populations. The presence of antibodies is not necessarily indicative of recent infection, especially in acute phase sera. Diagnosis is made by the observation of a significant four fold increase in antibody titre between sera taken in the acute phase and during convalescence three to six weeks later. The two samples should be tested concurrently (in parallel).

Culture
Legionellae are fastidious organisms and will not grow on conventional culture media. Culture for *Legionellae* must be specifically requested if the illness is suspected. Culture is the gold standard and the only method by which human specimens can be compared to environmental samples. Sputum samples for culture should be attempted for public health reasons even if there are already positive serological or urinary antigen results.

Nucleic acid testing
Detection of *Legionella* bacteria DNA in clinical specimens using polymerase chain reaction (PCR) techniques is now available in some reference laboratories. The sensitivities and specificities of such tests are variable. *Legionella* PCR requests should be discussed with the Department of Human Services.

Incubation period
The incubation period for Legionnaires' disease is two to ten days. For Pontiac fever it is 24 to 48 hours.

Public health significance and occurrence
Sporadic and epidemic forms of Legionnaires' disease occur in Australia. *Legionella* infections are believed to account for 5–15% of community-acquired pneumonias.

Outbreaks in Australia are generally associated with man-made water systems including water-cooling towers and spa baths. Home and institutional warm water systems are potential sources of *Legionella* infection but are only rarely implicated in Australia. *Legionella* outbreaks due to contaminated warm water systems are regularly reported from other countries.

Legionellosis in hospitalised and severely immunosuppressed patients carries a much higher case fatality rate.

Reservoir

Legionellae are ubiquitous in the environment. They are often isolated from water and wet areas in the natural environment such as creeks, hot springs, seawater, woodchips, mulch and soil. Potting mix is often colonised with Legionella species, particularly *L. longbeachae*.

Legionellae also thrive in man-made water systems if the water temperature is maintained at 20°C–43°C, which favours the proliferation of the bacteria. These may include cooling water towers associated with air-conditioning and industrial processes, spa baths and household warm water systems for bathing. Shower-heads, nebulisers, humidifiers, ultrasonic misting systems and fountains have also been implicated.

Evaporative air conditioners like those commonly used for domestic cooling are not associated with *Legionella* infections.

Mode of transmission

Legionellosis is generally transmitted through inhalation of contaminated aerosols of water or of dust. Microaspiration of contaminated water may be an important mode of transmission in certain subgroups, such as intubated patients and those receiving nasogastric feeding.

No human-to-human transmission has been recorded.

Susceptibility and resistance

There is a greater risk of more severe legionellosis in persons aged 50 years and over, regular smokers, and the immunosuppressed. More than 70% of infections in Victoria occur in patients over 50 years of age. The disease is extremely rare in children.

Nosocomial infections and infections in severely immunosuppressed patients have a much higher case fatality rate (up to 40%) when compared to the 7% overall mortality rate in Victoria.

Serological surveys identify *Legionella*-specific antibody in 10–20% of healthy adults with no history of clinical legionellosis. It is unclear whether this antibody confers protective immunity.

Control measures

Preventive measures

Smoking is an important risk factor for developing symptomatic infection in those exposed to *Legionella* bacteria, and it is presumed cessation of smoking reduces an individual's risk of infection.

Although total eradication of *Legionellae* from all artificial systems is not possible, the risk of legionellosis can be minimised through diligent maintenance of aerosol generating equipment and ensuring appropriate placement, design and compliance with legislation requirements by owners.

To minimise the risk of infection through potting mix gardeners should be advised to:

- open the bag with care to avoid inhalation of airborne potting mix
- moisten the contents to avoid creating dust
- wear gloves
- wash hands after handling potting mix even if gloves have been worn.

The same measures are also advisable when handling other gardening material such as compost.

Only sterile water should be used in the cleaning of nebuliser medication chambers and in the preparation of aerosol solutions for use in nebulisers or humidifiers. Flushing and instillation of drinking water through nasogastric tubes in intubated or immunosuppressed patients should also only be performed with sterile water.

Control of case

Early antibiotic treatment improves survival. Empirical treatment of severe pneumonia with erythromycin to cover the possibility of legionellosis is recommended.

The patient's environmental exposures during their incubation period are established by interview and compared to other cases.

Exposures of particular concern include:

- contact with hospitals and other health care facilities as a nosocomial source presents the greatest risk to others
- exposure to cooling towers
- use of spas
- use of potting mix.

The Department of Human Services routinely investigates workplaces of confirmed cases.

Control of contacts

Although there is no risk of person to person transmission,amongs an active search for other people who may have been exposed to the same environmental source is commonly undertaken as part of the investigation of cases.

Control of environment

After sampling of suspected environmental Legionellae sources, an immediate precautionary disinfection with an oxidizing biocide is undertaken. Disinfection may be impractical and omitted if the source is organic such as garden potting mix.

All cooling towers in Victoria are required by law to be registered and to undergo regular maintenance and water testing. Records of treatment may be sought and further disinfection may be required depending on the circumstances of the case, and in accordance with regulations.

Outbreak measures

When two or more cases are linked in time and place an investigation is generally undertaken to identify likely *Legionellae* sources in the common area. Environmental sources sampled during the Department of Human Services' investigations such as cooling towers and spa baths are generally requested to be disinfected as a precaution while laboratory testing is conducted.

Special settings

Health care facilities

When a nosocomial source is suspected, immediate testing and disinfection of possible sources is undertaken and active case finding is conducted throughout the institution.

Additional sources of information

- Victorian Department of Human Services Legionella Risk Management Program (including relevant regulations), http://www.health.vic.gov.au/environment

Leprosy
(Hansen's disease)

Victorian statutory requirement
Leprosy (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude until approval to return has been given by the Secretary. Contacts are not excluded.

Infectious agent
Mycobacterium leprae is the causative agent.

Identification
Clinical features

Leprosy is a slowly progressive bacterial infection involving the cooler body tissues, skin, superficial nerves, nose, pharynx, larynx, eyes and testicles. Skin lesions may occur as pale, anaesthetic macules, papules or erythematous infiltrated nodules.

Neurological disturbances are manifested by nerve infiltration and thickening with anaesthesia, neuritis, paraesthesia and trophic ulcers.

The disease is divided clinically and by laboratory tests into two overlapping types: lepromatous and tuberculoid. The lepromatous type (multibacillary or non-immune form) is progressive with nodular skin lesions, slow symmetric nerve involvement, numerous acid-fast bacilli in skin lesions and a negative lepromin skin test. The tuberculoid type (paucibacillary or immune form) is benign and non-progressive with localised skin lesions, asymmetric nerve involvement, few bacilli present in the lesions and a positive lepromin skin test.

Method of diagnosis

Clinical suspicion is the crucial factor in making an early diagnosis of leprosy in non-endemic parts of Australia, like Victoria. Leprosy should always be considered in any undiagnosed patient with chronic skin lesions or a peripheral neuropathy. This is particularly important if they have spent more than brief periods in areas where the disease is endemic, or they have been a contact of a patient known to have leprosy.

Confirmation of diagnosis depends on the form:

- lepromatous disease requires demonstration of plentiful acid-fast bacilli in skin or nasal smears. Skin smears are made by scraping a small amount of tissue fluid from a superficial scalpel cut over a lesion and smearing it on a glass slide.
- tuberculoid disease requires demonstration of typical granulomata with sparse acid-fast bacilli, in biopsies of either skin or nerve lesions.

Incubation period
The incubation period is difficult to determine. It probably ranges from nine months to 20 years with an average of four years for tuberculoid leprosy and eight years for lepromatous leprosy.

Public health significance and occurrence
Leprosy is occasionally detected on routine refugee screening. The world prevalence is estimated to be between ten to 12 million cases. The disease is endemic in tropical and subtropical Asia, Africa, Central and South America, Pacific regions and the USA (Hawaii, Texas, California, Louisiana, Puerto Rico).

Reservoir
Humans.

Mode of transmission
The mode of transmission is not clearly established. The disease is probably transmitted from person to person by aerosol with a high subclinical rate of infection. Household and prolonged close contact seem important. There is anecdotal evidence that rarely it may be transmitted by inoculation, such as by contaminated tattoo needles.

Period of communicability
Leprosy is not usually infectious after three months of continuous treatment with dapsone or clofazimine, or after two to three weeks of treatment with rifampicin.

Susceptibility and resistance
Everyone is susceptible to infection, however study results have suggested a strong age-related susceptibility to being infected or developing disease following close contact with a multi-bacillary case. Children aged between five and nine years are at greatest risk. The risk of progression to leprosy disease following infection is considered to be approximately the same as tuberculosis which is approximately a 10% lifetime risk.

Control measures

Preventive measures

BCG vaccination has some protective efficacy and is recommended for neonates born to a person diagnosed with leprosy.

Control of case

Isolation of tuberculoid (paucibacillary) cases is unnecessary. Isolation of lepromatous (multibacillary) cases is indicated until treatment is initiated, particularly if nasal smears are positive. Nasal discharges of infectious patients should be disinfected or disposed of as infectious waste.

Rifampicin is the key to early control of disease and rapid elimination of the risk of further transmission of infection to contacts. Minocycline can be used as an alternative.

The minimal regimen recommended by WHO for lepromatous leprosy is triple therapy with rifampicin, dapsone and clofazimine for twelve months.

For tuberculoid leprosy, the recommended regimen is rifampicin and dapsone for a period of six months (for detailed treatment regimens and duration, see current WHO recommendations).

Control of contacts

Investigation of contacts and source of infection, and early detection and treatment of new cases is required. Prophylactic BCG has resulted in a considerable reduction in the incidence of tuberculoid leprosy among contacts in some trials. Other studies are currently in progress using a single dose of rifampicin as prophylaxis following leprosy exposure. Currently, treatment of contacts in Australia is not recommended.

Control of environment

Not applicable.

Outbreak measures

Not applicable.

Additional sources of information

- Fine, PE, Sterne, JA, Ponnighaus, JM et al. 1997, 'Household and dwelling contact as risk factors for leprosy in northern Malawi', *Am J Epidemiol*, vol. 146, no. 1, pp. 91–102.
- World Health Organization, http://www.who.int/topics/en/

Leptospirosis

Victorian statutory requirement

Leptospirosis (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

Leptospires are members of the order of Spirochaetes. Pathogenic leptospires belong to the species *Leptospira interrogans* which is subdivided into serovars. In Australia, the most common serovar is
L. interrogans serovar hardjo.

Identification

Clinical features

This group of zoonotic bacterial diseases may present in a variety of manifestations. Common clinical features include fever (which may be biphasic), headache, chills, a rash, myalgia and inflamed conjunctivae. In endemic areas, many infections are either asymptomatic or too mild to be diagnosed.

More severe manifestations occur rarely and include, meningitis, haemolytic anaemia, haemorrhage into skin and mucous membranes, hepatorenal failure, jaundice, mental confusion, respiratory distress and haemoptysis.

The acute illness may lasts from a few days to three weeks or more, with full recovery often taking several months.

Method of diagnosis

Leptospires may be isolated from the blood (days 0 to 7), CSF (days 4 to 10) during the acute illness, and from the urine after 10th day.

The diagnosis is more commonly confirmed serologically by the demonstration of a fourfold or greater rise in *Leptospira* antibody in paired sera taken in the acute phase and at least two weeks later. A single *Leptospira* micro agglutination titre of 400 or greater is also highly suggestive of acute infection.

Incubation period

Typically 10 to 12 days, with a range of four to 19 days.

Public health significance and occurrence

Leptospirosis occurs worldwide in developed and developing countries in both rural and urban settings. The disease is an occupational hazard for farmers, sewer workers, miners, dairy and abattoir workers and fish workers. It is a recreational hazard to bathers, campers and some sportspeople in infected areas.

Farmers, farm workers and meat industry workers in Victoria are the occupational groups most commonly affected by leptospirosis.

Reservoir

Serovars vary with the wild and domestic animal affected. Animal hosts in Victoria include rats, cows and pigs. Asymptomatic kidney infections in carrier animals can lead to prolonged and sometime lifelong excretion of leptospires in the urine.

Mode of transmission

Primarily through contact of skin with water, moist soil or vegetation contaminated with the urine of infected animals. The infection may also be transmitted through direct contact with urine or tissues of infected animals or by the inhalation of aerosols of contaminated fluids, such as may occur in abattoirs. Ingestion of foods contaminated with urine of infected rats is an occasional route of infection.

Period of communicability

Direct transmission from person to person is rare. Leptospires may be excreted in the urine for a month, but urinary excretion in humans and animals for up to 11 months has been reported.

Susceptibility and resistance

Immunity to the specific serovar follows infection, but may not protect against infection with a different serovar.

Control measures

Preventive measures

There is no human vaccine available.

General preventive measures include:

- education for the public on modes of transmission, for example advise to avoid swimming or wading in potentially contaminated waters and to use appropriate personal protection when work requires such exposure
- protecting workers in hazardous occupations with boots and gloves
- rodent control around human habitations
- prompt treatment and isolation of infected domestic animals

The Department of Primary Industries can be consulted for advice on herd immunisation.

Control of case

The usual treatment is doxycycline or benzylpenicillin. Consult the current version of the *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) or seek specialist infectious disease advice.

Although person to person transmission is rare, cases should be nursed with blood and body fluid precautions. Any articles soiled with urine should be disinfected and the patient should be advised that they may continue to excrete leptospires in the urine for a month or more after the acute infection.

Control of contacts

Not applicable.

Control of environment

The exposure history of each case should be investigated to identify and control possible sources of infection such as exposure to infected animals and potentially contaminated bodies of water. Environmental control measures may include environmental clean-ups, and draining or restricting access to potentially contaminated water bodies.

The Department of Primary Industries investigates suspected animal industry sources such as dairies and piggeries, and may recommend animal vaccination or other disease control measures.

Outbreak measures

See Control measures, above.

Additional sources of information

- Victorian Department of Primary Industries, www.dpi.vic.gov.au, (03) 9296 4606 or 13 6186
- WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis, Australia and Western Pacific Region, Queensland Health, http://www.health.qld.gov.au/qhpss

Listeriosis

Victorian statutory requirement

Listeriosis (Group B disease) must be notified in writing within five days of diagnosis.

Laboratories are required to notify *Listeria monocytogenes* isolated from food or water.

Infectious agent

L. monocytogenes is a gram-positive bacterium belonging to the genus *Listeria*. Of the seven recognised species it is currently the only one implicated in human cases.

Identification

Clinical features

Listeriosis predominantly affects:

- people who have immunocompromising illnesses such as leukaemia, diabetes and cancer
- the elderly
- pregnant women and their foetuses
- newborn babies
- people on immunosuppressive drugs such as prednisone or cortisone.

Healthy adults are usually not affected but may experience transient, mild to moderate flu-like symptoms.

Infection in pregnant women may be mild and a temperature before or during birth may be the only sign. However the infection can be transmitted to the foetus through the placenta, which can result in stillbirth or premature birth. Babies may be severely affected with conditions such as septicaemia or meningitis (early-onset neonatal listeriosis).

Late onset neonatal listeriosis generally affects full-term babies who are usually healthy at birth.

The onset of symptoms in these babies occurs several days to weeks after birth (a mean of 14 days), possibly as a result of infection acquired from the mother's genital tract during delivery or postnatally through cross-infection.

In non-pregnant cases listeriosis usually presents as an acute meningoencephalitis or septicaemia. Focal infections such as pneumonia, endocarditis, infected prosthetic joints, localised internal abscesses and granulomatous lesions in the liver and other organs have been described. Symptoms may have a sudden onset. Fever, severe headache, nausea and vomiting can lead to prostration and shock.

The reported case fatality rate has been around 30% in both pregnancy and non-pregnancy related groups.

Method of diagnosis

Listeriosis is diagnosed by isolation of *Listeria monocytogenes* from blood, CSF, placenta, meconium, foetal gastrointestinal contents and other normally sterile sites.

Incubation period

The incubation period is mostly unknown. Outbreak cases have occurred 3–70 days after a single exposure to an implicated product. Median incubation is estimated to be three weeks.

Public health significance and occurrence

Listeriosis is an uncommon disease in humans. In Australia in 2003 the rate was three infections per million population for non-pregnancy Listeriosis cases and 4.6 infections per 100 000 births per year for maternal-foetal infections.

Although most human cases appear to be sporadic, three large outbreaks reported overseas have clearly established *L. monocytogenes* to be a food-borne pathogen. These three outbreaks in the Maritime Provinces (1981), Massachusetts (1983) and Los Angeles County (1985) involved a total of 232 cases. The overall case fatality rate was 36%. The implicated foods were coleslaw, pasteurised milk and Mexican-style soft cheese.

Reservoir

L. monocytogenes is widespread in the environment and commonly isolated from sewage, silage, sludge, birds, and wild and domestic animals. It has caused infection in many animals and resulted in abortion in sheep and cattle. The bacteria are commonly isolated from poultry. It is a common contaminant of raw food.

Asymptomatic vaginal carriage occurs in humans and faecal carriage of up to five per cent in the general population has been reported. The significance of these carriers in the epidemiology of listeriosis is unknown.

Mode of transmission

The main route of transmission is oral through ingestion of contaminated food. Other routes include mother to foetus via the placenta or at birth. The infectious dose is unknown.

Period of communicability

Mothers of infected newborns may shed the infectious agent in vaginal discharges and urine for seven to ten days after delivery. Infected individuals can shed the organisms in their stools for several months.

Susceptibility and resistance

Although healthy people can be infected, the disease generally affects vulnerable groups in the community such as:

- people who have immunocompromising illnesses (such as leukaemia, diabetes, cancer)
- the elderly
- pregnant women and their foetuses
- newborn babies
- people on immunosuppressive drugs (such as prednisone or cortisone).

There is little evidence of acquired immunity even after prolonged severe infection.

Control measures

Preventive measures

It is important to educate people in high risk groups about the foods likely to be contaminated and about safe food handling and storage.

People in high risk groups for listeriosis should avoid the following high risk foods:

- ready to eat seafood such as smoked fish and smoked mussels, oysters or raw seafood such as sashimi or sushi
- pre-prepared or stored salads, including coleslaw and fresh fruit salad
- drinks made from fresh fruit or vegetables where washing procedures are unknown (excluding canned or pasteurised juices)
- pre-cooked meat products which are eaten without further cooking or heating, such as pate, sliced deli meat including ham, strassburg and salami and cooked diced chicken (as used in sandwich shops)
- any unpasteurised milk or foods made from unpasteurised milk
- soft serve ice creams
- soft cheeses, such as brie, camembert, ricotta and feta (these are safe if cooked and served hot)
- ready-to-eat foods, including leftover meats which have been refrigerated for more than one day
- dips and salad dressings in which vegetables may have been dipped
- raw vegetable garnishes.

Safe foods include:

- freshly prepared foods
- freshly cooked foods, to be eaten immediately
- hard cheeses, cheese spreads, processed cheese
- milk- freshly pasteurised and UHT
- yoghurt
- canned and pickled food.

Safe food handling and storage:

- wash your hands before preparing food and between handling raw and ready to eat foods
- keep all food covered during storage
- place all cooked food in the refrigerator within one hour of cooking
- store raw meat, raw poultry and raw fish on the lowest shelves of your refrigerator to prevent them from dripping onto cooked and ready to eat foods
- keep your refrigerator clean and the temperature below 5°C
- strictly observe use-by and best before dates on refrigerated foods
- do not handle cooked foods with the same utensils (tongs, knives, cutting boards) used for raw foods, unless they have been thoroughly washed with hot soapy water between uses
- all raw vegetables, salads and fruits should be well washed before eating or juicing and consumed fresh

- defrost food by placing it on the lower shelves of a refrigerator or use a microwave oven
- thoroughly cook all food of animal origin
- keep hot foods hot (above 60°C) and cold foods cold (at or below 5°C)
- reheat food until the internal temperature of the food reaches at least 70°C (piping hot)
- reheat left overs until piping hot
- when using a microwave oven, read the manufacture's instructions carefully and observe the recommended standing times, to ensure the food attains an even temperature before it is eaten.

Foods are regularly tested for the presence of L. monocytogenes. Processed, packaged ready to eat foods found to be contaminated with *L. monocytogenes* are recalled from sale.

Control of case

Treatment is usually with penicillin or amoxyl/ampicillin either alone or in combination with trimethoprim+ sulfamethoxazole. For penicillin sensitive patients trimethoprim+ sulfamethoxazole may be used alone (see the current edition of *Therapeutic guidelines: antibiotic*, Therapeutic Guidelines Limited).

Investigation/outbreak measures

- Obtain medical history from treating doctor.
- Obtain a food history from patient.
- Test any available suspected foods.
- Assess the possibility of common source outbreaks if there is a cluster of cases.
- Epidemiological investigation of cases should be used to detect outbreaks and to determine source.
- Molecular subtyping should be used to determine the association between isolates from cases and any foods positive for *L. monocytogenes*.
- Investigate the source of any foods found to be positive for *L. monocytogenes* to determine at what point they became contaminated.
- Recall contaminated food if necessary.

Malaria

Victorian statutory requirement

Malaria (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not required.

Infectious agent

Malaria is caused by parasites of the *Plasmodium* spp. Four species of Plasmodium (P.) can infect humans: *P. vivax, P. ovale, P. malariae* and *P. falciparum*. Infection is most commonly caused by *P. vivax* or *P. falciparum*, the latter causing the most severe form of malaria. Mixed infections may occur.

Identification

Clinical features

The most prominent feature of malaria is fever. Classic descriptions of fever with a regular recurring pattern every two or three days is not usually present when the disease begins. Irregular fever also may occur due to mixed infections, ineffective use of prophylactic drugs and partial treatment. Patients commonly feel well on the days when fever is absent.

A presumptive diagnosis of malaria should be made for any person with a high fever who has been to a malarious area until proved otherwise, particularly with recent travel.

Early diagnosis with prompt appropriate treatment is essential as malaria can be a fatal disease. If the initial blood film is negative for malarial parasites it should be repeated within 12–24 hours and preferably when the temperature is rising. One negative test does not exclude the diagnosis, particularly if the patient has taken antibiotics which may result in partial treatment of the infection.

The rapidly rising temperature is commonly associated with shaking chills, muscle pains, back pain, nausea and headache, and the episode frequently ends with profuse sweating. Other symptoms may include confusion or other neurological signs, diarrhoea, dark urine, jaundice, cough and respiratory distress.

The following severe complications may occur, usually with *P. falciparum* infections: coma, acute encephalopathy, cerebral oedema, vomiting, renal failure, severe anaemia, thrombocytopenia, pulmonary oedema, shock, acidosis, coagulation defects, respiratory failure, liver failure and death. Case fatality rates in non-immune people may be 10–40%.

Atypical presentations can occur which predominantly involve a diarrhoeal illness and have resulted in delayed diagnosis and death. Other infections such as the bacterial infection typhoid fever may occur concurrently. These should be looked for, especially if the patient fails to respond well to appropriate treatment.

Individuals who are partially immune or have been taking anti-malarial chemoprophylaxis, may show an atypical clinical picture with wide variations in the incubation period. Malaria due to species other than *P. falciparum* is generally not life threatening except in the very young, very old and those with immunodeficiency or other concurrent disease.

Method of diagnosis

Malaria can be diagnosed by demonstration of malaria parasites in blood films. Blood samples should be sent to a laboratory with experience in the diagnosis of malaria by the use of thick and thin films. Repeated examination may be necessary due to variations in density of parasites. Confirmation of the species should be sought from a reference laboratory.

Incubation period

The time between an infectious mosquito bite and the first detection of parasites in a blood smear is generally 6–16 days. Symptoms may not occur at that time and the first presentation of the infection may be delayed for weeks or months. Commonly, clinical symptoms occur after 7–14 days for *P. falciparum*, 7–30 days for *P. malariae* and 8–14 days for *P. vivax* and *P. ovale*.

Suboptimal suppression with prophylactic drugs may delay the clinical presentation and transmission by blood transfusion usually results in a shorter incubation period.

Public health significance and occurrence

The malaria situation worldwide is deteriorating. There are increasing levels of transmission and it has returned to areas where it had previously been eradicated. Drug resistance has increased and there has been a spread of vector resistance to insecticides.

An estimated 220 million new infections a year occur worldwide. The disease is endemic in areas of Asia, Africa and Central and South America.

The World Health Organization certified Australia free of endemic malaria in 1981 but several hundred imported cases are recorded each year. However the region lying north of a line joining Townsville on the east coast and Port Hedland on the west coast remains receptive and vulnerable to the re-establishment of the disease. This is due to the presence of known or suspected vectors, suitable environmental conditions and the continual arrival of malaria-infected travellers.

Many cases occur among migrants who become infected after re-visiting their native country after a delay of many years when they may have lost their immunity. In Victoria, all malarial patients in recent times have provided travel histories which include countries with endemic malaria. Recently, the countries most commonly associated with imported malaria have been Papua New Guinea, East Timor and Indonesia.

Reservoir

Humans.

Mode of transmission

A female *Anopheles* mosquito ingests gametocytes from an infected human. The parasite must undergo 8–35 days of development within the mosquito before the infective sporozoites are formed. The sporozoites are transmitted to another person via the bite of an infected mosquito.

The disease may also be transmitted by blood or congenitally in untreated or inadequately treated cases.

Period of communicability

Infected cases may remain infectious for years if untreated or inadequately treated so that gametocytes persist. The infected mosquito remains infected for life.

Susceptibility and resistance

People traveling to malarious areas are at risk.

Control measures

Preventive measures

Travellers should be advised of the four principles of malaria protection:

- be aware of the risk, the incubation period, and the main symptoms
- avoid being bitten by mosquitoes, especially between dusk and dawn
- take antimalarial drugs (chemoprophylaxis) to suppress infection where appropriate
- immediately seek diagnosis and treatment if a fever develops one week or more after entering an area where there is a malaria risk.

Personal protection against mosquito bites remains the first line of defence against malaria. Measures to recommend include:

- avoiding outdoor exposure between dusk and dawn
- wearing long, loose clothing after dusk, preferably in light colours
- avoiding perfumes and colognes
- using effective insect repellents, for example products containing up to 20% DEET
- using knock-down sprays, mosquito coils, or plug-in vaporising devices indoors
- using mosquito nets, preferably pre-treated with an appropriate insect repellent.

There is no drug that is completely safe and completely effective for prophylaxis against malaria. The decision to recommend chemoprophylaxis and the choice of drug(s) must involve an analysis of the risks and benefits based on the following considerations:

- prevalence and type of resistance of the malarial parasite to the available drugs
- level of malaria transmission
- duration and place of stay, particularly in rural areas
- intensity of vector mosquito contact
- availability of adequate health care
- age
- traveller's current health and medical history
- risk of traveller not complying with recommendations.

All prophylactic drugs should be taken with unfailing regularity for the duration of the stay in the malaria risk area and should be continued for four weeks after the last possible exposure to infection as parasites may still emerge from the liver and cause disease during this period. The single exception is atovaquone/proguanil, which can be stopped one week after return. Over-reliance on chemoprophylaxis is ill-advised as drug resistance by the malaria parasite continues to change.

Malaria poses a serious threat to pregnant women as it can compromise foetal development, possibly resulting in premature labour or miscarriage. Pregnant women should be advised to avoid travel to malarious areas if possible. Similarly, malaria presents considerable risks for children, particularly the very young, and the choice of suitable drugs is limited. Mosquito avoidance measures should be emphasised.

There is no vaccine available.

Control of case

Isolation of the case is not required. Mosquito contact with the patient should be prevented, especially in tropical areas of Australia where mosquitoes capable of transmitting the disease are present. The country of acquisition of the disease should be determined. It is important to exclude acquisition within Australia or from an unusual source, such as a blood transfusion, that would need further investigation.

Treatment is complex and advice should be sought from an infectious disease physician. Most strains of *P. falciparum* today are resistant to chloroquine. Only *P. falciparum* contracted in some parts of China, Central America and the Middle East are still sensitive to chloroquine. *P. vivax, P. ovale*, and *P. malariae* are sensitive to chloroquine but *P. vivax* resistant to chloroquine has been found in Irian Jaya, Myanmar, Papua New Guinea and Vanuatu.

If the species cannot be identified with confidence, the patient should be treated as for the most serious infection with *P. falciparum*. Although primaquine reduces the risk of relapses of disease, relapses can occur.

Control of contacts

Travelling companions or recipients of any blood transfusion from the case should be warned that they may also be at risk of developing the disease and should seek help promptly if suggestive symptoms develop.

Control of environment

Not applicable as Victoria's ecology is unlikely to sustain endemic malaria, although this is possible in northern areas of Australia.

Outbreak measures

Any outbreaks of malaria in Australia require immediate public health interventions.

Additional sources of information

- Australian Government Department of Foreign Affairs and Trade, http://www.smartraveller.gov.au/
- Centers for Disease Control and Prevention, www.cdc.gov/travel
- World Health Organization, *International Travel and Health booklet*, www.who.int/ith

Measles (rubeola)

Victorian statutory requirement

Measles (Group A disease) must be notified immediately by telephone or fax followed by a written notification within five days.

School exclusion for cases and contacts is:

- cases should be excluded for at least four days after rash onset
- immunised contacts do not need exclusion
- unimmunised contacts should be excluded until 14 days after the first day of appearance of rash in the last case. If unimmunised contacts are vaccinated within 72 hours of their first contact with the first case or if they receive immunoglobulin within seven days of the contact they may return to school.

Infectious agent

Measles virus is a member of the genus *Morbillivirus*.

Identification

Clinical features

Clinical features of measles include prodromal fever, a severe cough, conjunctivitis, coryza and Koplik's spots on the buccal mucosa. These are present for three to four days prior to rash onset.

The most important clinical predictors are included in the clinical case definition for measles which is an illness characterised by all the following features:

- generalised maculopapular rash, usually lasting three or more days, AND
- fever (at least 38°C if measured) present at the time of rash onset, AND
- cough, coryza, conjunctivitis and Koplik's spots.

The characteristic red, blotchy rash appears on the third to seventh day. It begins on the face before becoming generalised and generally lasts four to seven days.

Complications can include otitis media, pneumonia and encephalitis. Sub-acute sclerosing panencephalitis (SSPE) develops very rarely as a late sequela.

Persons who have been previously immunised may present with non-classical features.

Measles infection (confirmed virologically) may rarely occur without a rash.

Method of diagnosis

The diagnosis should be confirmed by demonstration of anti-measles IgM antibody, detection of measles RNA by polymerase chain reaction (PCR) techniques (if available) or by viral culture. The latter is particularly useful for epidemiological purposes.

Suspected cases should be bled at the time of clinical diagnosis. The detection of anti-measles IgM increases to 100% for samples taken 4–14 days after rash onset. If testing is negative for anti-measles IgM on a sample collected three days or less after rash onset, it should be repeated between 4–14 days after rash onset.

The diagnosis can also be confirmed by demonstration of a fourfold or greater change in measles antibody titre between acute and convalescent-phase sera. These should be obtained at least two weeks apart, with the tests preferably conducted in parallel at the same laboratory.

Serodiagnosis is not possible between eight days and eight weeks after measles vaccination. Suspected measles cases who have been recently vaccinated prior to their illness onset can only be confirmed as cases if they have an epidemiological link to a confirmed measles case or if a non-vaccine strain is identified in a clinical specimen by culture or molecular methods.

Incubation period

The incubation period is approximately ten days, but varies from seven to 18 days from exposure to the onset of fever. It is usually 14 days until the rash appears.

Public health significance and occurrence

The use of measles vaccine in infant immunisation programs globally has led to a significant reduction in measles cases and deaths. In addition to providing direct protection to the vaccine recipient immunisation against measles results in the indirect protection of unimmunised persons (herd immunity) if high enough coverage is achieved. Measles vaccine has several major effects on measles epidemiology. These include achieving an increase of the mean age of infection and an increase in the time between epidemics.

Despite the availability of an effective measles vaccine for almost 40 years the disease still causes a considerable burden in many countries. This is primarily because of underutilisation of the vaccine. In 2001 it was estimated that there were 30 million measles cases and 777 000 deaths. Most deaths occurred in developing countries,

principally in Africa and Asia. Thirteen countries reported that routine measles vaccine coverage was below 50%. Large measles outbreaks continue to occur. These occur especially in areas of developing countries with low vaccine coverage and among children living in countries where there are unstable social conditions. These outbreaks frequently have high case-fatality rates.

In Australia, live attenuated measles vaccine was licensed in 1968 and included in childhood vaccination schedules in 1971. Even after the first national measles campaign in 1988, coverage remained too low (85%) to achieve herd immunity. This allowed major measles outbreaks in many areas in 1993–94. In 1994 a second dose of measles-mumps-rubella (MMR) vaccine was introduced for a year's cohort of children aged between 10 and 16 years. Although the incidence of measles declined, seroprevalence studies indicated that further measles outbreaks were likely.

In July 1998 a 'catch-up' campaign was conducted to give a dose of MMR vaccine to all primary school children before lowering the recommended age for the second dose of MMR vaccine to four years in 1999. After the measles control campaign an estimated 96% of children aged five to 12 years had received two doses of MMR.

Although the endemic spread of measles has now been interrupted in Australia, small outbreaks have continued to occur following importation of measles cases from overseas. Adults born after 1966 who have not been vaccinated are most at risk along with the small numbers of unimmunised children.

Reservoir

Humans.

Mode of transmission

Measles transmission is airborne by respiratory droplet nuclei spread or it can be transmitted by direct contact with infected nasal or throat secretions. The virus can persist in the environment for up to two hours. Transmission has been reported to people whose only apparent source of infection was a room presumably contaminated with measles virus when it had been occupied by a patient with measles up to two hours earlier.

Period of communicability

Cases are infectious from slightly before the beginning of the prodromal period, usually five days prior to rash onset. They continue to be infectious until four days after the onset of the rash.

Susceptibility and resistance

Natural infection provides lifelong immunity. A history of prior measles infection should be confirmed serologically before vaccination is deferred as reports of clinical measles infection are not always accurate.

Vaccination at 12 months of age produces a protective antibody in approximately 95% of recipients. The second dose of vaccine, recommended at 4 years, increases protection to approximately 99% of recipients.

Control measures

Preventive measures

Live attenuated measles vaccine is recommended for all persons born after 1966 unless specific contra-indications to live vaccines exist.

It is recommended that this vaccine be given as measles-mumps-rubella (MMR) vaccine at 12 months of age and a second dose at four years of age (prior to school entry). The second dose is not a booster but is designed to vaccinate the approximately five per cent of children who do not seroconvert to measles after the first dose of vaccine.

Older children and adults born after 1966 who are unimmunised and those with serological evidence of non-immunity should be given at least one dose of MMR and ideally a second dose of MMR one month later.

A dose of MMR should also be given postpartum for non-immune women, followed a month later with a repeat dose. Pregnancy should be avoided for 28 days after vaccination.

Unimmunised health care workers, including medical practice staff, born after 1966 are at high risk of measles infection. The measles vaccination status of all health care workers measles should be assessed prior to commencing work and non-immune workers should be vaccinated with two doses of MMR vaccine.

Control of case

There is no specific treatment for measles. Treatment is supportive with particular attention to the possible complications of measles, particularly pneumonia and encephalitis.

The case should be immediately isolated to minimise any possible ongoing transmission. If the case requires hospitalisation they should be nursed in an isolation room, preferably with negative pressure ventilation, using respiratory and standard precautions.

Cases not requiring hospitalisation should be advised to stay at home until they are no longer infectious, usually the fifth day after rash onset. Children are excluded from school or child care for at least four days after the onset of the rash.

The Department of Human Services actively investigates all suspected measles cases to confirm the diagnosis, identify the source of infection, identify other cases, and to identify and protect susceptible contacts in the community.

Control of contacts

The Department of Human Services will trace and manage susceptible community contacts of cases. The responsibility of identifying and protecting susceptible contacts exposed in health care institutions, such as medical practices or emergency departments, is the responsibility of the individual institution.

Control measures require:

1. Identification of susceptible contacts:

- make a list of other people who attended the same area at the same time or within two hours after the visit of the measles patient, including staff
- determine which of the contacts are likely to be susceptible to measles (see below).

2. Protection of susceptible contacts of a measles patient with:

- MMR if within 72 hours of first contact with the patient
- immunoglobulin if longer than 72 hours but within seven days from contact (see below); then offer MMR three months later.

Susceptible contacts are best identified by excluding contacts not considered to be at risk. These are:

- children aged one to four years who have documented evidence of having had one measles vaccine dose
- persons born after 1966 who have had two measles vaccine doses
  - susceptible contacts who have documented evidence of having had at least one measles vaccine dose do not require immunoglobulin but should be offered a second MMR dose
- persons born before 1966, as they are likely to have natural immunity
- persons with documented evidence of immunity through vaccination or natural infection.

Prophylaxis for contacts under 12 months of age

- Infants less than six months of age should not be given MMR and should not be offered immunoglobulin unless the mother is a case, or the mother has been tested and found to have no measles immunity.
- Infants six to nine months of age should not be given MMR but should be offered immunoglobulin if within seven days from first contact with a case.
- Infants between nine and twelve months of age should be offered early MMR if within 72 hours of first contact with a case, and receive a further dose at 12 months of age or four weeks after the first dose, whichever is later. This second dose does not replace the routine dose of MMR at four years but is given because children under 12 months have a lower likelihood of becoming immune (seroconverting) after measles vaccination.

If contact with the infectious case occurred between 72 hours and seven days, immunoglobulin should be offered (see below).

Immunoglobulin prophylaxis

The recommended dose of immunoglobulin is 0.2 mL/kg body weight (maximum dose = 15 mL) given by deep intramuscular injection. Children who have immunodeficiency diseases such as leukaemia, lymphoma, or HIV infection require a higher dose (0.5 mL/kg body weight, maximum dose = 15 mL).

Control of environment

Any room visited by a patient while infectious should be left vacant for at least two hours after the patient has left. This includes medical consulting rooms. Environmental clean-up is not generally recommended, although items contaminated with nasal and throat discharges should be disposed of carefully.

Outbreak measures

Outbreaks in the community setting occur sporadically as a result of imported measles cases exposing local susceptible people. The epidemiology of outbreaks has changed with the introduction of childhood vaccination, with young adults now at highest risk. Outbreaks in schools may still occur if there are significant numbers of unvaccinated students.

The Department of Human Services conducts detailed investigations of clusters of cases.

Special settings

Schools

Although outbreaks mainly affect unvaccinated children, highly vaccinated school populations have also been affected.

Cases are excluded from school and child care for at least four days after rash onset.

Immunised contacts are not excluded. Unimmunised contacts should be excluded until 14 days after the first day of appearance of the rash in the last case.

If unimmunised contacts are vaccinated within 72 hours of their first contact with the first case or if they receive immunoglobulin within seven days of the contact they may return to school.

During an outbreak, children and their siblings who are aged between one and four years should receive their routine second dose of MMR early (but not less than four weeks after their first dose). They are then considered to have completed their MMR vaccination schedule and so do not need a further dose at four years of age.

Child care

If there is a case of measles in a child care setting where infants between six to twelve months of age are present, they should be excluded from attendance for 14 days to interrupt local transmission: Infants can return if they receive MMR vaccination (9 –12 months) within 72 hours of their first contact or if they receive immunoglobulin (6–12 months) within seven days.

It is not necessary for infants under six months to be excluded unless the mother is a case, or where the mother is aware she has no protective immunity.

Additional sources of information

- Communicable Diseases Network Australia New Zealand 2000, *Guidelines for the control of measles outbreaks in Australia*, Communicable Diseases Intelligence technical report series, www.cda.gov.au

Melioidosis

Victorian statutory requirement

Notification is not required although it is recommended that cases of melioidosis with a history of travel to northern Australia be reported to public health units in the relevant state or territory.

School exclusion is not required.

Infectious agent

Burkholderia pseudomallei are small gram-negative, aerobic bacillus. It was previously named *Pseudomonas pseudomallei* or Whitmore's bacillus.

Identification

Clinical features

Pneumonia is the most common clinical presentation of melioidosis, ranging from a mild respiratory illness to a severe pneumonia with septicaemia, with a mortality rate often over 50%. Other presentations include skin abscesses or ulcers, internal abscesses of the prostate, kidney, spleen and liver, fulminant septicaemia, and neurological illnesses such as brainstem encephalitis and acute flaccid paralysis. Asymptomatic infection can occur and in a small proportion of these people the infection can re-activate from a latent form many years later.

Method of diagnosis

A definitive diagnosis of melioidosis can only be made by isolation of the organism from the respiratory tract, lung, blood or other sites.

The likelihood of a bacterial diagnosis is increased by using selective culture media (modified Ashbrown's broth), frequent sampling (sputum, throat, rectal and ulcer swabs) and collection of blood cultures.

Incubation period

Australian data suggests an incubation period of 1–21 days. This can be prolonged in infections which initially become latent.

Public health significance and occurrence

Melioidosis is endemic in South East Asia and northern Australia. It is now recognised in the northern areas of the Northern Territory as the most common cause of fatal community-acquired bacteraemic pneumonia and as the most common cause of severe community acquired sepsis in Thailand. The incidence of disease in Victorian residents is unknown.

In a 10-year prospective study in the Northern Territory 252 cases were identified, with a case fatality rate of 19%. The majority of cases in northern Australia occur during the wet season in November to April. Disease can affect all ages but is more common in adults and predominantly occurs in males and Australian Aboriginals. Risk factors for disease include diabetes, chronic lung and renal disease and excess alcohol consumption.

Reservoir

Burkholderia pseudomallei have been found in soil and water in tropical regions of northern Australia and South East Asia.

Mode of transmission

Infection is thought to be acquired through percutaneous inoculation, although inhalation and ingestion are also possible.

Period of communicability

The disease is only very rarely transmitted person to person.

Susceptibility and resistance

Disease in humans is uncommon even among people in epidemic areas who have close contact with soil or water containing the infectious agent. Approximately two thirds of cases have a predisposing medical or recrudescence in asymptomatic infected individuals.

Control measures

Preventive measures

There is no vaccine available. Basic hygiene can help limit the spread of many diseases including melioidosis and measures such as wearing shoes outside may prevent transmission.

Control of case

A history of travel to northern Australia or tropical regions of South East Asia should be determined.

Initial intensive antibiotic therapy usually consists of trimethoprim/sulfamethoxazole with ceftazidime, meropenem, or imipenem. The trimethoprim/sulfamethoxazole component is usually continued for three months to ensure eradication. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited). Specialist infectious disease advice should be sought for all cases.

Follow-up of cases and adherence to eradication therapy are critical to prevent relapse, which can be fatal.

Control of contacts
Investigation of potential sources is important. Human carriers are not known.

Control of environment
Not applicable.

Outbreak measures

Melioidosis has been identified as a potential bioterrorism agent. Any case or cases presenting without a clear history of exposure in an endemic area should be reported to the Department of Human Services for further investigation.

Additional sources of information

- Currie, BJ, Fisher, DA, Howard, DM, Burrow, JNC et al. 2000, 'Endemic melioidosis in tropical northern Australia; a ten year prospective study and review of the literature', *Clin Infect Dis*, vol. 31, pp. 981–986.
- Currie, BJ 2000 'Melioidosis: an Australian perspective of an emerging infectious disease', *Recent Adv Microbiol*, vol. 8, pp. 1–75.
- Currie, BJ, Fisher, DA, Howard, DM, Burrow, JNC et al. 2000, 'The epidemiology of melioidosis in Australia and Papua New Guinea', *Acta Tropica*, vol. 74, pp. 121–127.
- Northern Territory Centre for Disease Control, http://www.nt.gov.au/health

Meningococcal disease

Victorian statutory requirement

Suspected or confirmed meningococcal infection (Group A disease) must be notified immediately by telephone or fax followed by a written notification within five days.

School exclusion: cases should be excluded until adequate carrier eradication therapy has been completed. Contacts do not need to be excluded if receiving carrier eradication therapy.

Infectious agent

Neisseria meningitidis (the meningococcus) is a gram-negative diplococcus. Various serogroups have been recognised including groups A, B, C, 29E, H, I, K, L, W135, X, Y and Z. Within these groups, certain serotypes have been identified, for example, group B serotypes 2b and 15. In 2004 groups B and C were the most common in Victoria.

Identification

Clinical features

Clinical features of meningococcal infection include an acute onset of meningitis or septicaemia. Typical features of these include fever, intense headache, nausea, vomiting and neck stiffness. There may be a petechial or purpuric rash on the trunk and limbs that may sometimes cover large areas of the body. In fulminating cases there is sudden prostration and shock associated with the characteristic rash and this condition has a high fatality rate. Chronic meningococcal septicaemia can also occur with febrile episodes, skin rashes and fleeting joint pains.

Method of diagnosis

Diagnosis is usually made on clinical grounds confirmed by laboratory tests. Laboratory tests include:

- gram stain of cerebrospinal fluid, skin lesion smear or joint fluid
- culture of blood, CSF or other sterile site
- polymerase chain reaction (PCR).

Meningococcal isolates from all cases of invasive disease should be sent to MDU at the University of Melbourne to ensure appropriate monitoring of serogroups and to perform susceptibility testing. This needs to be authorised by the Communicable Diseases Section of the Department of Human Services.

Incubation period

The incubation period is commonly three to four days, but can vary from two to seven days. People who do not develop the disease in the seven days after colonisation may become asymptomatic carriers.

Public health significance and occurrence

Invasive meningococcal infections occur in endemic and epidemic forms. In Australia epidemic disease has not occurred for many years. Endemic disease is at low levels of incidence and cases are generally unrelated to each other. Despite this, invasive meningococcal disease is of public health importance and is frequently a cause of public alarm and receives a high level of media attention.

Meningococcal disease characteristically has a seasonal pattern with a peak of incidence in the winter and spring months. Although the reasons for this seasonality are not clear, there is evidence that influenza virus or *Mycoplasma pneumoniae* infections may predispose to invasive disease and that closer personal contact or lack of ventilation may facilitate transmission of meningococci.

The three major serogroups of meningococci cause different patterns of disease. Serogroup A meningococci cause outbreaks of infection in areas such as the meningitis belt of Africa where the incidence of meningococcal infection rises sharply towards the end of the dry season and declines rapidly with the onset of rains. The epidemics occur in 8–14 year cycles. Since 1990 New Zealand has been experiencing an epidemic of serogroup B meningococcal disease. Age-standardised rates for Maori and Pacific Island people were three and six times higher respectively than for the European population. Serogroup C meningococci are usually associated with sporadic disease but can cause small or large outbreaks. Attack rates for serogroup C are between those seen with serogroups A and B.

Meningococcal disease has had cyclical peaks of incidence. Notification of 'meningitis' reached a peak of 33.1 cases per 100 000 in 1942 (2371 cases) as part of a pandemic of serogroup A disease during World War II. Apart from another peak of activity in the early 1950s, there was a steady decline of notifications to less than 0.5 cases per 100 000 in 1987. The notification rate for meningococcal disease to the National Notifiable Diseases Surveillance System (NNDSS)

has been slowly increasing over the past 10 years from 1.6 per 100 000 in 1991 to 3.1 per 100 000 in 2000. In 2002 there were 129 notifications in Victoria (1/3 of the national total) of which 47 were serogroup B and 72 were serogroup C.

Reservoir

Human.

Mode of transmission

Respiratory droplets shed from the upper respiratory tract transmit meningococci from one person to another. Humans are the only natural hosts for meningococci and the organism dies quickly outside the human host. It is not able to be isolated from environmental surfaces or samples.

Salivary contact has in the past been regarded as a means of transmission of meningococci. There is little evidence to support this view. Available evidence indicates that neither saliva nor salivary contact is important in the transmission of meningococci. Saliva has been shown to inhibit the growth of meningococci. Carriage of meningococci has not been convincingly shown to be associated with saliva contact. A case-control study of United Kingdom university students found no association between carriage of meningococci and sharing of drinks or cigarettes and a weak association with 'intimate kissing' (OR = 1.4 & 95% CI, 1.0–1.8%).

It is unclear whether carriage in these circumstances is due to saliva contact rather than to droplets shed during household-like (close and prolonged) contact.

Period of communicability

It is communicable until the organisms are no longer present in discharges from the nose and mouth.

Susceptibility and resistance

Susceptibility to clinical disease is low as evidenced by the usual high ratio of carriers to cases. Susceptibility decreases with age although a secondary peak of meningococcal meningitis occurs in adolescents and young adults in the age group of 15–24 years. Patients deficient in certain complement components in the blood are prone to recurrent meningococcal infections. There is an increased and prolonged risk of secondary infections in close contacts. In one series, the incidence of such infection was 0.5% with a median interval of seven weeks between the index and secondary cases. Secondary cases have been reported up to five months later. The risk in household contacts is 500 to 800 times higher than in the general population.

There is no maternal immunity.

Control measures

Preventive measures

Note that most strains of meningococci do not cause disease, but instead provide protection. Other protective bacteria such as *Lactamicas* (*Neisseria lactamica* spp) also colonise the nasopharynx. By giving chemoprophylaxis when it is not needed these bacteria, which are protective, are also eradicated. People can carry meningococci with no ill effects for many months. Carriage produces protection.

There is no evidence to suggest carriers will suddenly become cases after weeks or months of carriage.

Conjugate vaccines are available that can give long lasting protection against meningococcal serogroup C disease. There is no vaccine for meningococcal serogroup B disease. There is polysaccharide quadrivalent vaccine available in Australia against groups A, C, Y and W135 however it cannot be given under two years of age and only protects for one to five years. This vaccine is considered a 'travel' vaccine for travellers to epidemic and highly endemic areas such as Brazil, Mongolia, Vietnam, India, Nepal and sub-Saharan Africa and is a requirement for visits to Mecca.

There are different brands of (conjugate) meningococcal serogroup C vaccine available. The vaccines contain meningococcal serogroup C 'sugars' joined with an inactive protein of either diphtheria or tetanus toxoid and additives aluminium phosphate or hydroxide.

Under the National Immunisation Program, a single dose of meningococcal serogroup C vaccine is given at 12 months of age. If parents wish to purchase vaccine to immunise their child prior to 12 months of age, infants from six weeks to four months of age at the commencement of vaccination receive three doses one to two months apart. Babies from four months to 11 months at the commencement of vaccination receive two doses one to two months apart.

The National Meningococcal C Vaccination Program is a four year program from 2003–2006 in which all persons aged 1–19 years in 2003 are eligible for a dose of meningococcal C vaccine.

Control of case

Prompt treatment with parenteral penicillin in adequate doses should begin when a presumptive clinical diagnosis is made prior to laboratory confirmation. In cases with a very acute onset, such treatment should commence prior to transfer of the patient to hospital even though there may be some interference with laboratory confirmation by culture. Suitable alternatives for patients who are allergic to penicillin include ceftriaxone, cefotaxime or chloramphenicol.

Since penicillin only temporarily suppresses but does not eradicate the organisms in the nasopharynx, all patients should be treated with a drug such as rifampicin prior to discharge from hospital. If ceftriaxone is used in treatment rifampicin need not be given. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Control of contacts

Meningococci are likely to have been acquired from an asymptomatic person (carrier) who either lives in the same household or is a sexual partner of the sick person. Children tend to acquire their disease from adults (in their household) whereas teenagers and adults are more likely to acquire their disease from close friends.

Clearance antibiotics should only be given to the following people (see below) who have had contact with the case seven days prior to the onset of the case's illness. They should be commenced as soon as possible after diagnosis.

Contacts include:

- **household contacts** are defined as those people living in the same house and include recent visitors who stayed overnight in the seven days preceding the onset of the case's illness
- **dormitory contacts** in boarding schools, military barracks, school camps, and hostels in the seven days preceding the onset of the case's illness
- **sexual (intimate) contacts**
- **medical, nursing, or paramedical staff** that have performed mouth-to-mouth resuscitation or intubation or suction or similar intimate treatment with a case of meningococcal disease *prior to being started on therapeutic antibiotics*.

The risk of meningococcal disease in close contacts, whilst higher than the general population, is still very low. The risk is highest in the first seven days after a case and falls rapidly during the following weeks. If antibiotic prophylaxis is not given, the absolute risk to an individual in the same household one to 30 days after an index case is about one in 300. The increased risk in household members may be due to a combination of genetic susceptibility in the family, increased exposure to virulent meningococci and environmental factors.

Clearance antibiotics should only be given to those people who are at risk of either being the source of disease in the case, or of having acquired the invading organism from the case. The aim of clearance antibiotics is to prevent further transmission.

There are three antibiotics currently used for the chemoprophylaxis of meningococcal disease. Each agent has advantages and disadvantages and each is the preferred agent in specific circumstances.

Clearance antibiotic for contacts of meningococcal disease:

Rifampicin can be dispensed for meningococcal prophylaxis as syrup for children or in capsules for older children and adults. The product information should be consulted for the adverse events and side effects of rifampicin, although it should be noted that the product information recommends a once-daily four-day regimen of rifampicin for the chemoprophylaxis of meningococcal disease. The two-day regimen (below) is recommended by the Communicable Diseases Network Australia.

Age	Dose	
0–2 months	1 mL syrup*	Twice daily for 2 days
3–11 months	2 mL syrup*	
1–5 years	7.5 mL syrup*	
6–12 years	300 mg capsule	
> 12 years	600 mg capsule	

*Rifampicin syrup contains 100 mg/5 mL

Ciprofloxacin: Adults 500 mg orally, single dose (minimum age 12 years and weight >40 kg). This is preferred in women taking the oral contraceptive pill. Although ciprofloxacin is not registered for chemoprophylaxis of meningococcal disease in Australia the Communicable Diseases Network Australia recommends it for that purpose.

Ceftriaxone: Adults 250 mg IM (recommended for pregnant contacts). This may be dissolved in lignocaine 1% solution to reduce pain at the injection site. Dosage for children (<12yrs) is 125 mg IM. Not for infants under 1 month of age.

Control of environment

Respiratory isolation is recommended for 24 hours after commencing treatment. There should be concurrent disinfection of discharges from nose or throat and articles soiled with such discharges. Articles used by the patient should be terminally cleaned.

Outbreak measures

When there are two or more cases in four weeks of exactly the same strain in a childcare centre, school or university, all students and staff in the same class or in the same group as the case will be given clearance antibiotics. If serogroup C disease is identified a vaccination campaign may be instituted.

Clusters of invasive meningococcal disease in people who have had a low level of salivary contact like footballers who have shared drink bottles or churchgoers who have shared a communion cup appear to be very rare. Although clusters have been described, for example, in association with sporting events and sports clubs, the reported details indicate that point-source salivary transmission was not involved. Secondary cases in situations where dribbling of saliva is common such as child day-care centres are also rare.

Additional sources of information

- Australian Government Department of Health and Ageing 2003, 'Annual report of the Australian Meningococcal Surveillance Programme', *Communicable Diseases Intelligence*, vol. 27, no. 2.
- Communicable Diseases Network Australia 2001, *Guidelines for the early clinical and public health management of meningococcal disease in Australia*.
- Public Health Laboratory Service 2002, Meningococcus forum: *Guidelines for public health management of meningococcal disease in the UK*, http://www.hpa.org.uk/infections
- Victorian Department of Human Services, *Advice for medical practitioners*, http://www.health.vic.gov.au/ideas

Molluscum contagiosum

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
Molluscipoxvirus is a member of the pox virus (Poxviridae) family.

Identification
Clinical features
This is a viral disease of the skin that produces firm, smooth, spherical, pearly white papules with a central dimple. Most papules are 2–5 mm in diameter, although papules may coalesce to form larger lesions.

Lesions in adults are more common on the lower abdomen, genitalia or inner thighs. In children lesions are more common on the face, trunk, and limbs. Lesions may disseminate more widely in patients with HIV infection.

Molluscum contagiosum may persist for six months to two years without treatment. Lesions may resolve spontaneously or possibly as a result of inflammatory responses secondary to bacterial infection or trauma.

Method of diagnosis
The virus has not yet been cultivated. Diagnosis can be confirmed by microscopy (the core of the lesion is expressed onto a slide then stained), by histology or by visualisation of the vesicle fluid by electron microscopy.

Incubation period
The incubation period is unknown. Clinical reports suggest a range from seven days to six months.

Public health significance and occurrence
Molluscum contagiosum infection occurs worldwide. Surveys in other countries suggest peak incidence occurs during childhood.

Lesions typically resolve without complication. Molluscum contagiosum may be more severe and more persistent in immunosuppressed patients and particularly in patients with HIV/AIDS.

Reservoir
Humans.

Mode of transmission
Molluscum is transmitted by direct contact, fomites or sexual contact. Autoinoculation through scratching is also suspected.

Period of communicability
The period of communicability is unknown but probably as long as the lesions persist.

Susceptibility and resistance
Any age may be affected although infection is more common in children. Infection is more common and more severe in the immunosuppressed.

It is unknown whether prior infection confers any protection against subsequent exposures.

Control measures
Preventive measures
Avoid close contact with the lesions of affected persons. Avoid sharing baths and spas with patients with lesions, and do not share face or bath towels.

Control of case
Isolation of case is not required. Infected children should either avoid contact sports or ensure lesions are adequately covered during play. No school exclusion is required.

Transmission through warm water is only very rarely observed. The risk of transmission through public swimming pool contact is very low and exclusion is rarely if ever necessary.

Many treatments are cited in the literature with destruction of the lesions as their common goal but there is minimal evidence to support them. Watchful waiting may still be the best option for many patients. Phenol ablation may produce significant scarring. Other topical preparations are under investigation.

Control of contacts
Not required.

Control of environment
Not required.

Outbreak measures
Consider suspending direct contact and sporting activities.

Additional sources of information
- Australian College of Dermatologists, http://www.dermcoll.asn.au/

Molluscum contagiosum information sheet

What is molluscum contagiosum?

It is a viral disease caused by the molluscum contagiosum virus (MCV).

How is it transmitted?

It is transmitted by skin-to-skin contact, which is usually sexual in adults and non-sexual in children. Towels and other objects might transmit occasional cases. Spots appear one week to six months (usually two to three months) after contact.

Signs and symptoms

Molluscum contagiosum may cause a variable number of skin lumps, sometimes even ten or twenty. The lumps may be located on or around the genitals. This includes the pubic area, the inner thighs and the abdomen. They are painless, but may be slightly itchy and often pearly white in appearance with a tiny central indentation.

Molluscum contagiosum can be mistaken for genital warts or pimples. If you think you have molluscum contagiosum, it is recommended that you see your doctor or other experienced health professional.

Can it become a serious disease?

Bacterial infections can complicate molluscum contagiosum, causing the lumps to become red and sore. However, these symptoms may also be signs that the lumps are about to disappear naturally. If symptoms don't resolve quickly, consult your doctor in case you need antibiotic treatment.

How is it treated?

Although molluscum contagiosum eventually resolves, freezing the lumps with liquid nitrogen can shorten the duration of symptoms.

A single treatment is all that is usually required. However, it may take a couple of weeks for the lumps to disappear.

Imiquimod cream 0.1% applied three times daily is also effective.

Do not scratch any irritated areas after treatment, as this may spread the infection. If no treatment is provided it is a mild, self-limiting disease. It can persist for six months to two years. However, any one lump will usually clear up in two to three months.

Further information

- Your local doctor
- Better Health Channel http://www.betterhealth.vic.gov.au
- Melbourne Sexual Health Centre http://www.mshc.org.au
- Victorian Department of Human Services, 1300 651 160

Mumps

Victorian statutory requirement

Mumps (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude for nine days or until swelling goes down, whichever is sooner.

Infectious agent

Mumps virus is a member of the family *Paramyxoviridae*.

Identification

Clinical features

Mumps is an acute febrile disease characterised by swelling and tenderness of one or more of the salivary glands, usually the parotid and occasionally the sublingual or submaxillary glands. Respiratory symptoms can occur, particularly in children under five years. Epididymo-orchitis occurs in up to a third of postpuberal males and is most commonly unilateral: sterility is an uncommon complication. Oophoritis occurs in up to 31% of females aged over 15 years and may cause lower abdominal or back pain. Many infections in children less than two years of age are subclinical. Mumps meningitis is a fairly common complication. It usually occurs two to ten days after the onset of parotitis and is self-limited with symptoms lasting three to five days.

Mumps very rarely causes sensorineural deafness, encephalitis and pancreatitis. Mumps during the first trimester may increase the risk of spontaneous abortion but there is no evidence that mumps during pregnancy results in congenital malformations.

Method of diagnosis

The predictive value of parotitis in the diagnosis of mumps is reduced in countries with high immunisation rates such as Australia. The diagnosis should be confirmed serologically by the detection of mumps specific IgM antibody, or a significant rise in mumps IgG antibody in acute and convalescent sera. Mumps virus can also be cultured from swabs of the buccal mucosa and from urine.

Incubation period

The incubation period ranges from 14 to 25 days. It is commonly 15–18 days.

Public health significance and occurrence

Occurrence is worldwide. There is generalised spread of the infection in communities with low immunisation rates; serologic studies show 85% or more of individuals in these communities have evidence of previous mumps infection by adult life. High childhood immunisation rates in Australia have resulted in a dramatic reduction in rates of mumps infection. Unimmunised children and adults, especially males, are the groups at highest risk of infection.

Reservoir

Humans.

Mode of transmission

Transmission occurs through via respiratory aerosols and respiratory droplet spread or by direct contact with contaminated saliva.

Period of communicability

Mumps is communicable from six to seven days before to nine days after the onset of parotitis. Asymptomatic and inapparent cases can also be infectious.

Susceptibility and resistance

Immunity is generally life long and develops after either inapparent or clinical infections. Individuals born prior to 1970 have a high likelihood of natural immunity even if they have had no history of clinical infection.

Control measures

Preventive measures

Live attenuated mumps vaccine is available combined with rubella and measles vaccine (MMR). Vaccination with this vaccine results in seroconversion to all three viruses in over 95% of recipients. Since the MMR vaccine viruses are not transmissible, there is no risk of infection originating from vaccines.

MMR vaccination is recommended for all children at 12 months of age, unless specific contra-indications to the vaccine exist. A second dose is recommended at four years of age, prior to school entry.

Control of case

There is no specific treatment. Cases requiring hospitalisation should be nursed in an isolation room using respiratory precautions until nine days after the onset of glandular swelling.

Exclude cases from school, child care or workplace until nine days after the onset of glandular swelling. Advise parents to keep the child away from other children and susceptible adults for the period of exclusion.

Control of contacts

Susceptible contacts should be offered immunisation with MMR vaccine. Immunoglobulin is not effective in preventing mumps. Contact isolation is not required.

Control of environment

Concurrent disinfection of articles soiled with nose and throat secretions.

Outbreak measures

Susceptible persons should be immunised, especially those at risk of exposure. Those who are not certain of their immunity can be vaccinated if no specific contra-indications to live vaccines exist.

Murray Valley encephalitis virus

Arboviruses are viruses which are spread by the bite of arthropods, particularly mosquitoes. They are divided into alphaviruses and flaviviruses.

Victorian statutory requirement

Murray Valley encephalitis (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion is not required.

Infectious agent

Murray Valley encephalitis virus is a flavivirus. It has the capacity to cause severe human disease, with encephalitis being the most notable clinical feature.

Murray Valley encephalitis virus (MVEV) was first isolated from patients who died from encephalitis in the Murray Valley in Victoria and South Australia in 1951. It was previously included as one of the causative agents in the disease called Australian encephalitis, which also included disease caused by Kunjin virus, another flavivirus. These viruses are now accepted as causing two separate diseases.

Identification

Clinical features

The MVE virus commonly infects humans without producing apparent disease (subclinical infection). It may also cause a comparatively mild disease with features such as fever, headache, nausea and vomiting. In a small percentage of all people infected, mild disease may be a prodrome to disease progression and involvement of the central nervous system. This can result in meningitis or encephalitis of variable severity. Signs of brain dysfunction such as drowsiness, confusion, fitting, weakness or ataxia indicate the onset of encephalitis.

Method of diagnosis

Infection is confirmed by a significant rise in antibody titre to the virus in two blood specimens taken seven to ten days apart. Sera for diagnosis should be sent to the Director of Virology, Victorian Infectious Diseases Reference Laboratory (VIDRL), preceded by telephone contact via the Royal Melbourne Hospital on (03) 9342 7000 advising the on-call Virologist that sera has been sent for urgent testing.

A diagnosis of MVEV encephalitis should be considered in any patient who presents with encephalitis and who has been in the Murray Valley area within the incubation period of the disease, especially in the period between November and March. The disease may also be acquired at any time in northern parts of Australia or Papua New Guinea.

Laboratory evidence requires one of the following:

- isolation of MVEV from clinical material
- detection of MVEV RNA in clinical material
- IgG seroconversion or a significant increase in antibody level or a fourfold rise in titre of MVEV specific IgG proven by neutralisation or another specific test
- MVEV-specific IgM detected in the CSF in the absence of IgM to Kunjin, Japanese encephalitis or dengue viruses
- MVEV-specific IgM detected in serum in the absence of IgM to Kunjin, Japanese encephalitis or dengue viruses. This is only accepted as laboratory evidence for encephalitic illnesses.

Confirmation of the laboratory result by a second arbovirus reference laboratory is required if the case occurs in areas of Australia not known to have established enzootic, endemic or regular epidemic activity.

Clinical evidence

Clinical evidence may be present as non-encephalitic illness, encephalitic illness or asymptomatic disease.

Non-encephalitic illness

Acute febrile illness with headache, myalgia and/or rash

Encephalitic disease

Acute febrile meningoencephalitis characterised by one or more of the following:

- focal neurological disease or clearly impaired level of consciousness
- an abnormal CT, MRI scan or EEG
- presence of pleocytosis in the CSF.

Incubation period

The incubation period is usually 7–28 days.

Public health significance and occurrence

Serological studies show that only one person in about every 800 of those infected with MVE virus develops clinical disease. Of those presenting with encephalitis in Victoria in the 1974 epidemic, approximately one-third died, one-third were left with residual brain damage and one-third recovered completely.

MVE virus is endemic in northern Australia and Papua New Guinea where sporadic cases or small outbreaks of MVE virus encephalitis occur every few years. This is usually at the end of the wet season. Seven outbreaks of MVE virus encephalitis have occurred at irregular intervals in southeastern Australia since 1917. The last of these was in 1974. During these times there was heavy rainfall leading to widespread flooding which promoted large increases in water bird and vector mosquito populations. The MVE virus numbers were amplified in the bird-mosquito-bird cycle and humans became infected when bitten by mosquitoes carrying the virus.

MVE virus encephalitis seems to occur in people who receive large numbers of mosquito bites during a single exposure. There are two theories as to how the MVE virus appears and causes outbreaks of MVE virus encephalitis in southeastern Australia; both may be correct. The first one postulates that the virus is carried from northern parts of Australia by birds migrating south in search of food after heavy rainfall down the southeastern parts of the continent. This occurs in repeated mosquito-bird-mosquito amplification cycles. The other suggests that the virus persists during inter-epidemic periods in cryptic foci along the Murray River and the MVE virus only amplifies and becomes evident when weather conditions are conducive to massive local mosquito and bird multiplication.

Reservoir

The primary hosts in Victoria of MVE virus during years of high virus activity are water birds. *Ardeiformes* (herons), particularly the Rufous night-heron and the *Pelicaniformes* (cormorants/darters) are the most commonly infected.

Mode of transmission

The primary mosquito vector during epidemics is *Culex annulirostris*. Other mosquitoes such as *Culex australicus* and some *Aedes* and *Ochlerotatus* species may be involved in other aspects of MVE virus ecology.

Period of communicability

There is no evidence of person to person transmission.

Susceptibility and resistance

Infection with MVE virus confers lifelong immunity.

Control measures

Preventive measures

Patients can be managed at any hospital, but facilities for providing intensive care and artificial respiration must be available. There is no preventative vaccine available.

Control of case

Investigate the source of infection. Search for unreported or undiagnosed cases of encephalitis from the Murray-Darling drainage basin.

The patient with suspected infection or friend or relative, should be asked to recall if in the month prior to onset of symptoms he or she had:

- been bitten by mosquitoes
- visited regions where arboviruses are endemic
- participated in recreational or other activities involving exposure to bushland or other mosquito habitat such as gardening, bushwalking, camping and picnicking.

Control of contacts

Not applicable.

Control of environment

To reduce or prevent virus transmission, interruption of human-mosquito contact is required by:

- suppression of the vector mosquito population
- avoidance of vector contact at biting times at dusk and dawn
- applying mosquito control measures in local municipalities
- using personal protection measures such as long sleeves, long trousers and mosquito repellents
- avoiding mosquito-prone areas.

Outbreak measures

Following notification of a seroconversion to MVE virus or information of human notification:

- an emergency meeting of the Victorian Arbovirus Task Force (VATF) will be convened by the Department of Human Services
- the presence of MVE virus in the area will be notified to relevant regional offices and local health council personnel
- suitable media releases will be made available
- appropriate VATF members will visit the area to consult and advise local councils, health and tourism authorities
- depending on the actual or potential severity of the epidemic, meetings of relevant personnel will be arranged in the affected area to consider control measures.

Additional sources of information

Victorian Department of Human Services, *Victorian Arbovirus Task Force contingency plan for outbreaks of MVE*.

Mycobacterial infections
(non-tuberculosis)

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
Mycobacterial agents include *M. avium-intracellulare* complex (MAC), *M. kansasii*, *M. scrofulaceum*, *M. fortuitum*, *M. marinum* and *M. chelonae*.

For infections due to *M. ulcerans* see separate chapter.

Identification
Clinical features

MAC and *M. kansasii* are rare causes of lung disease in humans, and mainly affect middle-aged and elderly persons with underlying chronic lung conditions. Disseminated MAC infection frequently occurs in people with advanced HIV infection, but is rare in immunocompetent hosts.

Cervical and submandibular lymphadenitis due to MAC, *M. scrofulaceum* and *M. kansasii* may occur in otherwise healthy young children.

M. fortuitum and *M. chelonae* cause skin and wound infections and abscesses. They are frequently associated with trauma or surgery.

M. marinum causes 'swimming pool granuloma', a nodular lesion that may ulcerate and is usually located on an extremity.

Method of diagnosis

Persons with immunodeficiencies or tissue damage such as skin trauma and pulmonary disease may be at increased risk of atypical mycobacterial infection.

Clinicians who suspect infection with atypical mycobacteria should liaise with a pathology laboratory to ensure that clinical specimens are appropriately collected and transported.

To establish a definite diagnosis of atypical mycobacterial infection, organisms must be cultured from a case with clinically compatible disease. Identification of acid-fast bacilli by direct smear on at least two occasions is highly suggestive of a mycobacterial infection. Histological examination of biopsies of clinical lesions may also assist in the diagnosis. Recent advances in gene probes and nucleic acid amplification procedures such as polymerase chain reaction (PCR) have allowed more rapid diagnosis of mycobacterial infections such as DNA probes for MAC and *M. kansasii*.

Incubation period
The incubation period of atypical mycobacterial infections can rarely be determined, but is probably weeks to several months.

Public health significance and occurrence
Disease due to atypical mycobacterial infection is relatively rare. Cases of *M. kansasii* lung infection have occurred in western Victoria in recent years. Infection with *M. marinum* is associated with contact with swimming pools, aquariums and other bodies of water.

Atypical mycobacteria may colonise and infect persons without causing clinical disease. Skin tests to tuberculin and other mycobacterial derivatives may be positive in such people.

Reservoir
Mycobacteria are ubiquitous in the environment, including many that are non-pathogenic to humans. Mycobacteria have been cultured from various environmental sources including ground waters, dust and soil. The environmental niches of many others remain unknown.

Mode of transmission
The mode of transmission can rarely be determined for individual cases. Atypical mycobacteria are probably transmitted by aerosol from soil, dust or water, by ingestion, or in *M. marinum* and infections by skin inoculation.

Person to person spread of atypical mycobacteria is rare except in people who are immunosuppressed. *M. avium-intracellulare* causes disease in poultry and pigs but animal to human transmission is rare.

Period of communicability
Communicability of human cases is usually not a practical concern except in cases of co-existing HIV infection. Localised foci of disease due to some atypical mycobacteria suggest that an established environmental focus of organisms may remain the source of infections for years.

Susceptibility and resistance
With the exception of *M. marinum* infections, atypical mycobacterial infections (in particular MAC) are more common in patients who are immunocompromised or in those with chronic respiratory disease.

Control measures

Preventive measures

As little information is known about their mode of transmission, prevention of atypical mycobacterial infections is difficult. Environmental contamination of skin lesions may be reduced by some measures, including the wearing of gloves and thorough hand washing when cleaning aquarium equipment (for *M. marinum*). Early medical advice should be sought in the event of skin lesions that do not heal.

Control of case

Cases of atypical mycobacterial infection usually require specialist management. Skin lesions and childhood lymphadenopathy are usually cured by surgery, sometimes in combination with anti-mycobacterial drugs.

Disseminated and pulmonary infections are treated with combinations of anti-mycobacterial drugs. The clinical outcome is strongly influenced by the underlying health of the host.

Control of contacts

No specific measures are needed for contacts of cases.

Control of environment

If infections can be linked with a specific environmental source it may be possible to modify the environment or practices to minimise further transmission.

Outbreak measures

Not applicable.

Mycobacterial infections (tuberculosis)

Victorian statutory requirement

Tuberculosis (Group B disease) must be notified in writing within five days of diagnosis including clinical suspicion.

School exclusion: exclude until receipt of a medical certificate from the treating physician stating that the child is not considered to be infectious.

Contacts are not excluded.

Infectious agent

Mycobacterium tuberculosis (human tuberculosis) and Mycobacterium bovis (cattle tuberculosis) are the infective agents.

Identification

Clinical features

Tuberculosis (TB) is an acute or chronic infection caused by the tubercle bacillus *Mycobacterium tuberculosis*, and rarely by *M. bovis* or *M. africanum*. The initial pulmonary infection usually goes unnoticed with lesions healing, sometimes leaving traces of calcified scar tissue. The infection may however progress to pulmonary tuberculosis, or through blood or lymphatic spread produce miliary, meningeal or other extrapulmonary involvement.

Common symptoms include:

- a chronic cough sometimes accompanied by haemoptysis
- fevers and night sweats
- loss of weight
- feeling generally unwell.

Clinical suspicion of active disease should be high in those who have a newly positive tuberculin reaction, juveniles with positive tuberculin reactions, and those with a history of inadequately treated active tuberculosis. Positive tuberculin reactors with inactive tuberculosis on chest X-ray without a previous diagnosis of active tuberculosis remain at some risk. The risk of developing the disease is highest in children under three years of age, lowest in later childhood, but rises again for adolescents, young adults and the very old.

Method of diagnosis

TB is diagnosed by a consideration of the following:

- clinical presentation
- tuberculin skin test using the Mantoux procedure
- radiographic examination, sometimes including CT scans
- bacteriology, direct staining and culture of sputum or other specimens for the presence of *M. tuberculosis*
- molecular amplification (PCR) and gene probes assist in rapid diagnosis.

Definitive diagnosis of TB rests on isolation of *M. tuberculosis* (or *M. bovis*) from sputum, urine, biopsy material, CSF or other clinical specimens. A negative sputum test does not rule out a diagnosis of TB. Recovery and identification of mycobacteria from specimens has become more rapid with test procedures such as liquid medium systems and DNA probes. Further information on these tests can be obtained from the Mycobacterium Reference Laboratory at Victorian Infectious Diseases Reference Laboratory.

Incubation period

Infection to the primary lesion or significant tuberculin reaction is about four to twelve weeks.

Public health significance and occurrence

Tuberculosis occurs worldwide and had been decreasing steadily over past decades in developed countries. This pattern was reversed with the arrival of HIV and increased mobility of the world's population. Tuberculosis in the USA was on the increase in the early 1990s.

A combination of factors is thought to be responsible for this increase including the high rate of HIV infection, overcrowding, limited health care resources and falling living standards. In the USA, large outbreaks of TB have occurred in institutions, particularly prisons and hospitals. These outbreaks have predominantly affected HIV-infected persons. The increasing trend in the US has now been reversed and the incidence rate is once again declining.

The World Health Organization (WHO) estimated that a third of the world's population is infected and tuberculosis accounts for three million deaths annually. One-fifth of all deaths in adults in developing countries relate to TB. Two-thirds of the world's tuberculosis-infected people reside in Asia and this will have a significant impact on the control of TB in Australia as a result of increased immigration.

Notified cases of TB in Victoria have dropped dramatically from 1000 cases in 1954 to 292 in 2000. However, the rate of decline in the incidence of TB has reached a plateau with an average incidence rate of 6.2/100 000 over the last five years (range 5.1/100 000 in 1998 to 7.0/100 000 in 1999).

The proportion of notified cases that were overseas-born has also increased from 37% in 1970 to 86% in 2000. In Victoria the highest country-specific incidence rates are in the Vietnamese, Indian, Filipino and African born populations. This reflects the pattern of disease in their countries of birth. Of the overseas-born patients, almost 50% present with disease within five years and 30% present within two years of their arrival in Australia.

Reservoir

Humans are the primary reservoir. Diseased cattle rarely act as reservoirs.

Mode of transmission

TB is transmitted mainly by inhalation of infectious droplets produced by persons with pulmonary or laryngeal tuberculosis during coughing, laughing, shouting or sneezing.

Invasion may occur through mucous membranes or damaged skin.

Extrapulmonary tuberculosis, other than laryngeal infection, is generally not communicable. Urine is infectious in cases of renal tuberculosis. Bovine tuberculosis results mainly from ingestion of unpasteurised milk and dairy products. Aerosol transmission has been reported among abattoir workers.

Period of communicability

In theory, the patient is infectious as long as viable bacilli are being discharged from the sputum. In practice, the greatest risk of transmitting infection is in the period prior to diagnosis of an open case. A sputum smear positive case is more infectious than a case only positive on culture. The risk of transmitting the infection is significantly reduced within days to two weeks after commencing appropriate chemotherapy.

Susceptibility and resistance

Everyone is susceptible to infection, however some groups are more susceptible to infection and progression to active disease than others. Special groups at risk are:

- recent immigrants and refugees from countries with a high incidence of tuberculosis including Vietnam, India, China, Africa and the Philippines
- those in close contact with a case of active TB
- Aboriginal people and Torres Strait Islanders in some parts of Australia
- immunosuppressed patients
- those with HIV infection and AIDS
- the elderly
- diabetics
- drug and alcohol-dependent people
- people living in substandard, overcrowded conditions
- institutionalised people including prisoners
- health professionals.

The disease does not always confer protective immunity as reinfection can occur.

Control measures

Preventive measures

BCG vaccination has limited application in developed countries where the incidence of TB is low. It is an effective vaccine in reducing TB meningitis and death in babies and children less than five years in countries of high TB prevalence. It is not recommended for general use in the Australian community but should be considered for specific high risk groups such as infants and young children travelling for extended periods to countries with a high incidence of TB. (Refer to *The Australian immunisation handbook*, National Health and Medical Research Council).

Control of case

With the introduction of potent anti-TB drugs, hospitalisation of tuberculosis patients is no longer mandatory unless social conditions or coexisting medical conditions dictate otherwise.

Patients with pulmonary TB should be isolated either at home or in hospital until they have been on adequate anti-TB therapy for 14 days and sputum smears are negative. Appropriate education and counseling about minimising the risk of transmission of infection should be provided to all patients, particularly those with pulmonary TB. There is no restriction on the movement of patients with non-pulmonary disease.

Written notification of tuberculosis is required within five days of diagnosis. On receipt of a notification, a public health nurse is allocated to the patient to provide support, assist with treatment compliance and to assess the requirements and extent of contact tracing.

Adequate anti-TB chemotherapy for an appropriate period of time will result in almost 100% cure rate. Short treatment regimens have been in use for some years. These involve the use initially of three or four drugs (isoniazid [INH], rifampicin, pyrazinamide and may include ethambutol) for two months, and continuing with isoniazid and rifampicin for a further four months. Where there is evidence of drug resistance to isoniazid or rifampicin or to both, short course anti-TB chemotherapy is inappropriate.

The success of treatment relies heavily on patient compliance and direct supervision should be the aim of any treatment program. Compliance is important to prevent the development of drug resistance.

Multi-drug resistant TB (MDRTB)

Resistance to at least isoniazid and rifampicin (whether or not it is also resistant to other drugs) is classified as multi-drug resistant. MDRTB is rare in Australia. It has remained at less than two percent per year in the past 15 years. There is however a potential risk of MDRTB in Victoria as most of the patients notified each year are overseas born, many from countries with high rates of drug resistant TB.

Control of contacts

Exclusion of contacts is not necessary, unless they have signs and symptoms consistent with pulmonary TB.

Contact tracing and surveillance are the responsibility of the Department of Human Services and are managed by the TB Program. Anyone identified by health care workers as a contact of a case of TB should be referred to the TB Program.

Contact investigation consists of:

- history taking
- tuberculin testing
- radiographic examination.

The extent of investigation is governed by the characteristics of the source case. The scope of investigation is extended when the following factors in the source case are present:

- acid fast bacilli (AFB) in sputum smear
- cavitation on chest X-ray
- laryngeal TB
- cough, particularly if productive of sputum, or
- evidence of tuberculin conversion in any of the contacts.

Note: Tuberculosis testing should never be omitted for child contacts.

Following tuberculin testing contacts can be grouped as:

- Negative reactors
  - Tuberculin conversion takes a few weeks and may not have occurred yet in these contacts.
  - Testing should be repeated in eight to 12 weeks after a break of contact or in some cases initial testing may be delayed for eight weeks.
  - Chest X-rays may be considered on an individual basis.
- Positive reactors
  - Initial positive reactors should be evaluated to exclude active disease. The positive tuberculin test may signify recent tuberculin conversion or an incidental finding.
  - Contacts identified by the TB Program as requiring further assessment are referred to specialist physicians for exclusion of active disease or consideration for treatment of latent infection.
  - When X-ray and physical examination are normal, contacts with positive reaction may be offered isoniazid treatment of latent infection, given once daily at a dosage of 5 mg/kg body weight to a maximum of 300 mg daily. Treatment should be for a minimum period of nine months with appropriate monitoring for liver toxicity.
  - Contacts with positive reactions, who do not undertake treatment of latent infection, should be kept under surveillance and followed up with chest X-rays taken at six months and 12 months.

Control of environment

There are no specific environmental controls as the greatest risk of transmission of infection is prior to diagnosis. However, a patient with pulmonary tuberculosis should be isolated from any new contacts and young children (either in hospital or at home) until at least 14 days after commencing appropriate anti-tuberculosis treatment.

Fresh air, sunlight and covering the mouth and nose when coughing are all appropriate patient education and environmental control measures.

Outbreak measures

It is unusual for an outbreak of TB to occur due to the chronic nature of the disease and the extended incubation period. In the event of two or more cases occurring concurrently in a single setting, contact tracing and investigation would be extended to identify a possible unknown source case.

Special settings

Nosocomial transmission of tuberculosis does occur, particularly in cases where diagnosis is delayed. It is important that a high index of suspicion for tuberculosis is maintained, particularly in patients with respiratory symptoms and belonging to a high risk group for TB such as overseas born from high prevalence countries, immunosuppressed patients and the elderly (both Australian and overseas born).

All suspected and active cases of tuberculosis must be placed in respiratory isolation and appropriate infection control measures implemented, including use of submicron or particulate filter masks for health care workers and surgical masks for patients during transport within the hospital. In the event of a health care worker being exposed to an undiagnosed case of tuberculosis, appropriate contact tracing and screening measures must be implemented. Investigation and management will be as for contacts (above).

Health care facilities are required to have protocols and guidelines for tuberculosis prevention and management in place, including a tuberculin skin test screening policy (refer to *Management, control and prevention of tuberculosis guidelines for health care providers*, Department of Human Services 2002).

International measures

All countries are required to report TB surveillance data to the World Health Organization. This data informs policies and strategies aimed at the global control of TB. Migrants and long term visitors to Australia are screened for evidence of TB prior to being granted a visa.

Additional sources of information

- Victorian Department of Human Services 2002, *Management, control and prevention of tuberculosis. Guidelines for health care providers (2002–2005)*, http://www.health.vic.gov.au/ideas
- World Health Organization, http://www.who.int

Mycobacterium ulcerans

Victorian statutory requirement

Mycobacterium ulcerans infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not required.

Infectious agent

Mycobacterium ulcerans is a member of the mycobacterium family. Tuberculosis, leprosy and many other environmental mycobacteria belong to this family. *Mycobacterium ulcerans* causes skin ulcers, variously known as the Bairnsdale ,Bairnsdale, Buruli, or Daintree ulcer.

Identification

Clinical features

The first sign of *M. ulcerans* is usually a painless, non-tender nodule or papule. It is often thought to be an insect bite. The lesion may occur anywhere on the body but it is most common on exposed areas of the limbs. Some patients complain that the lesion is itchy. In one or two months the nodule may become fluctuant and ulcerate, forming a characteristic ulcer with undermined edges. Ordinarily there is no regional lymphadenopathy, fever or systemic manifestations associated with the disease. If left untreated extensive ulceration can occur.

Occasionally the disease may present as a firm, painless elevated plaque with irregular edges. Or an entire limb or area may be indurated by oedema without an ulcer being present. The oedematous form may be associated with fever.

Method of diagnosis

Swabs from beneath the undermined edges of the lesion or a biopsy should be sent for staining for acid-fast bacilli (AFBs). Two other swabs should be taken; a dry swab for a polymerase chain reaction (PCR test) and another for culture should be placed in transport medium. Bacterial culture or a specific PCR should be performed to confirm the diagnosis. It should be stated on the request form that *M. ulcerans* is suspected.

A positive smear for AFBs makes the diagnosis likely. Culture or PCR is required for confirmation. A negative smear does not exclude the diagnosis.

The PCR test is performed at the Victorian Infectious Diseases Reference Laboratory (VIDRL). This test can give rapid confirmation of the diagnosis within a few days. Culture of the organism usually takes 8–12 weeks.

A biopsy of suspicious lesions which have not ulcerated can be sent for histology. The suspected diagnosis should be mentioned and a request made for AFB staining, specific PCR and bacterial culture. Biopsy specimens usually show extensive necrosis, especially of fat. Granulomatous inflammation is usually present in more chronic lesions. AFBs are frequently seen in large numbers.

Incubation period

This has not been clearly defined but is thought to be quite long, i.e. weeks to a couple of months.

Public health significance and occurrence

Although this is not a common cause of ulcers in Australia, it is important that it be considered in the focal areas in which it occurs as early diagnosis and treatment is advisable to minimise tissue damage.

After tuberculosis and leprosy this is the most common mycobacterial disease. The disease exists or is suspected in 31 countries. The majority of the cases occur in foci in west and central Africa, where large, severe disabling ulcers may result in severe contractures or death from extensive skin loss.

In Australia the disease exists in Far North Queensland around the Mossman area and in parts of coastal Victoria including East Gippsland (where it was first described in Bairnsdale and so named), Cowes on Phillip Island, Mornington Peninsula and most recently Bellarine Peninsula.

Reservoir

The organism appears to be associated with usually swampy or stagnant water. The exact reservoir remains unclear.

Mode of transmission

The exact method of transmission of *M. ulcerans* infection is unclear. Exposure to contaminated water, soil, or vegetation in areas where the disease is known to occur is thought to be required. The bacteria may enter through a breakage in the skin. Exposure to aerosols of contaminated water has been hypothesised to be a method of acquisition. Recently, some insects that live in water have been shown to contain the bacteria and they may play a role in transmission.

Period of communicability

M. ulcerans infection is not (or is rarely) transmitted from one person to another.

Susceptibility and resistance

Everyone is susceptible to infection.

Control measures

Preventive measures

Early recognition and diagnosis is important to minimise the disabling and disfiguring effects of this disease. Referral for treatment by doctors experienced in the management of this condition is recommended. Simple precautionary measures such as wearing appropriate protective clothing when gardening and undertaking recreational activities in identified risk areas may assist in preventing infection. Cuts and abrasions should be cleaned promptly and exposed skin contaminated by suspect soil or water should be washed following outdoor activities.

BCG vaccination is not used for prophylaxis.

Control of case

Isolation is not required. The ulcers do however contain large numbers of organisms and it is possible that person to person infection could occur through breaks in the skin. Thus, it is recommended that ulcers be kept covered and thorough hand washing be performed following dressing changes. Safe disposal of infected material should also occur.

The current mainstay of treatment is surgery with adequate (but not excessive) clearance of the undermined edges of the ulcer. Primary closure may be possible with small lesions but skin grafting of the area may be required for larger areas. Other forms of treatment that may be used, often as an adjunct to surgery, include:

- Heat treatment. Continuous local heating promotes healing but care must be taken to prevent burning. The organism grows at 32˚C and heat up to 40˚C has been used with some effect.
- Antibiotic treatment. A number of antibiotics have been found to be active against the organism in vitro. These include clarithromycin, rifampicin, azithromycin and amikacin. Combinations of these have been used, often to mop up residual organisms after surgery in an attempt to prevent recurrence.

Occasionally small lesions have been reported to heal spontaneously.

Control of contacts

Not applicable.

Control of environment

Not applicable.

Outbreak measures

Clusters of cases are investigated looking for a common source where an intervention may be feasible and advisable, including health or public alerts.

Additional sources of information

- World Health Organization, www.who.int/topics

Pediculosis or head lice

Victorian statutory requirement

Notification is not required.

School exclusion: readmit the day after appropriate treatment has been commenced.

Infectious agent

Pediculus humanus var. *capitus* is the infective agent.

Identification

Clinical features

Pediculosis is commonly said to be associated with an itchy scalp however this is an unreliable sign. Itching is only experienced in 14–50% of people with head lice. Head lice can be present for weeks or even months without causing an itch. Secondary scalp infections resulting from scratching can occur but are rare.

Method of diagnosis

Early detection makes treatment and control of head lice easier. Traditional scalp inspection is a poor method of detecting lice. It can result in 30% false positive and 10% false negative findings.

The technique known as 'conditioner and combing' is the most effective method for detection. This involves combing white hair conditioner through dry, brushed hair. The next step is to divide the hair into smaller sections and to comb each section using a head lice comb. After each combing the comb is wiped on to a tissue. This allows lice and eggs to be easily seen. The aim is to cylindrically coat each hair in conditioner and continue to comb hair until the majority of conditioner is removed.

Incubation period

The life cycle consists of three stages: egg, nymph and adult. The eggs are known as nits and hatch in six to seven days. There are three nymphal forms that each last one to eight days.

The female lays the first egg one or two days after mating and can lay approximately three to eight eggs per day for the next 16 days. After a life span of 32–35 days the louse dies.

Public health significance and occurrence

Head lice have been associated with humans for 10 000 years. Head lice occur worldwide. Anyone can get lice and given the opportunity head lice will move from head to head without discrimination. They are frequently associated with children.

Information on the prevalence of head lice varies around the world. In 2002 the prevalence of head lice among primary school children in Victoria was found to be 13%. Females were more than twice as likely to have head lice as males.

The prevalence of head lice in primary school aged children in other parts of Australia is reported to be up to 60%.

Head lice are not vectors of infectious disease. Louse-borne relapsing fever, trench fever and typhus, none of which occur in Australia, are all associated with the body louse *Pediculus humanus* var. *corporis*.

Reservoir

Humans are the only reservoir. The lice of other animals are not transmissible to humans.

Mode of transmission

Pediculosis is transmitted through direct head to head contact with a person with head lice. Nymphal and adult lice survive, dependent on the humidity of the environment, and according to Queensland research usually die within 24 hours of being stranded away from the head. There is no significant risk of transmission from the environment.

Period of communicability

Communicability continues as long as lice or their nymphs remain alive.

Susceptibility and resistance

Everyone is susceptible to infection.

Control measures

Preventive measures

Regular checking using the method known as 'conditioner and combing' allows early detection of head lice and will limit the establishment of large outbreaks.

Control of case

Treatment should concentrate on the head. There is no evidence that the environment is a significant cause of reinfection.

The conditioner and combing method can be repeated every second day until no lice are found for ten days.

If using an insecticidal product it is important to use a 'registered' or 'listed' product which should have two applications seven days apart. Applying with the least amount of water possible and the removal of as many eggs as possible will optimise the treatment.

Increasing resistance to the products has been reported and each head lice product should be tested after 20 minutes to ensure it has killed the lice.

Control of contacts

Contact tracing is recommended.

All household members or people who have had head to head contact with the case should be examined for head lice. Preferably this should be done using the conditioner and combing detection method and repeated every two days for ten days.

Special settings

Hairdressing salons

Head lice rarely fall from the head. Data from James Cook University show head lice on combs and brushes are easily killed by immersion in hot water at 60°C for one minute. There is then no subsequent risk of transmission from the comb or brush to the next client.

Outbreak measures

Schools

While head lice are often associated with schools they are not necessarily spread in schools. Schools experiencing difficulty should encourage families to check for lice using the combing and conditioner method on a weekly basis, or more often during an outbreak. Even on receipt of a single case report of head lice, parents or guardians of all children from the class should be asked to screen their child at home utilising the conditioner and comb method. Parents or guardians should then report any active head lice following the synchronised home screening.

If there is a cluster of cases in a class, for example 10% of pupils, a further school wide management program could be considered (see resources for schools, www.health.vic.gov.au/headlice)

Additional sources of information

- De Maeseneer, J, Blokland, I, Willems, S et al. 2000, 'Wet combing versus traditional scalp inspection to detect head lice in school children: observational study', *BMJ*, vol. 321, no. 7270, pp. 1187–8.
- Spear, R, Thomas, G, Cahill, C 2002, 'Head lice are not found on floors in primary school classrooms', *Aust N Z J Public Health*, vol. 26, no. 3, pp. 208–11.
- Victorian Department of Human Services, http://www.health.vic.gov.au/headlice/

Pertussis
(whooping cough)

Victorian statutory requirement
Pertussis infection (Group B disease) requires written notification within five days of diagnosis.

School exclusion for cases and contacts is:

- cases should be excluded for five days after commencing antibiotic treatment
- unimmunised sibling contacts under seven years of age and unimmunised close child care contacts must be excluded from school and children's services centres for 14 days from the last exposure to infection, or until they have taken five days of a ten day course of antibiotics.

Infectious agent
Bordetella pertussis.

Identification
Clinical features

The catarrhal state may be indistinguishable from a viral upper respiratory tract infection. The infection damages respiratory epithelium, producing respiratory obstruction and paroxysmal coughing. There is often a characteristic whoop. This is a crowing sound during inspiration preceding a bout of coughing.

There is little fever. Apnoea, seizures and encephalopathy may occur in very severe cases. Infants aged less than six months and adults often do not have the characteristic whoop. Paroxysms frequently end with the expulsion of clear, tenacious mucus. This is often followed by vomiting.

Pneumonia is the most common cause of death. Fatal encephalopathy, which is probably hypoxic, and severe weakness from repeated vomiting, occasionally occur.

Method of diagnosis

Pertussis can be diagnosed on a clinical basis if the patient has an acute illness lasting more than 14 days without another apparent cause, a classical paroxysmal cough with whooping and post-tussive vomiting. However bouts of coughing may occur without whoops or vomiting and the disease may only be suspected if the patient is a contact of a known case. Apnoea may be the only manifestation in infants. Laboratory confirmation can be problematic but should be sought where possible. A nasopharyngeal aspirate or swab is the best specimen to obtain to culture the bacterium. The likelihood of such cultures being positive is reduced 21 days after the cough onset or if effective antimicrobial therapy has commenced against *B. pertussis*. Serology using *B. pertussis* specific IgA may be falsely negative but a positive result is highly reliable in the presence of appropriate symptoms.

Incubation period
The incubation period is usually between six and 20 days. It is most commonly about 14 days.

Public health significance and occurrence
It is a distressing and often serious illness particularly in children under one year of age. The mortality rate is 0.5% in infants under six months. High immunisation levels reduce the number of cases and good nutrition and medical care reduce case fatality. Many vaccinated adults may have mild infection and act as a source of infection for younger children. Australia experiences an epidemic of whooping cough about every three or four years.

As it is not possible to completely control pertussis with the current vaccine, the highest priority should be given to protecting infants under 12 months of age.

The World Health Organization (WHO) estimates there were 40 million cases of pertussis in 1994 and 360 000 deaths. WHO believes only one to two per cent of cases are reported. In industrialised countries four children out of every 10 000 infected die from pertussis and its complications. In Australia the first pertussis vaccine was manufactured in the 1920s. There is a clear seasonal pattern with 65% of notifications occurring over the spring and summer months.

Reservoir
Humans are the only known natural reservoir of B. pertussis.

Mode of transmission
B. pertussis is highly infectious. It may be spread from person to person by close contact, usually by respiratory aerosols, infecting 70–100% of household contacts.

Period of communicability
It is highly communicable in the early catarrhal stage before the onset of paroxysmal cough. Thereafter communicability decreases and becomes negligible in about three weeks. When treated with a macrolide antibiotic the period of infectivity usually lasts five days or less after commencement of therapy.

Susceptibility and resistance

Maternal antibodies do not protect newborns against infection. Severity is greatest in young infants while milder and atypical cases occur in all age groups. Incomplete immunisation, waning immunity and the fact that vaccine efficacy is 70–80%, results in cases occurring in older children and adults. Lifelong immunity is not guaranteed, even after clinical disease.

Control measures

Preventive measures

Educate the public to the dangers of whooping cough and the advantages of initiating immunisation at two months of age and adhering to the immunisation schedule (DTPa at two, four and six months and four and fifteen years of age). Delay immunisation only for significant intercurrent infection or an evolving neurological disorder. Minor respiratory infections are not a contra-indication for immunisation.

Control of case

Antibiotics will have little effect on the clinical course of disease but can reduce the risk of transmission if commenced within 21 days of cough onset. Treatment generally consists of erythromycin or clarithromycin. If it is not tolerated alternative macrolides with fewer side effects may be considered. A patient who has been coughing for more than 21 days is no longer infectious and antibiotic treatment and school exclusion are not needed. Antibiotic treatment is required if there is complicating pneumonia. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Control of contacts

Erythromycin should not be given if more than 14 days have elapsed since the first contact with the infectious case (doses and duration as for cases). In special circumstances, such as a high risk exposure for an infant contact, antibiotics may be given within 21 days of first contact with an infectious case. Antibiotics rarely prevent secondary transmission and should be limited to household or child care contacts at high risk of severe complications that have had direct contact with an infectious case:

- infants <12 months of age regardless of vaccination status
- any child aged between 12 and 24 months who has received less than three doses of pertussis vaccine
- any women in the last month of pregnancy
- any child or adult who attends or works at a child care facility.

Control of environment

Not applicable.

Outbreak measures

See Control of contacts, above.

Clusters of infection are managed on a case-by-case basis. Contact the Department of Human Services for further advice.

Additional sources of information

- Communicable Diseases Network Australia 1997, *Guidelines for the control of pertussis in Australia, Communicable Diseases Intelligence Technical Report Series*, http://www.health.gov.au
- PHLS Communicable Disease Surveillance Centre 2002, 'UK guidelines for use of erythromycin chemoprophylaxis in persons exposed to pertussis', *Journal of Public Health Medicine*, vol. 24, pp. 200–206.

Pinworm infection
(threadworm)

Victorian statutory requirement
Notification and school exclusion are not required.

Infectious agent
Enterobius vermicularis is an intestinal nematode.

Identification
Clinical features
In the majority of children and adults infection is asymptomatic. Migration of the female worm from the rectum then anus to lay eggs on the perianal skin during the night can lead to perianal pruritus or disturbed sleep or irritability. Sometimes secondary infection of the scratched skin occurs. In children the pinworm can cause vulvovaginitis during its migration from the anus.

Pinworms or their eggs have occasionally been detected at other sites such as the liver and lung. Rarer clinical manifestations include salpingitis, pelvic pain and the formation of granulomas in the peritoneal cavity.

Method of diagnosis
The diagnosis should be suspected in children with a perianal itch and this is confirmed by detection of their characteristic eggs. Applying clear sticky tape ({with sticky side outward) to the perianal skin and examining it for eggs is the best way to make the diagnosis. This is best done in the morning prior to bathing, as the worms migrate during resting periods. Microscopy on faeces can be conducted although finding eggs is exceptional.

Incubation period
The lifecycle requires two to six weeks to complete. The eggs are fully embryonated and are infective within a few hours of being deposited. Male and female pinworms vary in size ranging between 2–13mm in length, up to 0.5mm wide and are yellowish white in colour. A long, thin and sharply pointed tail distinguishes the female worm.

Public health significance and occurrence
The pinworm is the most common helminth parasite of temperate regions. These infections are found worldwide and affect all socio-economic groups.

Less attention is paid to the pinworm in tropical regions of the world presumably because of the prevalence of more important parasites. Pinworm infections predominantly affect paediatric populations where the prevalence is reported to between 10–50% in some groups.

Reservoir
Humans are the only reservoir. Pinworms of other animals are not transmissible to humans.

Mode of transmission
Pinworms are transmitted by direct transfer of infected eggs by hand from anus to mouth of the same or another person. It can also be transmitted indirectly through bedding, clothing, food or other articles. Spread is facilitated by conditions of overcrowding.

Period of communicability
Communicability continues as long as the eggs are being discharged on the perianal area. The eggs can survive for several days in the right conditions. Reinfection from contaminated hands is common.

Susceptibility and resistance
Infection does not confer immunity.

Control measures
Preventive measures
- Effective hand washing, particularly before eating or preparing food.
- Keep nails short, discourage scratching bare anal area and nail biting.
- Daily bathing or showering.
- Change to clean underwear, nightclothes and bed sheets frequently preferably after bathing.

Control of case
There are a number of drugs available for treatment including pyrantel pamoate, mebendazole or albendazole. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Care should be taken to change linen and underwear of infected person daily for several days after treatment with care to avoid dispersing the eggs into the air.

Control of contacts
Not applicable.

Special settings
Public health education on the importance of hand washing may assist.

Outbreak measures
Not applicable.

Additional sources of information

- Markell, E, John, D, Krotoski, W 1999, *Markell and Voge's medical parasitology*, 8th edn, ed Saunders.

Plague

Victorian statutory requirement

Plague (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Plague is subject to Australian quarantine.

Infectious agent

Yersinia pestis is the plague bacillus.

Identification

Clinical features

Plague is an acute, severe bacterial infection usually transmitted through a flea bite and most commonly presents as bubonic, pneumonic or septicaemic forms.

Initial symptoms are often non-specific and may include fever, chills, muscle aches, nausea and lethargy.

Bubonic plague is the most common form. It is characterised by swelling and inflammation of the local lymph nodes (buboes) draining the site of the flea bite or elsewhere. The nodes are tender, firm and fixed, and may suppurate in the second week.

Pneumonic plague may be primary due to respiratory transmission from an external source, or secondary as a complication of bubonic plague. Onset of primary plague pneumonia is usually abrupt with high fever, tachycardia and headache. Cough develops within 24 hours. Sputum is mucoid at first and then becomes bright red and foamy. Chest X-rays show a rapidly progressing pneumonia.

All forms of plague infection may progress to septicaemic plague with bloodstream spread around the body, including to the meninges. This includes some with no preceding localising signs or buboes. Sepsis may lead to disseminated intravascular coagulation (DIC).

Method of diagnosis

Visualisation of characteristic 'safety-pin' ovoid gram-negative organisms in material aspirated from buboes, sputum or CSF is highly suggestive of plague infection.

Fluorescent antibody (FA) testing or antigen capture ELISA is more specific and particularly useful in sporadic cases.

Seroconversion using the passive haemagglutination (PHA) test is also highly suggestive of recent infection.

The diagnosis is confirmed by culture and identification of the organism from bubo aspirates, the blood, CSF or sputum.

Incubation period

The incubation period is from one to seven days. For primary plague pneumonia it is one to four days.

Public health significance and occurrence

Y. pestis is not endemic in Australia but it is widely distributed around the world.

The World Health Organization (WHO) reports 1000 to 3000 cases of plague every year. Plague is endemic in some parts of South East Asia including parts of Indonesia, Burma and Vietnam. Wild rodent plague exists in areas of the USA, South America, Africa, Central and South East Asia.

Untreated bubonic plague has a case fatality rate of about 50–60%. Case fatality rates are significantly higher for pneumonic and septicaemic plague.

Given that techniques for mass production and aerosol dissemination are well described the threat of a bio-terrorist attack using plague is a potential public health concern.

Reservoir

No enzootic (animal) reservoir exists in Australia. In affected countries wild rats and other rodents are the natural reservoir. Other animal reservoirs include ground squirrels (especially in North America), rabbits, hares and domestic cats.

Plague bacteria are killed within a few hours of exposure to sunlight although they may persist for several weeks in water and on moist grains and pulses.

Mode of transmission

Plague is most commonly transmitted from rodent to human by the bite of an infected flea, especially the oriental rat flea *Xenopsylla cheopis*.

Respiratory droplets from people or domestic pets with plague pharyngitis or pneumonia may also transmit plague. Unprotected handling of plague infected animal tissues or laboratory specimens are also possible aerosol routes for plague transmission.

If plague bacteria were to be used as a bioterrorist agent, it would most likely be spread in the form of an aerosol or an aerosolised powder. This would result primarily in pneumonic plague. A deliberate release of infected fleas is also possible.

Period of communicability

Infected fleas may remain infectious for months.

Bubonic plague is not usually transmitted from person to person unless there is direct contact with pus from suppurating buboes.

Pneumonic plague may be highly communicable under appropriate climatic conditions.

Patients are usually no longer infectious after receiving 48–72 hours of appropriate antibiotic treatment.

Susceptibility and resistance

Everyone is susceptible to infection. The disease does not always confer protective immunity.

Control measures

Preventive measures

A vaccine is available against plague that provides some short term protection. It may be recommended for laboratory workers handling plague specimens and visitors to epidemic areas but should not be relied upon as the sole prevention measure.

The vaccine is not protective against primary pneumonic plague.

Control of case

Hospitalise the case in a single room.

Treatment usually consists of gentamicin or doxycycline. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Use an appropriate insecticide to rid the patient (including clothing and baggage) of fleas.

For cases with bubonic plague, if there are no respiratory symptoms contact precautions are indicated until the completion of at least three days of appropriate antibiotic therapy with clinical improvement.

For patients with pneumonic plague, isolate the case to prevent droplet spread and use respiratory precautions until the completion of at least three days of appropriate antibiotic therapy with clinical improvement.

Disinfect all sputum and purulent discharges and soiled articles concurrently.

Use respiratory and contact precautions during the handling, and autopsy, of bodies of patients suspected, or confirmed of dying from plague.

Control of contacts

Contacts of the case should be identified, examined for and, if appropriate, disinfested of fleas. Contacts are placed under surveillance to detect symptoms of early infection for six days from the last exposure.

Close contacts of confirmed or suspected pneumonic plague cases should also be given chemoprophylaxis (doxycycline or ciprofloxacin) supervised by an experienced physician.

Control of environment

As currently there is no enzootic plague in Australia, the greatest risk of infection is associated with overseas travel.

Any suspected local sources of infection should be investigated and managed as a public health emergency (see Outbreak measures, below).

Special settings

The control measures described above apply to all settings.

Outbreak measures

A single case of plague constitutes an outbreak and should be considered as a public health emergency.

If one or more cases are found with no history of travel to an endemic plague area, a deliberate release of plague bacteria must be considered.

If a focus of infection is identified, outbreak control measures should include:

- active case finding
- alerting medical practitioners
- alerting specialist treatment centres
- release of appropriate public information
- instigation of intensive flea control around the focus in expanding circles of control
- destruction of rodents within affected areas
- control of contacts (as described above) including field workers involved in environmental control measures.

International measures

Governments are required to notify WHO and adjacent countries of the first cases of plague in any area previously free of the disease within 24 hours of diagnosis, in accordance with International Health Regulations.

Measures applicable to ships, aircraft, land transport and international travellers arriving from plague areas are specified in the International Health Regulations 1969, third annotated edition 1983, WHO, Geneva.

Prior to departure from an area where there is an epidemic of pulmonary plague, those suspected of significant exposure should be placed under surveillance to detect symptoms of early infection for six days from the last exposure.

On arrival of a suspected infected ship or aircraft, travellers should be disinfested of fleas and kept under symptom surveillance for six days from the date of arrival.

Additional sources of information

- Australian Government Department of Health and Ageing fact sheet, http://www.health.gov.au
- Centers for Disease Control and Prevention, Atlanta USA, *Public health emergency preparedness and response*, http://www.bt.cdc.gov

Poliomyelitis

Victorian statutory requirement

Poliomyelitis (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion: applicable for at least 14 days from onset. Re-admit after receiving medical certificate of recovery.

Infectious agent

Poliovirus is an enterovirus; types 1, 2 and 3 cause disease.

Identification

Clinical features

The majority of polio infections are either inapparent or present as a non-specific febrile illness. Flaccid paralysis occurs in less than 1% of poliovirus infections.

Symptoms of minor illness include fever, malaise, headache, nausea and vomiting. If the disease progresses to major illness, severe muscle pain and stiffness of the neck and back with flaccid paralysis may occur.

The most characteristic feature of polio paralysis is its asymmetric distribution, which affects some muscle groups while sparing others. Fever and muscle pain are generally present at onset with the maximum extent of paralysis usually reached within three to four days. Progression of paralysis almost invariably halts when the patient becomes afebrile. The site of paralysis depends upon the location of nerve cell destruction in the spinal cord or brain stem. Proximal muscles of the extremities tend to be more involved than distal. The legs are more often affected than the arms. Paralysis of the respiratory and swallowing muscles is life threatening.

After 60 days the degree of existing paralysis is likely to be permanent. Sensory loss is very rare and its occurrence should strongly suggest some other diagnosis such as Guillain-Barré syndrome.

Post-polio syndrome is an infrequent recurrence of muscle weakness that may occur many years after initial infection. It is thought to be due to progressive dysfunction and loss of motor neurons that compensated for the neurons lost during the original infection, not to persistent or reactivated poliovirus infection.

Vaccine-associated paralytic poliomyelitis (VAPP) is a very rare complication in recipients of oral polio vaccine or their contacts, with approximately one case per 2.4 million doses of vaccine. The risk is greater for the first dose than subsequent doses and is slightly greater for adults than children.

Method of diagnosis

A clinical history including vaccination status of case and household contacts and any recent travel is important.

Diagnosis is made by isolation of virus from cerebrospinal fluid (CSF), faecal specimens or oropharyngeal secretions. Two separate faecal specimens taken at least 24 hours apart and within 14 days of onset of symptoms give the best chance of diagnosis. CSF usually reveals a mild elevation in protein and a lymphocytosis.

The Department requires that all suspected cases of polio have appropriate faecal specimens sent for analysis by the National Poliovirus Reference Laboratory (NPRL), managed by the Victorian Infectious Diseases Reference Laboratory. The NPRL can also differentiate between 'wild-type' and vaccine-associated strains.

The Department coordinates with clinicians and the NPRL to ensure that appropriate infection control procedures are followed in the collection, transfer and analysis of all clinical specimens from patients with suspected polio.

Incubation period

The range is between three to 35 days with seven to 14 days for paralytic cases.

Public health significance and occurrence

Prior to vaccination programs polio occurred worldwide. Since the Global Polio Eradication Initiative was launched in 1988, three WHO regions have been certified polio-free: the Americas in 1994, the Western Pacific (of which Australia is a member) in 2000, and Europe in 2002. Polio cases have dropped from an estimated 350 000 in 125 countries in 1988 to just 480 reported cases in only ten polio-endemic countries in 2001.

By 2003, six countries were still reporting new polio cases: India, Niger, Pakistan, Afghanistan, Egypt, and Nigeria.

In endemic areas, cases of polio occur both sporadically and in epidemics. In temperate climates an increase in cases occurs during the late summer and autumn, in tropical countries an increase is less pronounced but can occur as a seasonal peak in the rainy season.

In countries where polio has been eradicated, importation from non-vaccinated individuals remains a threat.

Polio remains a predominantly childhood illness with 80% to 90% of cases occurring in children less than five years old.

Reservoir

Humans.

Mode of transmission

Wild poliovirus is spread through faeces and saliva. It is primarily transmitted through faecal-oral spread and is an important consideration where sanitation is poor.

'Live' oral polio vaccine (OPV) virus can be shed in the faeces for six weeks and may lead to infection in unvaccinated contacts. Unvaccinated household contacts of a case should be vaccinated at the same time. Stressing the importance of hand washing for parents following nappy changing and disposal is important.

Period of communicability

The risk of transmission of infection is greatest for the seven to ten days prior to and following the onset of symptoms.

The virus persists in the pharynx for approximately one week and in the faeces for up to six weeks, or longer in the immunosuppressed.

Transmission of the virus is possible for as long as the virus is excreted.

Susceptibility and resistance

All non-immune people are susceptible to infection.

After infection from both clinically recognisable and inapparent infections, type specific lifelong immunity occurs. Reinfection is rare but can occur if infected with poliovirus of a different type.

Vaccine efficacy of OPV and Inactivated Polio Vaccine (IPV) after a primary course is 95% and thought to be life long. Both vaccines give protection against all three types of poliovirus.

Infants born of immune mothers have transient passive immunity.

Control measures

Preventive measures

Universal vaccination in early childhood is the most effective means of preventing and eradicating poliomyelitis. Catch-up immunisation is also recommended for unimmunised or partially immunised adults at risk of exposure such as those travelling overseas and health care workers in possible contact with polio cases.

Immunisation can be given as an intramuscular IPV, or as a live OPV.

Under the National Immunisation Program, polio immunisation consists of a primary course of OPV given as two drops by mouth at 2, 4 and 6 months of age with a booster at four years of age. IPV is given for individuals with immunosuppression from disease or chemotherapy and for their siblings and household contacts.

Both IPV and OPV give mucosal and humoral protection, however IPV produces considerably lower levels of intestinal immunity than OPV.

Due to the successful elimination of polio in some regions and the concern with OPV of Vaccine Associated Paralytic Poliomyelitis (VAPP), many industrialised countries have now changed to IPV alone for routine immunisations. IPV is the vaccine recommended on the ASVS subject to the availability of further combination vaccines. The Australian Government is currently reviewing this funding decision.

OPV is still recommended in developing countries because of the higher risk of exposure to wild poliovirus, the low cost of the vaccine, the ease of its administration and its excellent capacity to provide population-level immunity.

Control of case

There is no specific treatment against poliovirus. Cases require expert supervision and may need ventilation support. Early physiotherapy may increase the level of function and reduce the risk of physical deformities as a result of paralytic polio.

Enteric precautions should be initiated in hospital settings. These are often of little benefit in household settings as susceptible contacts are likely to have been exposed prior to diagnosis.

In communities with appropriate modern sewerage systems, faeces and urine from infected patients can be disposed of directly into sewers without preliminary disinfection. Terminal disinfection is required for all other potentially contaminated items.

Control of contacts

Vaccination of families and other close contacts is recommended but may not contribute to immediate control due to susceptible contacts often being infected by the time the first case is recognised.

Active case finding, especially among children, ensures early detection of related cases and facilitates control.

Control of environment

In communities with modern sewerage systems, faeces and urine can be disposed of directly into the system without preliminary disinfection.

Cases and carers should be advised about the importance of strict hand washing, covering the mouth when coughing, sneezing into disposable tissues, and the appropriate cleaning or disposal of contaminated objects.

Outbreak measures

In countries such as Australia where polio has been eradicated a single case of polio is considered a public health emergency and the Department of Human Services must be notified immediately. The Department investigates to:

- determine whether the patient's disease represents an indigenous, imported or VAPP case
- if believed to be a VAPP case, obtain details of vaccine history, batch number, virus type, severity and persistence of residual paralysis 60 days after onset

supervise all appropriate case and contact control measures, as outlined above.

Psittacosis (ornithosis)

Victorian statutory requirement

Psittacosis (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

Chlamydia psittaci is an obligate intracellular bacterium.

Identification

Clinical features

The onset of psittacosis is usually abrupt with fever, prominent headache, photophobia, myalgia, and upper or lower respiratory tract symptoms. Dry cough is a common feature. Pulse-temperature dissociation, splenomegaly and rash may occur. In association with pneumonia these are said to be suggestive of the diagnosis. Chest X-rays may show patchy or focal consolidation.

The illness usually lasts for seven to ten days and is mild or moderate. It may be severe in pregnant or older, untreated patients. Asymptomatic infection or mild flu-like illness may also occur.

Complications include encephalitis, endocarditis, myocarditis and thrombophlebitis. Relapses may occur, especially when there has been inadequate treatment.

Method of diagnosis

Humans

Infection is generally diagnosed by seroconversion on paired acute and convalescent phase sera, although a single high acute phase titre in the setting of clinically-compatible illness is significant. Low positive titres are common in high risk groups. False positives may occur in *C. pneumoniae*, *C trachomatis* and occasionally in *Legionella* infections.

Antibiotic treatment may delay or attenuate antibody formation so convalescent sera should be taken at least two weeks after the acute specimen.

Culture of the organism is generally not performed because of danger to laboratory workers.

Birds

Birds suspected of being infected should be referred to a veterinarian for diagnosis and treatment as required. The Avian Medicine Section at Primary Industries Research Victoria, Attwood (03) 9217 4200 has further details of specimen collection and transport requirements.

Incubation period

The incubation period is four days to four weeks, commonly ten days.

Public health significance and occurrence

Most cases are sporadic but outbreaks of infection may occur rarely within individual households or through contact with affected pet shops or poultry processing plants.

Reservoir

Birds of all types act as a reservoir. This is especially common for psittacine birds (parrots, lorikeets, cockatiels, budgerigars) but also pigeons, turkeys, ducks and occasionally chickens. Healthy birds may be carriers. Cats, dogs, goats or sheep may be infected but this is rare.

Mode of transmission

Infection is generally acquired by inhaling dust from dried faeces or fresh or dried ocular and nasal secretions from infected birds. Direct contact with birds is not required for infection. Rare person to person transmission has occurred.

Period of communicability

Infected birds may shed the agent intermittently for a prolonged period. Shedding may be precipitated by stress on the birds such as cold, crowding or shipping. Dried secretions may remain infectious for many months.

Susceptibility and resistance

Chlamydia psittaci is highly infectious. At risk groups include bird owners, pet shop employees, veterinarians, poultry-processing workers, zoo workers and taxidermists.

Older adults and pregnant women may have a more severe illness. Immunity following infection may be incomplete and reinfection occurs occasionally.

Control measures

Preventive measures

Educate the public about the danger of household or occupational exposure to infected pet birds.

Wearing gloves and dust masks is recommended when cleaning areas with which birds have frequent contact such as cages and bird feeders.

Prevent or eliminate infections of birds by quarantine and antibiotic treatment.

Appropriate surveillance of commercial flocks, pet shops and aviaries should be instituted.

Destroy or treat infected birds and disinfect premises.

Control of case

Isolation is not necessary, but instruct the patient to cough into disposable tissues. Treatment with tetracyclines should be continued for 10–14 days after fever settles. If tetracyclines are contraindicated erythromycin can be used. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Control of contacts

A diagnosis of psittacosis should be considered in symptomatic contacts.

Control of environment

If birds were recently purchased the origin of suspected birds should be traced. This is the responsibility of the Department of Human Services in liaison with the Department of Primary Industries.

Prophylactic use of tetracyclines can suppress, but not eliminate, infection in flocks and may complicate investigations.

For disinfection of floors and cages use a 1:100 dilution of household bleach in water or 70% isopropyl alcohol.

Outbreak measures

All cases should be thoroughly investigated in order to identify more extensive outbreaks.

Outbreaks should be reported to the Department of Human Services, Victoria.

Additional sources of information

- Centers for Disease Control and Prevention 2000, 'Compendium of measures to control Chlamydia psittaci infection among humans and pet birds', *Morbidity and Mortality Weekly Report*, vol. 49, RR08, pp. 3–17, http://www.cdc.gov/mmwr

Psittacosis information sheet

What is psittacosis?

Psittacosis is bacterial disease of both wild and domestic birds that can affect people. In birds it is also known as avian chlamydiosis (AC).

What are the symptoms in humans?

Psittacosis in humans may cause a flu-like illness or pneumonia. Symptoms may include fever, headache, aching muscles and chills, while cough is characteristically dry or may be absent. If pneumonia occurs, symptoms such as shortness of breath or chest pain may occur.

Where is psittacosis found?

Birds, especially parrots, can carry the disease. Birds do not have to be sick to spread the disease. Rarely ill cats, dogs, goats or sheep can spread infection.

How is psittacosis spread?

The disease is spread by breathing in the bacteria which is present in the infected bird's droppings, nose or eye secretions. Dried secretions can remain infectious for many months. The risk of getting the disease is greater when the birds are under stress, for example just after being bought. You may unknowingly come into contact with infected birds while feeding wild birds, cleaning feeding stations or cleaning contaminated aviaries. The spread of psittacosis from person to person is rare.

What about my pet bird?

Sick birds may have eye or nasal discharge or ruffled feathers, and may feed poorly. If your bird is ill seek advice from your vet. Stop wild birds getting close to your pet bird's cage as they can spread disease.

I think I may be infected - what should I do?

See your local doctor and tell them about your contact with birds. This disease can be readily treated with antibiotics.

How can I avoid getting psittacosis?

Avoid contact with wild birds and do not feed wild birds.

Try to avoid stressing birds by crowding or cold conditions and do not buy birds which appear ill.

Wear gloves and a dust mask when cleaning cages and wet down the area prior to cleaning to prevent dust formation. Don't use an ordinary vacuum cleaner as it can throw infectious dust into the air.

Clean cages, food and water bowls daily and use litter which creates dust such as newspaper.

Use a 1:100 diluted solution of household bleach to disinfect any ill bird's cage, bowl etc. Throw away material which cannot be disinfected and rinse all disinfected items before replacing them.

Do not allow birds to get close to your face and wash hands thoroughly after contact with birds.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Q fever

Victorian statutory requirement

Q fever infection (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

The rickettsia-like bacterium *Coxiella burnetii* is the causative agent.

Identification

Clinical features

The onset of Q fever infection is usually acute and characterised by fever, chills, sweats, severe headache (especially behind the eyes), weakness, anorexia, myalgia and cough. Transient mild rashes are an occasional feature. Orchitis occurs rarely. Abnormal liver function tests are common.

Chronic complications include granulomatous hepatitis and endocarditis. The latter is the most serious concern as it usually involves the aortic valve and occurs months to years after the acute illness. A relapsing fatigue syndrome may occur in 20–40% of cases.

Method of diagnosis

Acute and some chronic manifestations of Q fever can be diagnosed by serology.

Acute Q fever can be diagnosed by a fourfold rise in specific complement fixation (CF}) antibodies or by direct immunofluorescence (IF) antibody testing between acute and convalescent sera collected at least 14 days apart.

The diagnosis is supported by the detection of phase II IgM by ELISA testing but this may not appear until 10 days after the onset of symptoms. Q fever IgM may persist for many months after infection; hence its presence does not necessarily confirm the diagnosis.

If Q fever antibodies are present within three to four days of the onset of symptoms it is more likely to indicate past exposure rather than recent infection.

Chronic Q fever is suggested by a high CF antibody titre(320) to phase I and II antigens and low or absent IgM antibody.

IgA class antibody to phase I antigen is highly suggestive of Q fever endocarditis.

Incubation period

The incubation period is typically 19–21 days although the range is from two weeks to two months.

Public health significance and occurrence

It is an acute febrile rickettsial disease of low mortality but significant morbidity. It is most commonly found in abattoir workers who have recently handled contaminated stock such as feral goats or sheep from interstate endemic areas. It is an occupational hazard for tannery and knackery workers, shearers, meat inspectors, dairy workers, animal-farm workers, animal transporters, wool sorters and veterinary personnel. It also occurs in others handling fomites such as those laundering contaminated clothing.

Outbreaks are usually of short duration.

Reservoir

There is no known endemic reservoir in Victoria. The organism is commonly introduced in stock from interstate from animals including goats, cattle, sheep, other farm and domestic animals and some wild animals (including kangaroos and bandicoots).

Mode of transmission

Q fever is contracted through the respiratory route after inhalation of Coxiellae-contaminated dust or aerosols. This most commonly occurs as a result of:

- inhaling water droplets and dust contaminated by placental tissues, birth fluids or excreta of infected animals. Contaminated dust particles may occasionally be carried downwind for a considerable distance from the source
- direct contact with contaminated materials in establishments processing infected animals or their by-products, contact with contaminated straw, wool or hides, or the contaminated clothing of workers.

Although Q fever is occasionally transmitted sexually, there is no evidence of person to person transmission through other routes. Direct transmission through blood and bone marrow transfusion has also been reported.

Drinking non-pasteurised milk from an infected animal has been suggested as a possible route but this has not been proven.

Period of communicability

Person to person spread occurs very rarely by the sexual route. Contaminated clothing may also be a source.

Susceptibility and resistance

All non immune people are susceptible to infection. Most cases are in male adults but this is probably due to their higher frequency of exposure to high risk environments, rather than differential susceptibility.

Infection usually confers lifelong immunity.

Control measures

Preventive measures

Immunisation of those in high risk occupational groups is the primary preventive measure against Q fever. There is a risk of severe local reactions to the vaccine in those people previously exposed to Q fever or the vaccine. To assess prior exposure to Q fever or the vaccine, pre-vaccination screening is necessary and involves:

- checking for a clinical history of Q fever or Q fever vaccination
- antibody testing
- an intradermal skin test, read after seven days.

Any positive result on screening precludes vaccination. Vaccination induces lifelong immunity in most vaccinees. Training is recommended for medical practitioners intending to conduct Q fever screening and vaccination.

Access to high risk environments such as abattoirs and meat-processing plants should be restricted to immunised persons. This includes visitors, contractors and delivery drivers. Workers in these environments should also be educated about the nature of the disease.

Control of case

Acute cases of Q fever generally require treatment with doxycycline or chloramphenicol. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

In chronic disease or endocarditis, prolonged combination therapy together with cardiac surgery may be required. Consultation with an infectious diseases physician should be sought.

Isolation is not necessary. Articles contaminated with blood, sputum and excreta should be disinfected using standard precautions.

Control of contacts

No specific measures are required for household contacts.

Vaccination during the incubation period does not prevent the disease. Post-exposure prophylaxis is not recommended.

Control of environment

If a clear source is identified, disinfection can be performed using 0.05% hypochlorite (500 ppm available chlorine) or 5% peroxide.

Outbreak measures

All notified cases are investigated to ascertain the most likely source of exposure and to identify any other linked cases. If two or more cases are linked in time and place to a workplace, other staff should be assessed for immune status (if not already known) with antibody levels and skin testing.

Non-immune staff should be excluded from the worksite until vaccinated.

Rabies and Australian bat lyssavirus

Victorian statutory requirement

Rabies (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Australian bat lyssavirus (Group B disease) must be notified in writing within five days of diagnosis.

Rabies is subject to Australian quarantine.

Infectious agents

Rabies virus and Australian bat lyssavirus (ABL) are closely related members of the genus *Lyssavirus*.

Identification

Clinical features

Rabies is an acute viral disease of the central nervous system (CNS). CNS symptoms are preceded by a non-specific prodrome of fever, headache, malaise, anorexia, nausea and vomiting lasting one to four days. This is followed by signs of encephalitis manifested by periods of excitation and agitation leading to delirium, confusion, hallucinations and convulsions. Signs of brain stem dysfunction begin shortly after with excessive salivation and difficulty in swallowing. This produces the classical picture of 'foaming at the mouth'.

Even with medical intervention the disease is almost invariably fatal. Death from respiratory paralysis generally occurs within two to six days of the onset of symptoms.

The criterion for a suspect rabies case is progressive encephalitis with a past history of exposure in a rabies endemic area.

The criteria for a confirmed case are a clinically compatible neurological illness and one or more positive results from the three laboratory tests described below.

Symptoms of encephalitis due to ABL include numbness, muscle weakness, collapse and coma. A confirmed case requires laboratory definitive evidence only.

Method of diagnosis

The Australian Animal Health Laboratory at Geelong is the reference laboratory for the diagnosis of rabies and ABL. The State Chief Quarantine Medical Officer at the Department of Human Services should also be advised at the time of submitting any specimen. Transfer of human and animal specimens is coordinated by the State Chief Quarantine Medical Officer in consultation with the chief veterinary officer.

Rabies and ABL can be diagnosed by:

- detection of virus antigen by direct fluorescent antibody of a clinical specimen such as neural tissue (preferred), skin snips from the nape of the neck, saliva or CSF
- isolation in cell culture or laboratory animal of the virus from saliva, CSF or CNS tissue
- identification of rabies-neutralising antibody in the serum or CSF of unvaccinated persons.

Confirmation by all the above methods is recommended.

Incubation period

The incubation period for rabies is usually three to eight weeks. It is rarely as short as nine days or as long as seven years. It tends to be shorter for wounds in areas of the body with rich nerve supply and close to the head.

The incubation period for ABL is not well characterised but it is assumed to be similar to rabies. The first case, reported in 1996, is believed to have had an incubation period of at least several weeks. In the second patient the incubation period was greater than two years.

Public health significance and occurrence

Rabies is endemic in Asia, India, Africa, North and South America and parts of Europe. High rates of rabies are reported from the Philippines, Thailand and Indonesia with the exception of Bali, which is rabies-free.

Australia is currently rabies-free. Rabies is a very rare infection of travellers to endemic areas outside of Australia. Only two imported human cases were reported between 1900 and 1995 (1987 and 1990).

Two human cases of ABL infection have been reported. One of these was from Northern New South Wales (1996) and the other from Rockhampton in Queensland (1998). Both patients had a history of bites and scratches from a bat and both died from their infections.

Rabies is subject to human quarantine controls under the *Commonwealth Quarantine Act 1908*. Rabies is a quarantinable disease because of Australia's freedom from this disease. It is also reportable to the World Health Organization.

The primary quarantine concern is the prevention of the introduction of rabies virus to local dog and wildlife populations.

ABL is an emerging infectious disease which has much in common with rabies. The risk of human exposure increases with increasing human contact with Australian bat environments. This risk would increase significantly if ABL became established in terrestrial animal populations, particularly dogs.

Reservoir

Rabies is a disease primarily of animals. Most wild and domesticated dog-species (including foxes, coyotes, wolves and jackals) are susceptible to infection. Infected dogs remain the highest risk source for human transmission. Other species include skunks, racoons and bats.

In developed countries rabies is mainly found in wild animal hosts. Disease is spread from wild hosts to domestic animals and humans. In contrast dogs continue to be the main hosts in most African, Asian and Latin American countries, and are responsible for most of the rabies deaths that occur worldwide.

Australia is one of a growing number of countries in the world where the animal population is free of rabies.

ABL is known to infect all four *Megachiroptera* (fruit bats and flying foxes) species in Australia and at least three species of *Microchiroptera* (insectivorous bats). Ongoing serological testing and virus studies suggest that this lyssavirus is widely distributed in Australia. It is therefore assumed that all Australian bats have the potential to carry and transmit ABL.

There is no evidence that lyssaviruses in bats can establish and spread amongst terrestrial animals, although isolated cases in humans may occur on rare occasions.

Mode of transmission

Rabies virus and other lyssaviruses are usually transmitted to humans via bites or scratches which provide direct access of the virus in saliva to exposed tissue and nerve endings. It can also occur where mucous membrane exposure to bat saliva has occurred such as eyes, nose or mouth.

The most frequent way that humans become infected with rabies is through the bite of infected dogs, cats, wild carnivorous species like foxes, raccoons, skunks, jackals and wolves, and insectivorous and vampire bats. Cattle, horses, deer and other herbivores can become infected with rabies but rarely transmit the virus to other animals, although they may transmit the disease to humans.

People are not exposed to ABL through tactile contact with bats where parenteral or mucous membrane exposure does not occur. Contact such as patting bats or exposure to urine and faeces does not constitute a likely exposure to ABL, although bat urine and faeces may carry other human pathogens.

Transmission from person to person is theoretically possible but it has only ever been documented through corneal transplantation.

Period of communicability

In dogs and cats rabies is usually communicable three to seven days before onset of clinical signs and throughout the course of the illness. Viral excretion up to 14 days prior to clinical signs has been observed in some animal species. Similar communicability can be assumed for human cases.

Communicability for ABL is not known but assumed to be similar to rabies.

Susceptibility and resistance

All mammals are susceptible to varying degrees. In one case series, only 40% of children bitten by known rabid dogs developed the disease.

Control measures

Preventive measures

Pre-exposure vaccination is recommended for people whose occupation or recreational activities place them at increased risk of being bitten or scratched by a bat. It is also recommended for travellers who will be spending prolonged periods (i.e. more than one month) in rural parts of rabies endemic areas (see rabies/ABL vaccination information sheet below).

The World Health Organization maintains data on rabies infected countries – see www.who.int/csr

Control of case
There is no specific treatment available. Intensive supportive treatment is required.

The patient should be placed in a private room with standard isolation precautions implemented for respiratory secretions for the duration of the illness. There should be concurrent disinfection of all saliva-contaminated articles. Although transmission from a patient to attending carers has not been documented, health care workers should be advised to wear gowns, gloves and masks while attending patients. Blood and urine are not considered infectious.

Control of contacts
Other individuals exposed to the source animal are identified and offered post-exposure prophylaxis. Contacts that have open wound or mucous membrane exposure to a patient's saliva should be offered full post-exposure prophylaxis.

Post-exposure treatment
Proper cleansing of the wound is the single most effective measure for reducing the transmission of rabies virus and this is likely to be also true for ABL.

When a person has been injured by a potentially infected animal overseas, or any Australian bat, the wound should be washed thoroughly for approximately five minutes as soon as possible with soap and water. If available, a virucidal antiseptic such as povidone-iodine, iodine tincture, aqueous iodine solution or alcohol (ethanol) should be applied after washing. Exposed mucous membranes such as eyes, nose or mouth should be flushed well with water.

The decision to offer post-exposure prophylaxis (rabies vaccine and rabies immunoglobulin) to a potentially exposed person should be made in consultation with the Communicable Diseases Section of the Department of Human Services (see rabies/ABL vaccination information sheet below).

Control of environment
See Outbreak measures, below.

Outbreak measures

If the source of the ABL infection is likely to be in Australia, a search should be made for the infected animal in collaboration with animal health authorities. Where possible, without placing other persons at risk of exposure, the bat should be kept and the Department of Human Services consulted about arranging testing of the bat for virus carriage.

If a rabies case, human or animal, is believed to have been locally acquired, the AUSVETPLAN rabies control procedures should be implemented. In designated areas animal owners may be required to have susceptible animals vaccinated with rabies vaccine. Animal movements are restricted and stray animals destroyed.

ABL is unique to Australia and currently it is only found in Australian bat species. If a human case of ABL is diagnosed in Victoria or ABL is found in another animal species such as a dog or cat, investigation and control measures similar to those for a rabies case, should be instigated.

Additional sources of information

- Animal Health Australia 1996, *AUSVETPLAN*, http://www.aahc.com.au/ausvetplan/
- Communicable Diseases Network of Australia 2001, *Australian bat lyssavirus – Information for medical practitioners*.

Rabies and Australian bat lyssavirus exposure information sheet

Pre-exposure prophylaxis

Pre-exposure vaccination should be recommended to those people whose occupation or recreational activities place them at increased risk of being bitten or scratched by a bat. For example:

- bat carers, bat handlers, researchers and students
- veterinarians and veterinary assistants
- veterinary laboratory staff
- fruit pickers
- wildlife officers (including local government officers)
- managers of display or research colonies of bats
- power line workers who frequently remove bats from power lines.

Pre-exposure vaccination should also be recommended for travellers who will be spending prolonged periods (i.e. more than one month) in rural parts of rabies endemic areas. The World Health Organization maintains data on rabies infected countries – see www.who.int/csr

Pre-exposure prophylaxis consists of three deep subcutaneous or intramuscular doses of 1.0 mL rabies vaccine given on days 0, 7 and 28. Doses should be given in the deltoid area, as rabies neutralising antibody titres may be reduced after administration in other sites. In children, administration into the anterolateral aspect of the thigh is also acceptable.

The vaccine should not be administered by the intradermal route.

Post-exposure treatment for persons bitten or scratched

The decision to offer post-exposure prophylaxis to a potentially exposed person should be made in consultation with the Department of Human Services. If post-exposure prophylaxis is indicated, the Department of Human Services will arrange for rapid delivery of vaccine and immunoglobulin as required.

Post-exposure treatment should be considered in the following scenarios:

- person bitten or scratched by bats in Australia
- person bitten or scratched by any animal in a country with endemic rabies.

Assessment

Rabies virus and other lyssaviruses are usually transmitted to humans via bites or scratches which provide direct access of the virus in saliva to exposed tissue and nerve endings, or where mucous membrane such as eyes, nose or mouth exposure to bat saliva has occurred. This means that people would not be exposed to lyssavirus through tactile contact with bats alone or other animals where parenteral or mucous membrane exposure does not occur. Contact such as patting bats (Australia) or other animals or exposure to their urine and faeces does not constitute a possible exposure to ABL, although bat urine and faeces may carry other human pathogens. Pre-exposure vaccination should however be offered if the person has ongoing contact with bats.

If the exposure is connected to an Australian bat, where possible without placing other persons at risk of exposure, the bat should be kept so that the Department of Human Services can arrange for testing of the bat.

First aid

Proper cleansing of the wound is the single most effective measure for reducing the transmission of classic rabies virus.

Where a person has been injured by a potentially infected animal, the wound should be washed thoroughly for approximately five minutes as soon as possible with soap and water. If available, a virucidal antiseptic such as povidone-iodine, iodine tincture, aqueous iodine solution or alcohol (ethanol) should be applied after washing. Exposed mucous membranes such as eyes, nose or mouth should be flushed well with water.

Post-exposure treatment – not previously vaccinated against rabies

Rabies vaccine

Post-exposure prophylaxis for persons not previously immunised against rabies consists of five doses of 1.0 mL of rabies vaccine given as deep subcutaneous or intramuscular injection, on days 0, 3, 7, 14 and 28. Doses should be given in the deltoid area, as rabies neutralising antibody titres may be reduced after administration in other sites. In children, administration into the anterolateral aspect of the thigh is also acceptable. The vaccine should not be administered by the intradermal route.

Rabies immunoglobulin

Rabies immunoglobulin (RIG) should be given as a single dose at the same time as the first dose of the post-exposure vaccination course. The dose for RIG is 20 International Units (IU) per kilogram of body mass. RIG should be infiltrated in and around all wounds using as much of the calculated dose as possible, and the remainder administered intramuscularly. It should not be given at the same site as the vaccine, and if administered in the buttock, care should be taken to ensure that the dose is given intramuscularly and not into adipose tissue.

Although the RIG and first dose of rabies vaccine should preferably be given on the same day, if necessary the RIG can be given up to seven days after the first dose of vaccine, but not thereafter.

RIG should be infiltrated into finger wounds using a 25 or 26 gauge needle, and to avoid a compartment compression syndrome the RIG should be infiltrated very slowly, and should not cause the adjacent finger tissue to go pale or white. If necessary a ring-block using local anaesthesia may be required. If the wounds are severe and the calculated volume of RIG is inadequate for complete infiltration, the RIG may be diluted in saline to make up an adequate volume for the infiltration of all wounds, but as most bat bites are small and fine, this should not be necessary.

Post-exposure treatment - previously vaccinated against rabies

Post-exposure prophylaxis for persons who have previously completed the recommended course of either pre-exposure vaccination or post-exposure prophylaxis or who have documented rabies neutralising antibodies, comprises a total of two doses of rabies vaccine (1.0 mL each) given by either deep subcutaneous or intramuscular injection on day 0 and day 3. In cases where prior vaccination status is uncertain, or the person has been vaccinated by inappropriate intradermal injection, a full course of post-exposure prophylaxis (RIG plus five doses of vaccine) should be offered. It is therefore advisable to ensure that people are given adequate written documentation as to any RIG and vaccines administered.

(Note that product information recommends a routine 6th dose at 90 days. This dose is not considered necessary, except for immunosuppressed persons. See the current edition of the *Australian immunisation handbook*, National Health and Medical Research Council, for more information).

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160
- Australian Immunisation Handbook, www.immunise.health.gov.au

Victoria
The Place To Be

Rickettsial infections

Includes scrub typhus and Queensland tick typhus (spotted fever)

Victorian statutory requirement

Notification and school exclusion are not required.

Infectious agents

Numerous species of *Rickettsiae* are of concern to humans. *Rickettsiae* (and their associated diseases) of particular importance in Australia are *R. australis* (Queensland tick typhus, Spotted fever), *R. tsutsugamushi* (Scrub typhus), *R. honei* (Flinders Island spotted fever) and *R. typhi* (murine typhus).

Identification

Clinical features

There is great variation in the severity of illness produced by each organism. Infection most commonly begins with a papule forming at the site of the bite where the infection was introduced. This usually becomes necrotic and forms a typical black eschar (scab). Four days to two weeks after the bite symptoms begin with fever and malaise followed by adenitis in the lymph glands draining the bite site. As the organisms spread throughout the body, fever, malaise and headache increase and general lymphadenopathy occurs in most cases. About a week after onset the main features are continuous fever, cough and signs of bronchitis or pneumonia, photophobia, conjunctivitis, generalised adenopathy, delirium, deafness and a maculopapular rash most commonly over the trunk and proximal limb parts. Splenomegaly occurs in some cases.

Fever may persist for 14 days without antibiotic treatment. The fatality rate in untreated cases is 1–40%. This increases with age and depends on the infection site, the type of *Rickettsiae* involved and previous exposure.

Method of diagnosis

In endemic areas the clinical picture is sufficiently distinctive for a clinical diagnosis. A biopsy of the eschar can be used to demonstrate rickettsiae by immunofluorescence. Specific diagnosis is seldom possible early enough to help in the management.

Definitive diagnosis can be made by isolation of the rickettsia after inoculation of the patient's blood into mice. Serological methods are also available although these need to be interpreted with caution because of cross-reactivity between strains.

Incubation period

The incubation is from two to 14 days. The variation in incubation may be in part related to the inoculum size.

Public health significance and occurrence

The epidemiology varies in different parts of the world. Disease occurrence is often associated with the modification of natural habitats by humans such as when a forest is felled and replaced by a secondary growth of scrub. *R. australis* occurs along the eastern side of Australia, *R. honei* has been recognised on Flinders Island near Tasmania and *R. typhi* occurs throughout many states of Australia. Scrub typhus occurs in Queensland but its geographic distribution in the rest of Australia is less clear (Odorico, Graves et al 1998). The public health impact on lives or productivity lost is largely unmeasured but it is suspected to be high.

Reservoir

Humans are incidental hosts and are not useful in propagating the organism in nature. Scrub typhus is transmitted by rodent mites. It occurs in a large area from the Indian subcontinent to Australia and in much of Asia including Japan, China, Korea and parts of Russia. The reservoir also includes rats, mice and other small mammals. An exception is louse-borne typhus (*R. prowazekii*), which does not occur in Australia. Humans are the principal reservoir for louse-borne typhus and the human body louse (*pediculosis humanus var humanus*) is the vector.

Mode of transmission

The disease is not directly transmitted from person to person. Humans are infected by the bite of an infected larval mite or in the case of scrub typhus, a rat.

Period of communicability

The person is infective for lice during the febrile illness and probably two or three days after the temperature returns to normal. People are at risk of infection for as long as they remain in infected areas.

Susceptibility and resistance

All non immune people are susceptible to infection and according to environmental exposure. Long-lasting immunity probably follows infection.

Control measures

Preventive measures

There is no vaccine available. People who enter infected areas can be protected by impregnating their clothing with dimethyl phthalate and renewing the repellent frequently. Chemoprophylaxis can be successfully used short term and for this a consultation with an infectious diseases specialist is recommended. People camping can also help prevent tick bites by using camp beds for elevation from the floor.

Control of case

Treatment is generally doxycycline or chloramphenicol. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

In severe disease, consultation with an infectious diseases specialist is recommended.

Control of contacts

Consider active case finding if other people were exposed to the same setting as the case such as a camping holiday or military exercise.

Control of environment

Not applicable. The mites themselves act as reservoirs so no immediate effect is achieved by rodent control.

Special settings

Not applicable.

Outbreak measures

Except in the case of an epidemic of louse borne typhus, no outbreak measures are necessary.

International measures

In the event of an epidemic of louse borne typhus occurring in Australia, the Department of Human Services will notify the World Health Organization (WHO) and neighbouring countries of this occurrence in an area previously free of the disease.

Additional sources of information

- Bell, D 1995, *Tropical Medicine*, 4th edn, Blackwell Science.
- Odorico, D, Graves, S, Currie, B, Catmull, J, Zoltan, N, Ellis, S, Wang, L and Miller, D 1998, 'New Oriental *tsutsugamushi* Strain from Scrub Typhus in Australia'. *Emerging Infectious Diseases*, vol. 4, no. 4.

Ringworm or tinea

Tinea capitis (head), tinea corporis (body), tinea pedis (feet), tinea unguium (nails)

Victorian statutory requirement

Notification is not required.

School exclusion: readmit the day after appropriate treatment has commenced.

Infectious agent

Microsporum spp. includes *Microsporum canis* as the primary causative agent in Australia of tinea capitis and corporis. *Trichophyton* spp. also cause disease for example *T. rubrum*, *T.mentagrophytes* and *Epidermophyton floccosum*.

Identification

Clinical features

The clinical features of tinea infections are those of superficial fungal infection of the skin, nails or hair:

- tinea capitis results in a small papule that spreads peripherally leaving fine, scaly patches of temporary baldness. Infected hairs become brittle and break off easily.
- tinea corporis appears as a flat, red, ring-shaped lesion of the skin. It is usually dry and scaly or moist and crusted but sometimes contains fluid or pus. The lesion tends to heal centrally.
- tinea pedis is commonly known as 'athlete's foot'. It occurs as itchy, scaling, cracking of the skin or blisters containing a thin watery fluid. This occurs commonly between the toes.
- tinea unguium is a chronic fungal disease involving one or more nails of the hands or feet. The nail gradually thickens and becomes discoloured and brittle. Caseous-looking material forms beneath the nail or the nail becomes chalky and disintegrates.

Method of diagnosis

Diagnosis can be made by microscopic examination of material from the affected area or by fungal culture.

Incubation period

The incubation period differs:

- tinea corporis has an incubation period of four to ten days
- tinea capitis has an incubation period of 10–14 days
- the incubation period of tinea pedis and tinea unguium is probably weeks but exact limits are unknown.

Public health significance and occurrence

Tinea capitis mainly affects children.

M. canis is usually contracted from infected kittens or puppies.

The highly contagious *M. audouinii* spreads from person to person and does not occur in Australia.

Tinea capitis may extend to tinea corporis. It occurs worldwide.

Tinea corporis occurs worldwide and relatively frequent. Males are infected more than females. Infection can occur from direct or indirect contact with skin and scalp lesions of infected persons or animals.

Tinea pedis occurs in children and adults and is spread by using communal facilities such as showers at swimming pools. Adults are affected more often than children and males more than females. Infection is more frequent and severe in hot weather.

Tinea unguium occurs commonly but there are low rates of transmission, even to close family associates. It is spread by direct contact with skin or nail lesions of infected persons or indirectly through contact with contaminated floors or showers.

Reservoir

Reservoirs for tinea are:

- tinea capitis: humans and animals including dogs, cats and cattle
- tinea corporis: humans, soil and animals including cattle, kittens, puppies, guinea pigs, mice and horses
- tinea pedis: humans
- tinea unguium: humans and rarely animals or soil.

Mode of transmission

Direct transmission occurs through human to human contact, for example *T. rubrum* and *T.mentagrophytes*. Animal-to-human contact also occurs, for example *M. canis* and *T. verrucosum*. Tinea can be transmitted indirectly through contaminated soil, for example *M. gypseum*.

Period of communicability

The fungus persists on contaminated materials as long as lesions or animal hair harbour viable spores.

Susceptibility and resistance

Young children are particularly susceptible to tinea capitis (Microsporum canis). All ages are susceptible to infections particularly those caused by Trichophyton spp.

Susceptibility to tinea corporis is widespread. It is aggravated by friction and excessive perspiration in axillary and inguinal regions, and when environmental temperatures and humidity are high.

Susceptibility is variable for tinea pedis and infection may be inapparent. Repeated attacks are frequent.

An injury to the nail predisposes to tinea unguium infection. Reinfection is frequent.

Control measures

Preventive measures

Measures differ according to cause:

- for tinea capitis parents should be educated about modes of spread from infected children and animals
- for tinea corporis shower bases, mats and floors adjacent to showers should be disinfected. Infected animals should be avoided
- for tinea pedis gymnasiums, showers and similar sources of infection should be thoroughly cleaned and washed. Shower areas should be frequently hosed and rapidly drained. Users of such areas should be encouraged to carefully dry (and perhaps powder) between their toes.

Control of case

Control depends on the cause:

- for tinea corporis infected children should be excluded from schools and swimming pools until at least 24 hours following the commencement of appropriate treatment. It can be treated effectively with topical medications
- for tinea capitis oral griseofulvin is the treatment of choice for resistant infection, for example *T. tonsurans.* Topical anti-fungal medication may be used concurrently
- for tinea pedis topical fungicides are recommended but oral griseofulvin may be indicated in severe protracted disease. Feet should be kept dry as possible and exposed to air by wearing sandals. Socks of heavily infected individuals should be boiled or discarded to prevent reinfection
- for tinea unguium oral terbinafine should be given daily for six weeks for finger nails and twelve weeks for toe nails.

Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Note: M. canis infection is self-limiting in children before puberty and griseofulvin may not be necessary. Consult a specialist about treatment.

Control of contacts

Investigate household contacts, pets and farm animals for evidence of infection. Treat infected contacts, human or animal.

Control of environment

See Preventative measures, above.

Outbreak measures

Children and parents should be educated about modes of spread, prevention and the necessity of maintaining a high standard of personal hygiene. In case of epidemics, consider examination of all children to identify cases. Disinfect contaminated articles.

Ringworm (tinea) information sheet

What is ringworm?

Ringworm is a fungal infection that can affect any part of the body.

How do you get ringworm?

Ringworm is spread by direct and indirect contact with humans, animals, and soil.

Humans get infections through skin and scalp lesions of infected persons, contaminated clothing, bath mats, towels, floors and showers.

Animals get infections through cats, dogs, mice, and guinea pigs. Cattle and horses may be infected.

How long is the incubation period?

The incubation period lasts from one to three weeks. It varies with the site of infection.

How do you recognise ringworm?

Ringworm of the skin – This appears as a flat, spreading, circular lesion with a reddish outer edge. It is usually dry and scaly or moist and crusted, but it may contain fluid or pus. Single or multiple rings may appear. The centre of the patch may appear to be healing.

Ringworm of the scalp and beard – This condition begins as a small pimple. It spreads outward leaving fine, scaly patches of temporary baldness. Infected hairs become brittle and break off easily.

Ringworm of the foot (commonly known as tinea or athlete's foot) – The characteristics of this common condition are itchy scaling or cracking of the skin, especially between the toes, or blisters containing a thin watery fluid.

Ringworm of the nail – This condition tends to be a long term fungal disease and is difficult to treat. It usually affects one or more nails of the hands or feet. The nail gradually thickens and becomes discoloured and brittle. Cheesy-looking material forms beneath the nail, or the nail becomes chalky and disintegrates.

How do you control ringworm?

Seek medical advice to confirm diagnosis and receive appropriate treatment. Exclude infected persons from communal swimming and bathing facilities until appropriate treatment has commenced.

Maintain hygiene by regular, thorough bathing with soap and water and special attention to drying moist areas.

- Do not share clothing or personal linen.
- Frequently launder clothing and linen in hot water.
- Wash pets with anti-fungal solution.
- Dry carefully between toes.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Ross River virus disease

Arboviruses are viruses which are spread by the bite of arthropods, particularly mosquitoes. They are divided into alphaviruses and flaviviruses.

Three infective alphaviruses include Ross River, Barmah Forest and Sindbis viruses.

These all have the capacity to cause a similar disease in humans characterised by fever, joint involvement and a rash. Molecular studies of epidemiologically distinct isolates of Ross River and Sindbis viruses have shown changes in isolates from different areas (distinct topotypes). This may explain varying disease patterns which sometimes occur in certain geographic locations and the differing transmissibility of some strains by different vector mosquitoes.

Victorian statutory requirement

Ross River virus infection (Group B disease) requires notification within five days of diagnosis.

School exclusion is not required.

Infectious agent

First isolated in 1959 from *Aedes vigilax* mosquitoes collected near the Ross River in Townsville, the causative role of Ross River virus (RRV) was confirmed in 1971 by its isolation from the blood of an indigenous child with the disease. Some Aedes species have recently been renamed *Ochlerotatus* spp. mosquitoes.

Identification

Clinical features

Pyrexia and other constitutional symptoms are usually slight. A rash can occur up to two weeks before or after other symptoms. It can be absent in about one–third of cases. The rash is variable in distribution, character and duration and may be associated with buccal and palatal enanthems. Rheumatic symptoms are present in most patients except for the few who present with rash alone: these consist of arthritis or arthralgia primarily affecting the wrist, knee, ankle and small joints of the extremities. Prolonged symptoms are common. In some cases there may be remissions and exacerbations of decreasing intensity for years. Cervical lymphadenopathy occurs frequently and paraesthesiae and tenderness of the palms and soles are present in a small percentage of cases.

Method of diagnosis

Serology shows a significant rise in antibody titre to RRV. The virus may be isolated from the blood of acutely ill patients. Virological tests are necessary to distinguish RRV disease from other causes of arthritis. In the event of a local outbreak clinical diagnosis may be sufficient, but outbreaks of RRV disease sometimes occur concurrently with BFV disease making diagnosis difficult.

Laboratory evidence requires one of the following:

- isolation of RRV from clinical material
- detection of RRV by nucleic acid testing
- a significant rise in IgG to RRV
- detection of RRV-specific IgM.

Incubation period

The incubation period is usually three to eleven days.

Public health significance and occurrence

Infection is subclinical in up to 60% of cases. Clinical features of infection are rare before puberty after which the disease has a similar pattern at all ages. The disease can cause incapacity and inability to work for two to three months. About one quarter of patients have rheumatic symptoms which persist for a year or more.

RRV disease is the commonest and most widespread arboviral disease in Australia, sometimes thousands of clinical cases occur in epidemics. Disease notifications in Australia average about 4800 per year. Major outbreaks have occurred in all parts of Australia. These occur chiefly in the period from January to May. RRV has been detected and probably transmitted to humans in most major metropolitan areas of Australia including Perth, Brisbane, Sydney and Melbourne. In 1993, 1216 cases of RRV disease were notified in Victoria. Epidemics usually follow heavy rains or after high tides which inundate salt marshes or coastal wetlands. Sporadic cases occur in mainland and coastal regions of Australia and Papua New Guinea at other times of the year. In 1979, a major outbreak of RRV disease which was probably exported from Australia occurred in Fiji and spread to other Pacific islands, including Tonga and the Cook Islands.

Reservoir

The virus is maintained in a primary mosquito–mammal cycle involving macropods (particularly the Western Grey kangaroo) and possibly other marsupials and wild rodents. A man–mosquito cycle may occur in explosive outbreaks. Horses can act as amplifier hosts and appear to develop joint and nervous system disease after infection with RRV. Fruit bats might act as vertebrate hosts in some areas. Vertical transmission in desiccation-resistant eggs of Ochlerotatus spp. mosquitoes may be a mechanism to enable the virus to persist in the environment for long periods. This could explain the rapid appearance of cases of RRV disease after heavy rains. RRV is endemic throughout Australia, Papua New Guinea, adjacent Indonesia and the Solomon islands

Mode of transmission

RRV is transmitted by mosquitoes. Culex annulirostris is the major vector in inland areas whilst O*chlerotatus vigilax* in New South Wales and *Ochlerotatus camptorhynchus* in southern parts of Victoria and Tasmania are the vectors in coastal regions.

Period of communicability

There is no evidence of transmission from person to person.

Susceptibility and resistance

Infection with the RRV confers lifelong immunity.

Control measures

Preventive measures

RRV infection can be prevented by:

- mosquito control measures
- personal protection measures such as wearing long sleeves and mosquito repellents
- avoidance of mosquito-prone areas and exposure during biting times at dusk and dawn.

Control of case

Second attacks are unknown. Treatment is symptomatic with rest advisable in the acute stages of the disease. There is no vaccine currently available commercially to protect against RRV disease.

Control of contacts

Unreported or undiagnosed cases should be sought in the region where the patient had been staying during the incubation period of their illness. All family members should be questioned about symptoms and evaluated serologically if necessary.

Control of environment

To reduce or prevent virus transmission, interruption of human-mosquito contact is required by:

- suppression of the vector mosquito population
- avoidance of vector contact through personal protection and education.

Outbreak measures

Conduct a community survey to determine the species of the vector mosquito involved. Identify their breeding places and promote their elimination.

Use mosquito repellents for persons exposed to bites because of their occupation, or other reasons.

Identify the infection among animal reservoirs, for example kangaroos, small marsupials, farm and domestic animals.

International measures

Airport vector control in Australia and Papua New Guinea may be necessary to prevent spread from endemic areas to other countries where local vectors such as *Aedes polynesiensis* may transmit the disease.

Rotaviral gastroenteritis

Victorian statutory requirement
Isolated cases are not notifiable.

School exclusion: exclude from school or child care centre until at least 48 hours after symptoms have ceased.

Infectious agent
Rotavirus, predominantly Group A, is the causative agent.

Identification
Clinical features
The disease is characterised by vomiting and watery diarrhoea lasting for three to eight days. Fever and abdominal pain occur frequently. Treatment is symptomatic. Maintenance of hydration is the most important measure.

Method of diagnosis
Diagnosis may be made by rapid antigen detection of rotavirus in stool specimens. Strains may be further characterised by enzyme immunoassay or reverse transcriptase polymerase chain reaction. Stools for these tests should be collected in the acute phase of illness.

Incubation period
The incubation period is approximately 24–72 hours.

Public health significance and occurrence
Disease usually occurs in infants and young children, particularly under two years of age. Adults can also be infected although their resultant disease tends to be mild. In temperate climates it is more common in the winter months. Rotavirus gastroenteritis is the leading cause of infant viral gastroenteritis worldwide. The cost of managing rotavirus disease in Australia is estimated at $26 million annually.

Reservoir
Humans.

Mode of transmission
Rotavirus is transmitted predominantly via the faecal-oral route. Rotavirus has been detected in respiratory secretions. Because the virus is stable in the environment transmission can occur through ingestion of contaminated water or food and contact with contaminated surfaces.

Period of communicability
Rotavirus is communicable during the acute stage of disease and while viral shedding continues. Excretion of virus for greater than 30 days has been documented.

Susceptibility and resistance
Everyone is susceptible to infection. Immunity after infection is incomplete, but repeat infections tend to be less severe than the original infection.

Control measures
Preventive measures
Prevention is primarily through good personal, food and home hygiene.

Control of case
Provide advice regarding personal hygiene, exclusion from work or school or child care and attempt to identify source of infection. Health care workers and food handlers should be excluded from work until at least 48 hours after diarrhoea has ceased.

Control of contacts
Identify whether any contacts are ill. Provide advice about strict personal, food and home hygiene.

Control of environment
Rigorous attention to clean-up procedures and personal and home hygiene is essential to prevent further transmission.

Outbreak measures
An outbreak is defined as two or more related cases of gastroenteritis. The primary aim is to prevent further disease by identifying the source, cleaning contaminated environments and isolating cases.

Special settings
Specific protocols for the management of outbreaks in special settings are available from the Communicable Diseases Section of the Department of Human Services, phone 9637 4126.

Additional sources of information
- Centers for Disease Control and Prevention, Atlanta USA, *Viral gastroenteritis*, http://www.cdc.gov/ncidod

Rubella
(German measles)

Victorian statutory requirement
Rubella and congenital rubella syndrome (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: excluded until fully recovered or at least four days after the onset of the rash.

Infectious agent
Rubella virus of the *Togaviridae* family is the infective agent.

Identification
Clinical features

Rubella is a mild febrile viral illness characterised by a diffuse punctate and maculopapular rash. Children usually experience few or no constitutional symptoms but adults may experience a one to five day prodrome of low-grade fever, headache, malaise, mild coryza and conjunctivitis. Postauricular, occipital and posterior cervical lymphadenopathy is common and precedes the rash by five to ten days.

Complications include arthralgia and less commonly arthritis, particularly among adult females. Encephalitis is a rare complication.

Congenital rubella syndrome (CRS) occurs in less than 25% of infants born to women who acquire rubella during the first trimester of pregnancy. The risk of a single congenital defect falls to approximately 10–20% by the 16th week of pregnancy. Defects are rare when the maternal infection occurs after the 20th week of gestation.

Differential diagnosis includes measles, human parvovirus ('slapped cheek') infection, human herpesvirus 6 (roseola) infection and a large number of other rashes of varied aetiology.

Method of diagnosis

Clinical diagnosis should be confirmed by one or more of the following:

- demonstration of rubella-specific IgM antibody, except following rubella immunisation
- fourfold or greater rise in rubella antibody titre between acute and convalescent-phase sera obtained at • least two weeks apart
- isolation of rubella virus from a clinical specimen.

Consider also testing for other similar exanthems such as measles and human parvovirus.

Incubation period
The incubation period is usually 14 to 17 days. It ranges from 14 to 21 days.

Public health significance and occurrence
Rubella occurs worldwide and is universally endemic except in remote and isolated communities. It is most prevalent in winter and spring.

A combined measles-mumps vaccine was first added to the routine childhood immunisation schedule in 1983. Although this has clearly led to a dramatic reduction in the number of reported cases the epidemiology of rubella infection in Australia is not clear because of the acceptance of clinical diagnoses without laboratory confirmation.

Routine serological testing of reported clinical cases of rubella in Victoria has revealed that only a small proportion of these cases can be confirmed in the laboratory. The remainder are likely to be due to other causes.

Unimmunised travellers and their unimmunised contacts remain at risk of infection.

Congenital rubella syndrome (CRS) was a major cause of congenital abnormalities including deafness prior to the infant immunisation program. Although CRS is now rare, the risk of infection remains for unimmunised pregnant women. Such women have been infected primarily by persons who have not been included in rubella vaccine programs.

Reservoir
Humans.

Mode of transmission
Rubella is transmitted by droplet spread or direct contact with infectious patients.

Infants with CRS shed the rubella virus in their nose, pharyngeal secretions and urine for months or even years.

Period of communicability
Rubella is communicable approximately one week before and for at least four days after the onset of the rash.

CRS infants may shed the virus for months or longer after birth.

Susceptibility and resistance

Immunity after natural disease is usually life long. Immunity after vaccination is long term and usually lifelong, although reinfection of vaccinees has been observed.

Passive maternal immunity is acquired transplacentally. Infants born to immune mothers are ordinarily protected for six to nine months depending on the amount of maternal antibodies transferred.

Control measures

Preventive measures

MMR vaccine is recommended in the ASVS for all infants at the age of 12 months and at again at four years of age.

Women of childbearing age should be tested for immunity to rubella prior to pregnancy if possible. All non-pregnant seronegative women should be offered rubella vaccine.

Women receiving rubella vaccine should be instructed to avoid pregnancy for 28 days after vaccination. Inadvertent rubella vaccination during pregnancy has not been associated with any CRS-like defects; it is not necessary to consider termination.

Women attending for antenatal care who are unaware of their immune status should be tested for rubella antibodies and if negative, be vaccinated immediately post partum.

All health care workers should receive MMR vaccine if not immune.

Control of case

There is no specific treatment.

The case should be excluded from school and childcare for at least four days after onset of the rash. Adults should not go to work for the same period of time.

Patients with rubella should avoid contact with other people while infectious, particularly pregnant women.

If a person with suspected rubella is pregnant, the diagnosis should be confirmed serologically and the patient referred to a specialist obstetrician for advice, taking care not to expose other pregnant women to possible infection in the process.

Control of contacts

School contacts should not be excluded from school regardless of immunisation status.

Although immunisation is generally recommended for non-immune contacts (except pregnant women) it is unlikely to reduce the risk of infection or illness. Immunoglobulin is not generally recommended, except for pregnant contacts.

Pregnant women in whom immunity to rubella has not been confirmed for the current pregnancy and who may have been exposed to rubella must be investigated serologically. This should occur irrespective of a history of vaccination, clinical rubella or previous positive rubella antibody.

Immunoglobulin should be considered after exposure to rubella in early pregnancy. It may not prevent infection or viraemia, but may modify abnormalities in the baby.

Control of environment

Not applicable.

Outbreak measures

All suspected outbreaks should be reported promptly to the Department of Human Services.

Mass immunisation may be recommended during an outbreak of rubella in a school regardless of immune status.

Salmonellosis

Victorian statutory requirement

Salmonellosis (Group B disease) must be notified in writing within five days of diagnosis.

Laboratories are required to notify *Salmonellae* isolated from food or water.

School exclusion: exclude cases from child care and school until after the diarrhoea has ceased.

Infectious agent

Approximately 2000 known serotypes exist of *Salmonella* spp, a small number of which usually account for the majority of infections.

Identification

Clinical features

Salmonellosis commonly presents as an acute gastroenteritis with fever, vomiting, nausea, abdominal pain, headache and diarrhoea. Dehydration may occur, especially among infants and the elderly. Infection may also present as septicaemia and occasionally may be localised in other body tissues resulting in endocarditis, pneumonia, septic arthritis, cholecystitis and abscesses. Symptoms usually last three to five days.

Method of diagnosis

Infection is diagnosed by isolation of *Salmonella* spp. from faeces, blood or other clinical specimen.

Incubation period

The incubation period is usually 6–72 hours with an average of 12–36 hours.

Public health significance and occurrence

Salmonella infection occurs worldwide and only a small proportion of cases are detected and reported. The incidence of infection is highest in infants and young children. Mortality is low however it may be increased in the elderly and immunocompromised people. Salmonellosis may incur significant social and economic costs due to lost productivity and the impact on industry and agriculture.

There are approximately 1000 cases of salmonellosis reported in Victoria each year. The most common serovar is *S. typhimurium*. The majority of cases are sporadic, but outbreaks in institutions and child care centres and those associated with retail food premises are not uncommon. The emergence of strains resistant to single or multiple antibiotics is of increasing concern worldwide.

Reservoir

Domestic and wild animals including poultry and reptiles act as reservoirs. Patients and convalescent carriers including mild and unrecognised cases can also act as reservoirs.

Mode of transmission

Transmission is via person to person or animal to person spread via the faecal-oral route.

Ingestion of the organisms via contaminated or improperly cooked foods also occurs. This occurs particularly with:

- raw and undercooked eggs and egg products
- raw milk and raw milk products
- poultry and poultry products
- raw red meats
- unwashed salads, fruits and vegetables, grains, seeds and nuts
- some shellfish and filter feeders such as oysters

Period of communicability

Salmonellosis is communicable through the course of infection, usually several days to several weeks. One per cent of infected adults and five per cent of children under the age of five years excrete the organism for more than one year. Antibiotics given in the acute illness can prolong the carrier state.

Susceptibility and resistance

Susceptibility may be increased by some medical conditions and treatments including immunosuppressant therapy, prior or concurrent broad-spectrum antibiotic therapy, gastrointestinal surgery, antacid use, achlorhydria and malnutrition.

Severity of the disease varies with:

- the serotype
- the numbers of organisms ingested
- the vehicle of transmission
- host factors.

Control measures

Preventive measures

Thoroughly cook all food derived from animals sources, particularly poultry, pork, egg products and meat dishes. Inadequate temperature control and incorrect storage of food during and after the cooking process facilitates bacterial multiplication and are important risk factors.

- Avoid recontamination from raw food within the kitchen or refrigerator, after cooking is completed.
- Emphasise the importance of refrigerating food and maintaining a sanitary kitchen.
- Avoid consuming raw or incompletely cooked eggs, or using dirty or cracked eggs.
- Pasteurise all milk and egg products.
- Educate food handlers on the importance of hand washing and separating raw and cooked foods.
- Inspect and supervise abattoirs, butcher shops, food-processing plants and egg-grading stations.

Control of case

Treatment is supportive and antibiotics are not indicated in uncomplicated gastroenteritis as they may prolong the carrier state and promote antibiotic resistance. The exceptions are patients at high risk of more severe disease including infants under two months of age, the elderly and immunocompromised (particularly those with HIV), and food handlers who are chronic carriers. For systemic disease, the choice of antibiotic should be based on the antibiograms of the relevant serovar and local antibiotic guidelines.

Use standard enteric precautions when handling faeces, contaminated clothing and bed linen from hospitalised patients.

Exclude symptomatic cases from food handling and direct care of children, the elderly and immunosuppressed patients until after the diarrhoea has ceased. Children are excluded from school and child care until diarrhoea has ceased.

Instruct asymptomatic individuals in strict personal hygiene. Stress proper hand washing.

Control of contacts

Consider the diagnosis in symptomatic contacts. Active case finding is not routinely undertaken in sporadic cases.

Control of environment

Sources of contamination such as use of uncooked products and inadequate cooking should be investigated. Attention should be paid to environmental cleaning, particularly in institutions, child care centres and food premises.

Outbreak measures

Two or more related cases of gastroenteritis are suggestive of an outbreak and should be reported to the Department of Human Services immediately. The aims of an outbreak investigation are to rapidly identify the source and prevent further cases. Epidemiological, environmental and laboratory investigations will be implemented immediately.

Stools should be collected from cases and attempts made to identify a common source by obtaining food histories and potentially relevant environmental exposures. Any implicated foods should be retained for analysis at the Microbiological Diagnostic Laboratory. Staff of the Communicable Diseases Section, the Department's Food Safety Unit, and Local Government Environmental Health Units usually conduct environmental investigations.

Refer to the Department's *Guidelines for the investigation of gastrointestinal illness* for specific details.

International measures

International outbreaks are increasingly being recognised, primarily due to the increased dissemination of food and agricultural products worldwide. Investigation of imported products should be coordinated through Food Standards Australia New Zealand.

Scabies

Victorian statutory requirement

Notification is not required.

School exclusion: exclude until the day after appropriate treatment has commenced.

Infectious agent

The *Sarcoptes scabiei* mite is a tiny eight legged creature barely visible to the naked eye. Females are 0.3 to 0.4mm long and 0.25 to 0.35 mm wide. Males are less than half the size of the female.

Scabies life cycle

The mite undergoes four stages in its life cycle: egg, larva, nymph and adult. The female mite burrows into the skin, lays eggs, larvae travel to the skin surface where they moult into nymphs and become adult mites. The period from fertilization to adult mite ranges from 10 to 14 days. Female mites live about two months, laying three eggs a day and travel up to three centimetres a minute.

Identification

Clinical features

Scabies is a highly contagious parasitic skin infestation characterised by thin, slightly elevated, wavy grey-white burrows that contain the mites and eggs. Multiple papules and vesicles soon appear.

The most common sites for burrows are between the fingers and toes, anterior surfaces of the wrists and elbows, axillae, lower abdomen, beneath female breasts and genitalia. The face, head, palms and soles are seldom involved in adults but in infants any area of skin may be infected.

Immunosuppressed people, those living in institutions and the elderly may also show a clinical pattern of infestation similar to that in infants.

Itching varies from person to person but may be severe. It tends to be more marked at night or after a hot bath. Scratching may lead to secondary bacterial infections.

Crusted (Norwegian) scabies

This is a particularly virulent infestation that can occur in the elderly, debilitated or immunosuppressed patients including those with HIV infection. These patients are highly infective and difficult to treat. Large areas of the body may appear scaly and crusted with thousands of mites and eggs. Treatment applied directly to the skin such as creams and lotions may not penetrate the crusted thickened skin and result in treatment failure.

Crusted scabies may be misdiagnosed as psoriasis or eczema.

Method of diagnosis

Diagnosis is commonly made clinically by examining the burrows or rash. The diagnosis may be confirmed by scraping the burrows with a needle or scalpel blade and identifying the mites or eggs under a microscope. A negative result on skin scraping is not always conclusive as the infested person may have few mites (on average 10 to 15) and these can easily be missed on skin scraping.

Incubation period

It may take two to six weeks before itching occurs in a person not previously exposed to scabies.

Symptoms develop much more quickly if a person is re-exposed, often within one to four days.

The incubation period may be shorter if infestation is acquired from a person with crusted (Norwegian) scabies. In this case it is between 10 to 14 days.

Public health significance and occurrence

Scabies occurs worldwide regardless of age, sex, race, socio-economic status or standards of personal hygiene.

Cyclical epidemics occur at intervals of 10 to 15 years.

Outbreaks may occur in childcare centres and kindergartens, and are frequently reported in nursing homes and institutions. Scabies is more likely to spread in situations of overcrowding.

Reservoir

Humans are the primary reservoir. Other species of mite from animals or birds can also live on humans but do not reproduce in the skin.

Mode of transmission

Scabies is transmitted by:

- skin contact with an infected person
- contact with towels, bedclothes and under-garments if these have been contaminated by infested persons within the last four to five days.

The mites cannot jump or fly. Adult scabies mites may survive off the skin for up to 48 hours in room conditions.

Period of communicability

Scabies is communicable until mites and eggs are destroyed by treatment, usually two courses one week apart. Itching may persist for two or more weeks after successful eradication of the mite.

Susceptibility and resistance

Fewer mites succeed in establishing themselves in persons previously infested than in those with no prior exposure. Diminished resistance to infestation is also suggested by the observation that immunologically compromised persons are most susceptible to severe infestations.

Control measures

Preventive measures

Educate the public about the mode of spread, early diagnosis and treatment, and promote good personal hygiene.

Control of case

For simple scabies the usual treatment is permethrin applied topically to the whole body including face and hair (avoid eyes and mucous membranes) and left overnight, or benzyl benzoate 25% emulsion applied topically, including face and hair (avoid eyes and mucous membranes) and left for 24 hours.

For children less than two months of age sulfur 5% cream or crotamiton 10% cream are alternatives (see the current edition of *Therapeutic guidelines: antibiotic*).

For crusted (Norwegian) scabies, the addition of oral ivermectin may also be considered. Seek specialist infectious disease or dermatological advice.

For moderate and severe infections, repeat scabicide treatment in 14 days.

Infested persons should be excluded from school or workplace until the day following the first application of appropriate treatment.

For hospitalised patients or patients in nursing homes contact isolation should be used until appropriate treatment has commenced. In order to prevent nosocomial infection, affected staff should be excluded until appropriate treatment has commenced.

Control of contacts

Investigate contacts and source of infestation.

Treat all household contacts, sexual contacts, and those considered 'at risk' by virtue of close contact in nursing homes and institutions simultaneously.

Control of environment

Clothing, towels and bedclothes used by the infested person in the 48 hours prior to treatment should be laundered using the hot cycle or dry cleaned. Alternatively, items may be placed in a plastic bag and sealed for one week before laundering as mite cannot survive lengthy periods off the human body.

Outbreak measures

Special settings

School and childcare facilities

Exclude the case until the day after appropriate treatment has been given.

Advise staff and parents of other children who may have had direct contact with the infested person and may require treatment. Treat all those who have had close skin to skin contact with the case, this includes family members, playmates and staff. Treatment should occur simultaneously to reduce the risk of reinfestation. Generally, prolonged close contact is required for transmission.

Nursing homes, aged care and other residential facilities

See below, Guide to scabies management in residential care facilities.

Additional sources of information

- Centres for Disease Control and Prevention, *Scabies fact sheet*, http://www.cdc.gov/ncidod
- Centres for Disease Control and Prevention 1988, 'Epidemiologic notes and reports scabies in health-care facilities – Iowa', *MMR Weekly*, vol. 37, no. 11, pp. 178–9.
- Degelau J 1992, 'Scabies in long-term care facilities', *Infection Control and Hospital Epidemiology*, vol. 13, no. 7. pp 421–425.
- Lemmon, J 1998, 'Scabies outbreak among nursing staff', *1998 Nursing monograph*, St Vincents Hospital & Sacred Heart Hospice NSW, http://www.clininfo.health.nsw.gov.au

Scabies information sheet

What is scabies?

Scabies is an infestation caused by the microscopic mite Sarcoptes scabiei. It is found worldwide and affects people of all races and social classes. Scabies spreads rapidly in conditions where there is frequent skin-to-skin contact between people, such as aged care facilities, childcare centres and residential facilities.

The female mite, which is only a few millimetres long, burrows into the top layer of the skin where she lays her eggs. The eggs hatch into larvae after 10 to 14 days and travel back up to the surface of the skin. Female mites live for about two months, laying three eggs a day and travel up to three centimetres a minute.

What are the symptoms of scabies?

The main symptoms are:

- pimple-like irritations, burrows or rash of the skin, especially the webbing between the fingers; the skin folds on the wrist, elbow, or knee; the penis, the breast, or shoulder blades.
- intense itching, especially at night and over most of the body.
- sores on the body caused by scratching. These sores can sometimes become infected by bacteria.

How do you get scabies?

By direct, prolonged, skin-to-skin contact with a person already infested with scabies. Contact must be prolonged (a quick handshake or hug will usually not spread infestation). Infestation is easily spread to sexual partners and household members. Infestation may also occur by sharing clothing, towels, and bedding. Anyone can get scabies regardless of age, sex, race or standards of personal hygiene.

Did my pet spread scabies to me?

No. Pets become infested with a different kind of scabies mite. If your pet is infested with scabies, (also called mange) and they have close contact with you, the mite can get under your skin and cause itching and skin irritation. However, the mite dies in a couple of days and does not reproduce. The mites may cause you to itch for several days but you do not need to be treated with special medication to kill the mites. Until your pet is successfully treated, mites can continue to burrow into your skin and cause you to have symptoms.

Who is at risk for severe infestation?

People with weakened immune systems and the elderly are at risk for a more severe form of scabies, called crusted or Norwegian scabies.

How long does it take until symptoms start?

It may take 4 to 6 weeks for symptoms to develop in people who haven't had scabies before. People who have had scabies before usually develop symptoms much more quickly if they are exposed again, usually within one to four days.

How long are people with scabies infectious to others?

People with scabies can pass on the scabies mite until the day after they have commenced their treatment for scabies. The scabies mite can live for two to three days on the clothes, bed linen and other personal items of people who have scabies.

How is scabies diagnosed?

Diagnosis is commonly made by examining the characteristic burrows or rash. The diagnosis may be confirmed by scraping the burrows with a needle or scalpel blade and identifying the mites or eggs under a microscope. A negative result on skin scraping is not always conclusive as the infested person may have few mites (on average 10 to 15) and these can easily be missed on skin scraping.

Can scabies be treated?

Yes. A number of effective anti-scabies lotions or creams are available from your local pharmacist. A prescription from your doctor is not required.

Recommended treatments include permethrin preparations (e.g. Lyclear cream or Quellada lotion) or benzyl benzoate 25% preparations (e.g. Ascabiol, Benzemul 25%).

Note that:

- Lyclear cream or Quellada lotion should not be used during pregnancy, lactation, for children less than two or in those with extensive dermatitis
- for children under 2 months of age sulfur 5% cream or crotamiton 10% cream (e.g. Eurax) are alternatives
- Ascabiol and Benzemul 25% preparations should be diluted for children less than 2 years of age (dilute with 3 parts water) and for children 2 to 12 years of age and adults with sensitive skin (dilute with equal parts water)
- the anti-scabies preparations should be used according to the manufacturer's directions
- ensure that all household members are treated simultaneously.

The lotions or creams are applied to the whole body from the neck to the toes. The treatment may also need to be applied to the face and scalp if these areas are clearly involved. Avoid contact with the eyes, nose and mouth.

People will no longer be infectious within 24 hours of treatment, but it can take up to two months until the skin lesions and itch to disappear completely.

A repeat treatment may be advised 14 days after the first treatment, particularly for moderate to severe infestations.

Antihistamines, calamine lotion and Eurax are sometimes useful to counteract itchiness. Antibiotics may be needed if there is secondary bacterial infection from scratching.

What else should I do to stop the spread of scabies?

Preventing the spread of scabies requires:

- maintaining good personal hygiene
- not sharing clothes, towels or bed linen with others
- excluding affected children from school and child care centres until treatment has commenced
- limiting close physical contact with others until appropriate treatment has commenced.

As the scabies mite can live on the bed linen, clothes, towels and other personal used by the person with scabies prior to starting their treatment, these items should be machine washed in hot water. Blankets can be dry cleaned or placed in a tumble dryer on a hot setting for half an hour. Alternatively, scabies mites can be killed by sealing these items in a plastic bag for one week before laundering, as the mite cannot survive lengthy periods off the human body.

Further information

- Your local doctor
- Better Health Channel, www.betterhealth.vic.gov.au
- Victorian Department of Human Services, 1300 651 160

Victoria
The Place To Be

Guide to scabies management in residential care facilities

Contact precautions should be employed for suspected cases until the diagnosis is excluded by skin scraping, and, if confirmed, until the day after appropriate therapy has been commenced. As skin scraping is not always positive in true cases, therapy may need to be commenced if the clinical suspicion remains high.

Treating cases

Residents and/or staff should be treated with Permethrin 5% cream (e.g. Lyclear, Quellada) topically, to the whole body including face, hair, behind ears and fingernails (avoid eyes and mucous membranes), leave 8 to 12 hours (e.g. overnight) or Benzyl benzoate 25% emulsion (e.g. Ascabiol, Benzemul 25%) topically, to the whole body including face, hair, behind ears and fingernails (avoid eyes and mucous membranes), leave for 24 hours. For adults with sensitive skin, dilute with equal parts water. Consult the current version of Therapeutic guidelines: antibiotic (Therapeutic Guidelines Limited).

A repeat treatment is advised 14 days after the first treatment, particularly for moderate to severe infestations.

Occasionally large nodular lesions (scabies nodules) can develop in the elderly. These can remain itchy for several months after successful treatment. In these circumstances the use of topical steroids (e.g. Betnovate cream, Elecon cream) may be necessary.

When crusted (Norwegian) scabies is suspected, a single oral dose of ivermectin is often prescribed in addition to the scabicide. Specialist infectious disease or dermatological advice should be sought.

Treating contacts

Close contacts without symptoms who have had recent close contact with cases should be advised of their possible exposure to scabies and be treated simultaneously as above, but with no repeat treatment required. Close contacts include:

- residents with recent close contact, including those who share the same room
- family members and regular visitors with recent close contact with the case
- staff members with recent close contact, including staff handling laundry items.

Close contacts with symptoms consistent with scabies, such as itch or rash, should be treated as if they were a case (see Treating cases, above).

Any agency staff should be notified as they will also need treatment, especially as many attend different health care facilities. If necessary notify the Agency in order to trace staff.

Everyone identified as needing treatment should ideally be treated within the same 24 to 48 hour period. Itch may persist for over 2 weeks after successful treatment and this does not signify treatment failure.

Scabies infection control measures

Linen and bed linen should be hot washed while the treatment application is on and linen used during the application period hot washed after the treatment is washed off.

Clothes and towels used by the affected persons in the 48 hours prior to treatment should be machine washed in hot water. Linen, blankets and clothing can also be dry cleaned or placed in a tumble dryer on a hot setting for half an hour, or sealed in a plastic bag for one week before laundering.

Severe acute respiratory syndrome (SARS)

Victorian statutory requirement

SARS – CoV infection (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Infectious agent

SARS-associated coronavirus (SARS-CoV).

Identification

Clinical features

Severe Acute Respiratory Syndrome (SARS) is a recently recognised lower respiratory tract infection. In the first week of illness the patient develops influenza-like symptoms, which include fever, malaise, myalgia, headache, and rigors. No individual symptom or cluster of symptoms has proven specific, however a history of fever is the one most frequently reported.

The patient progresses to develop a cough (initially dry), dyspnoea and often diarrhoea (large volume and watery) usually in the second week of illness, although these features may occur earlier.

Severe cases progress to a rapidly increasing respiratory distress and oxygen desaturation of which approximately 20% require intensive care.

Upper respiratory symptoms such as rhinorrhea and sore throat may occur but are uncommon.

Method of diagnosis and case definition

Any specific testing for SARS should only be performed after consultation with the Communicable Diseases Section of the Department of Human Services (DHS), (see *Guidance for recognition, investigation and infection control of SARS and avian influenza*, http://www.health.gov.au/
At that time the status of any outbreak can be ascertained, the exposure and epidemiological links clarified, the case may be notified, appropriate infection control processes confirmed, and suitable patient transfer arranged.

The testing algorithm for SARS is heavily dependent upon the prevalence of the disease worldwide and locally, and this can be found on Department of Health and Ageing web site http://www.health.gov.au

The microbiological investigation of a possible SARS infected patient will include the concurrent testing for other more common and likely respiratory pathogens through normal means (sputum, blood, nasal swabs, urine) as well as specific tests aimed to detect SARS-CoV.

The samples to be collected for SARS CoV specifically include:

- a left and right nasal swab and a posterior pharyngeal wall swab all placed into the same viral transport medium. These can have PCR testing for many different respiratory viruses (respiratory multiplex) as well as for SARS-CoV if that is considered appropriate. An alternative to this is a nasopharyngeal aspirate
- stool samples if diarrhoea is present
- serum for antibody titres and, where appropriate, convalescent serum for parallel testing.

Testing is only performed at the Victorian Infectious Diseases Reference Laboratory (VIDRL) and all tests should be clearly labelled 'For urgent SARS testing at VIDRL'.

Suspect case

A person presenting after 1 November 2002 with history of:

- high fever (>38°C) AND
- cough or breathing difficulty AND
- one or more of the following exposures during the 10 days prior to onset of symptoms:
  - close contact with a person who is a suspect or probable case of SARS
  - history of travel, to an area with recent local transmission of SARS (http://www.health.gov.au)
  - residing in an area with recent local transmission of SARS

OR

A person with an unexplained acute respiratory illness resulting in death after 1 November 2002, but on whom no autopsy has been performed AND one or more of the following exposures during to 10 days prior to onset of symptoms:

- close contact with a person who is a suspect or probable case of SARS
- history of travel to an area with recent local transmission of SARS
- residing in an area with recent local transmission of SARS

Close contact: having cared for, lived with, or had direct contact with respiratory secretions or body fluids of a suspect or probable case of SARS.

Probable case

A suspect case with

- radiographic evidence of infiltrates consistent with pneumonia or respiratory distress syndrome (RDS) on chest X-ray (CXR)

OR

A suspect case with

- autopsy findings consistent with the pathology of RDS without an identifiable cause.

Exclusion criteria

A probable or suspect case should be excluded if:

1. No convincing possibility of exposure

Visited a SARS affected area, but was in transit for less than 8 hours (in total, if multiple stopovers) and remained within the airport.

2. An alternative diagnosis is made

e.g. a clinical diagnosis of probable bacterial pneumonia, which could be defined by X-ray findings of lobar consolidation and clinical response to antibiotics. A case initially classified as suspect or probable, for whom an alternative diagnosis can fully explain the illness, should be discarded after carefully considering the possibility of co-infection.

A suspect case should be excluded if they have had a mild self limiting illness however, persons are not to be downgraded should signs of clinical illness remain.

Laboratory confirmed SARS

A person with symptoms and signs that are clinically suggestive of SARS

AND

With positive laboratory findings for SARS-CoV, based on one or more of the following diagnostic criteria:

a) PCR positive for SARS-CoV

PCR positive using a validated method from:

- at least two different clinical specimens (e.g. nasopharyngeal and stool) OR
- the same clinical specimen collected on two or more occasions during the course of the illness (e.g. sequential nasopharyngeal aspirates) OR
- two different assays or repeat PCR using a new RNA extract from the original clinical sample on each occasion of testing.

b) Seroconversion by ELISA or IFA

- Negative antibody test on acute serum followed by positive antibody test on convalescent phase serum tested in parallel OR
- Fourfold or greater rise in antibody titre between acute and convalescent phase sera tested in parallel.

c) Virus isolation

- Isolation in cell culture of SARS-CoV from any specimen AND
- PCR confirmation using a validated method.

Reclassification of cases

A suspect case who, after investigation, fulfils the probable case definition should be reclassified as 'probable'. A suspect case with a normal CXR should be treated, as deemed appropriate, and monitored for 7 days. Those cases in whom recovery is inadequate should be re-evaluated by CXR.

A suspect case who dies, on whom no autopsy is conducted, should remain classified as 'suspect'. However, if this case is identified as being part of a chain of transmission of SARS, the case should be reclassified as 'probable'. If an autopsy is conducted and no pathological evidence of RDS is found, the case should be 'discarded'.

Maintaining vigilance and SARS alert clusters

If SARS does reemerge, it is unlikely but not impossible, that the first place it is recognised will be Australia. The most likely scenario is that this will occur in another country or countries (particularly Southern China, the source of the original outbreak), providing time for Australia to institute targeted surveillance and investigation of illness in travelers from defined outbreak areas, as was undertaken in the initial outbreak period.

Although both WHO and Australian health authorities regard Australia as a low likelihood country to first recognise a new SARS outbreak, a cautious approach is being taken. Maintaining vigilance for SARS (www.health.gov.au/sars) is a surveillance protocol that seeks to ensure that Australian health authorities will detect any new SARS outbreak by the detection of 'alert' clusters of cases. These are clusters of apparent hospital-acquired cases in staff, patients, and visitors to the same health-care facility, and that meet the new WHO post outbreak clinical case definition for SARS.

Definition of a SARS alert

a) Two or more health care workers in the same health care facility fulfilling the clinical case definition of SARS (see below) and with onset of illness in the same ten day period.

OR

b) Apparent hospital-acquired illness in three or more persons (health care workers and/or other hospital staff and/or patients and/or visitors) in the same health-care facility fulfilling the clinical case definition of SARS (see below) and with onset of illness in the same 10-day period.

Clinical case definition of SARS alert cases (post-outbreak period)

The following clinical case definition has been developed for public health purposes.

A person with a history of:

a) Fever (≥ 38°C)

AND

b) One or more symptoms of lower respiratory tract illness (cough, difficulty breathing,

shortness of breath)

AND

c) Radiographic evidence of lung infiltrates consistent with pneumonia or acute respiratory distress syndrome (ARDS) OR autopsy findings consistent with the pathology of pneumonia or RDS without an identifiable cause.

AND

d) No alternative diagnosis can fully explain the illness.

If 'alert' clusters are detected from one institution then those cases should be urgently isolated and the situation immediately discussed with an infectious disease physician AND with the Communicable Disease Section, DHS. This is likely to lead to testing for SARS-coronavirus and the adoption of enhanced infection control measures.

Incubation period

The mean incubation period is five days with the range of 2–10 days although there are infrequent isolated reports of longer incubation periods.

Public health significance and occurrence

SARS came to the world's attention in early 2003 when WHO declared a global public health alert in response to a severe respiratory illness due to an unidentified communicable pathogen.

The pathogen emerged out of Southern China creating a local outbreak of atypical pneumonia and subsequent infection of international travellers resulted in the importation of possible SARS cases to 29 other countries around the world. Hong Kong, Hanoi, Singapore and Toronto received such infected travellers early in the outbreak and further transmission within these cities resulted in local outbreaks, affecting many hundreds of people.

The overall case fatality rate was approximately 10% and was highest (>50%) in those over 60 years of age. A characteristic feature of the SARS outbreak was its unprecedented degree of nosocomial spread, which resulted in 21% of all cases involving health care workers. This has resulted in a requirement for heath care staff to adopt a new standard of infection control and personal protection.

WHO declared the outbreak interrupted on 5 July 2003 at which time there were more than 8400 cases and approximately 900 deaths. Mainland China reported over 5300 cases and 349 deaths. Australia had a single confirmed case of SARS who had visited NSW prior to the global alert and was detected in retrospect by authorities in her home country. She did not transmit the illness to any of her close contacts.

Five international flights were associated with the transmission of SARS however there has been no evidence of confirmed transmission on any flights after WHO recommended control measures, which included border exit screening.

Reservoir

There has been much interest in determining the source of this new virus, with particular focus on the animal species involved and animal husbandry methods seen in Southern China. Early investigations have pointed in the direction of certain animal species (palm civet, racoon dog) however these are not conclusive and more work in this area needs to be completed.

Mode of transmission

During the SARS outbreak, the predominant mode of transmission of the SARS CoV appeared to be by direct mucus membrane contact with respiratory droplets from either infected persons or fomites.

The evidence to date suggests that spread is predominantly through direct contact or exposure to larger virus-laden droplets that are thought to travel only one to two metres, than by lighter airborne particles. It has been postulated that these lighter and smaller aerosols may have been generated by procedures such as nebulisers or intubations, resulting in episodes where significant amplification of transmission was observed.

Infective stool may also pose a transmission risk but the risks associated with this are not yet clear.

New cases occurred primarily in persons with close contact to those very ill with SARS, which was seen in health care and household settings. Less frequently, transmission occurred to casual and social contacts after intense exposure to a case of SARS (in workplaces, airplanes or taxis).

Maximum excretion of the virus from the respiratory tract seems to occur near day 10 of illness and then declines. The efficiency of transmission appears to be greatest following exposure to severely ill patients or those experiencing rapid clinical deterioration, both of which usually occur during the second week of illness.

On reviewing cases of SARS it was found that when symptomatic cases were isolated within 5 days following onset of illness, few cases of secondary transmission occurred.

Period of communicability

SARS-CoV is not thought to be transmissible during the asymptomatic incubation period and there has been no evidence that the virus has been spread ten days after fever has resolved.

Susceptibility and resistance

The elderly are more prone to severe disease and pose a particular challenge in the recognition of SARS as they may present with an afebrile illness or with a concurrent bacterial sepsis or pneumonia.

In the setting of a SARS outbreak the diagnosis should be considered for almost any change in health status, even in the absence of typical clinical features of SARS-CoV disease, when such patients have epidemiologic risk factors for SARS-CoV disease (e.g. close contact with someone suspected to have SARS-CoV disease or exposure to a location [domestic or international] with documented or suspected recent transmission of SARS-CoV).

During the 2003 outbreaks, infants and children accounted for only a small percentage of patients and had much milder disease with better outcomes. There have been two reported cases of transmission from children to adults and no reports of transmission from children to other children. Three separate epidemiological investigations have found no evidence of SARS transmission in schools. Furthermore, no evidence of SARS has been found in infants of mothers who were infected during pregnancy. Further investigation is required to determine whether children may have asymptomatic or mild infections.

Control measures

Preventive measures

There are no vaccines available for SARS-CoV.

As a result of the global outbreak of SARS there has been resurgence in interest and prominence of respiratory hygiene and cough etiquette as an attempt to reduce transmission of all forms of respiratory pathogens, including SARS-CoV.

This includes encouraging all persons with signs and symptoms of a respiratory infection to:

- cover the nose and mouth when coughing or sneezing
- use tissues to contain respiratory secretions
- dispose of tissues in the nearest waste receptacle after use
- wash hands after contact with respiratory secretions and contaminated objects and materials.

Health care facilities should ensure the availability of materials for adhering to respiratory hygiene/cough etiquette in waiting areas for patients and visitors:

- provide tissues and no-touch receptacles for used tissue disposal
- provide conveniently located dispensers for alcohol-based hand rub
- provide soap and disposable towels/hand driers for hand washing where sinks are available.

During periods of increased respiratory infection in the community, it may be possible for healthcare facilities to offer surgical masks to persons who are coughing and encourage coughing persons to sit at least three feet away from others in waiting areas.

Healthcare workers should practice droplet precautions, in addition to standard precautions, when examining a patient with symptoms of a respiratory infection.

=Once there exists an index of suspicion of SARS then the appropriate infection control measures need to be activated and suitable PPE worn, (see http://www.icg.health.gov.au). These will dependent on the specific facility involved and the resources available at the time. They include:

- use of standard precautions (ie hand hygiene) and contact and droplet precautions (ie use of long-sleeved gowns, gloves and protective eyewear for contact with patient or environment)
- use of airborne precautions that include the use of a P2 (N95 equivalent) mask (respirator) for all persons entering the room and where available, a negative pressure respiratory isolation room (with en-suite)
- restriction of patient movement (and fitting of a surgical mask if they must leave their room)
- avoiding the use of nebulisers, chest physiotherapy, bronchoscopy, gastroscopy or any intervention that may disrupt the respiratory tract
- placing surgical masks over nasal oxygen prongs.

It will become increasingly important for clinicians to elicit epidemiological information from their patients as part of normal history taking. Travel history, recent attendance to hospitals or exposure to others who are ill, may assist in the refinement of a patient's differential diagnosis and associated risk.

The following points may become appropriate to consider in the primary care setting as a means of managing the issues of SARS:

- signage at the reception desk may advise potential cases to report their concerns to the practice as early as possible
- any case that could be reasonably regarded as possibly SARS should be discretely offered a mask and diverted out of the waiting room and into a single room (e.g. returned travellers from affected region with SARS like symptoms)
- if seen in the practice the clinician should close the door, open a window, turn off the air-conditioning, put on a mask (N2 if possible), gown, gloves and eye protection
- wash hands after consultation
- do not self contaminate by touching ones own mucus membranes with contaminated hands
- make an assessment and call the Communicable Diseases Section, DHS, for an update of the SARS situation and to develop a suitable management strategy
- where possible any cases of concern should be seen at home with the appropriate PPE.

For further details see the *Australian interim control guidelines*, http://www.health.gov.au

Control of case

Suspected cases will be managed on their clinical merits with home care regarded as a suitable option if the domestic situation, including its suitability in terms of infection control, is judged to be adequate. In such circumstances, cases will be advised to voluntarily restrict their movements.

Probable and confirmed cases will require hospitalisation and isolation in a suitable health facility, which will be determined by the Communicable Diseases Section DHS in consultation with the treating clinician. The receiving hospital will activate its SARS protocol to suitably manage such a patient.

All suspected, probable and confirmed cases will be excluded from school and work until clearance is obtained from DHS.

There are no specific treatment recommendations for SARS. The application of intensive supportive therapy and empirical antimicrobial therapy, to cover other infective agents, is the usual approach. Antiviral and pulse steroid therapy have been used in the past, in different countries with varying degree of success.

Discontinuation of SARS isolation precautions

SARS isolation precautions should be discontinued only after consultation with the local public health authorities and the evaluating clinician.

Control of contacts

Only people who have been close to an unwell person with SARS are at any significant risk of acquiring infection. For this reason only close contacts are sought to implement public health contact tracing measures and control disease spread. A close contact is a person who has lived, worked or had other dealings with a SARS case that have caused them to be within a meter of the case or who has had direct contact with respiratory secretions from a case while not wearing personal protective equipment.

Contact tracing will be undertaken for those *close contacts of probable cases* of SARS who were exposed after the patient became symptomatic (see details in the *Recommendations for tracing & managing contacts of SARS cases* http://www.health.gov.au
Contact tracing will not be undertaken for suspected cases of SARS while SARS has not been locally transmitted in Australia.

The aims of contact tracing is to find, provide information to and manage persons those who may have been exposed to the SARS CoV and who may be incubating or have early signs of the disease. Management of these contacts depends on who they were exposed to and the circumstances surrounding the exposure.

Well *close contacts* will be placed under either passive or active surveillance, whilst all unwell close contacts of probable cases will be placed under active surveillance and isolated in an appropriate setting.

It should be remembered that one of the most important available measures to prevent the spread of SARS CoV is the application of respiratory precautions and scrupulous hand washing. Contacts should be advised of such and also for the need to seek immediate medical attention if they develop the initial symptoms of SARS. Daily temperature monitoring for ten days after a break in exposure from the SARS case is advisable.

Close contacts of cases or returned travellers from regions of SARS outbreak as defined by DoHA will be allowed to attend school on the provision that they remain completely asymptomatic. Such persons should measure their temperatures daily to ensure that fever is not present during the ten days incubation period.

Cleaning and disinfection

Early studies of SARS-CoV show that if uninterrupted by cleaning or disinfectants it can survive on surfaces in the environment, such as on stainless steel benches, plastic, wood or cotton, for between 12 and 72 hours. However, the virus is not difficult to kill. It is important to clean surfaces with detergent and water and then to disinfect them. Remember that disinfectants need the appropriate time at the appropriate concentration to be effective.

The different methods available for disinfecting include:

Heat (56 degrees Celsius) is very effective, so dishes, linen and other washable items can be disinfected by washing in hot water and detergent.

Alcohol is effective. Tests show that 75% ethanol kills the virus at room temperature in less than 5 minutes. Slightly lower concentrations of alcohol would take a slightly longer time. Alcohol can be found in alcohol rubs (for hands), alcohol impregnated wipes and swabs such as used to disinfect skin, and methylated spirits.

Acetone is effective. 10% acetone will kill the virus in less than 5 minutes.

Phenol (2%) is effective and may be found in some hospital grade disinfectants.

Bleach has not yet been tested against the SARS coronavirus. However bleach is an effective disinfectant for many other viruses and is likely to be effective. Surfaces to be disinfected with bleach must first be cleaned with detergent and water. An appropriate dilution of 1 in 100 of most household bleach provides sodium hypochlorite at 500 ppm.

Additional sources of information

- Australian Government Department of Health and Ageing, http://www.health.gov.au
- Australian Government Department of Health and Ageing 2004, *Severe acute respiratory syndrome (SARS) case definitions*, http://www.health.gov.au
- Centres for Disease Control and Prevention, Atlanta USA see http://www.cdc.gov/ncidod/sars/
- Centres for Disease Control and Prevention 2004, *Clinical guidance on the identification and evaluation of possible SARS-CoV disease among persons presenting with community-acquired illness* Version 2, http://www.cdc.gov/ncidod
- Centres for Disease Control and Prevention 2004, *In the absence of SARS-CoV transmission worldwide: guidance for surveillance, clinical and laboratory evaluation, and reporting*, Version 2, http://www.cdc.gov/ncidod
- Communicable Diseases Network Australia 2003, *Recommendations for tracing and managing contacts of SARS cases*, Australian Government Department of Health and Ageing, http://www.health.gov.au
- World Health Organization http://www.who.int/csr
- World Health Organization 2003, *Alert, verification and public health management of SARS in the post-outbreak period*, http://www.who.int/csr
- World Health Organization 2003, *Consensus document on the epidemiology of severe acute respiratory syndrome (SARS)*, http://www.who.int/csr

Shigellosis

Victorian statutory requirement

Shigellosis (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude until after diarrhoea has ceased.

Infectious agent

The genus *Shigella* consists of four species:

- Group A: *Sh. dysenteriae*
- Group B: *Sh. flexneri*
- Group C: *Sh. boydii*
- Group D: *Sh. sonnei*

Groups A, B and C are further divided into approximately 40 serotypes, designated by numbers.

Identification

Clinical features

Shigellosis is characterised by an acute onset of diarrhoea, fever, nausea, vomiting and abdominal cramps. Typically the stools contain blood, mucus and pus, although some persons will present with watery diarrhoea. Complications include toxic megacolon and reactive arthritis. Rarely haemolytic uraemic syndrome can occur. The infectious dose required to produce disease is low and may be as few as ten organisms.

Illness is usually self-limited and lasts from several days to weeks with an average of four to seven days. The severity of infection depends on host factors such as age and nutritional status and the serotype. Infections with *Sh. sonnei* usually result in a short clinical course and low case fatality rate. In contrast, *Sh. dysenteriae* is often associated with serious disease and a high case fatality rate.

Asymptomatic infections occur and carriage may persist for months.

Method of diagnosis

Diagnosis is made by isolation of *Shigella* spp. from a clinical specimen.

Incubation period

The incubation period depends on the serotype. It varies from twelve hours to seven days but is usually one to three days.

Public health significance and occurrence

Shigella infection occurs worldwide however the incidence of specific serotypes varies by country. *Sh. sonnei* is the most common type reported in Victoria and Australia. *Sh. dysenteriae* and *Sh. flexneri* are usually acquired overseas and are often resistant to multiple antibiotics. In Victoria outbreaks have occurred in child care centres and amongst men who have sex with men.

Two-thirds of the cases and most of the deaths worldwide are in children less than ten years. The disease is rare in infants under six months of age, particularly those who are breastfed.

Secondary attack rates in households may be as high as 40%.

Reservoir

Humans.

Mode of transmission

Faecal-oral transmission is the most important mode of transmission of Shigella however infection may be spread via contaminated food, water, milk or by flies.

Period of communicability

Shigella is communicable during the acute phase and while the infectious agent is present in faeces which is usually no longer than four weeks. Asymptomatic carriage and excretion may persist for months.

Susceptibility and resistance

Everyone is susceptible to infection, with infection following ingestion of a small number of organisms. In endemic areas the disease is usually more severe in young children. The risk of infection is increased in men who have sex with men, those with immune deficiency disorders, by attendance at child care or contact with a child in child care, and in international travellers who do not take adequate food and water safety precautions.

Control measures

Preventive measures

Good personal hygiene is the single most important preventive measure. Frequent and thorough hand washing is important before eating and food handling and after toilet use, especially in young children who may not be completely toilet trained.

Educate travellers on the need for safe food and water consumption.

Control of case

Treatment is usually supportive for mild illnesses. Antibiotics may shorten the duration and severity of illness however their use should be based on the serotype, severity of illness and host characteristics, for example if they are a child in child care, food handler or suffer chronic illness. Multi-drug resistance is common, particularly for overseas-acquired strains. The choice of antibiotic

should be based on the antibiogram of the serotype. Anti-motility drugs are thought to increase the risk of prolonged carriage.

Cases should be educated on the importance of personal hygiene, particularly after using the toilet and before and after food handling.

Food handlers should be excluded from work until two negative stools have been obtained, or until at least 48 hours after the diarrhoea has ceased and rigid personal hygiene measures can be assured.

Cases in institutions should be separated from non-infected residents if possible.

Control of contacts

The diagnosis should be considered in symptomatic contacts however stool cultures may be confined to food handlers and those in situations where the spread of infection is particularly likely (child care centres, hospitals, institutions).

Symptomatic contacts of shigellosis patients should be excluded from food handling and the care of children or patients until investigated.

Control of environment

Remove contaminated food and/or water sources. Strict attention should be paid to environmental hygiene in child care centres, institutions and food premises.

Outbreak measures

Two or more related cases should be considered indicative of an outbreak and require investigation. These cases should be reported immediately to the Department of Human Services. Attempt to determine a common source of infection and identify those at risk of infection.

Refer to the *Guidelines for the investigation of gastrointestinal illness* for further advice and management of outbreaks.

Smallpox (variola)

Victorian statutory requirement

Smallpox (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Smallpox is included on the Commonwealth Quarantine List and all cases will need to be notified immediately to the State Chief Quarantine Officer.

Smallpox is subject to Australian quarantine.

Infectious agent

Variola virus is a DNA virus of the genus *Orthopoxvirus*.

The virus used in the live smallpox vaccine is known as the vaccinia virus and is also a member of the genus Orthopoxvirus.

Identification

Clinical features

Smallpox is a severe prostrating illness characterised by fever and a macular, papular, vesicular and pustular rash with an observed mortality rate of 30%. There are three major forms. The most common form described below occurs in 90% of cases. The remaining two are known as haemorrhagic and malignant (flat) variants. These both have significantly higher mortality (greater than 95%) and seem to be related to alterations in immune status.

Common smallpox begins with symptoms of fever (100%), headache (90%), backache (90%), chills (60%), vomiting (50%), malaise, prostration and cough. Less commonly pharyngitis and severe abdominal pain are observed. In pale-skinned patients an erythematous rash sometimes accompanies the prodromal phase. This occurs rarely as a petechial rash. This may be misdiagnosed as meningococcal disease, erythema multiforme or measles.

By the second or third day fever begins to descend from its peak (40.5 to 38.5 C) and the eruptive phase begins with the development of rash lesions. These lesions first appear on the buccal and pharyngeal mucosa and then emerge on the face, forearms and hands. The rash spreads down, and within a day the trunk and lower limbs are involved.

Smallpox produces a single crop of lesions which are distributed in a centrifugal pattern: most profuse on the face, more abundant on the forearms and lower legs than the upper arms and thighs, and often involve palms and soles.

Prominent surfaces and areas most exposed to irritation are more heavily involved by the rash. Protected surfaces such as flexures and depressions (axilla) are usually spared.

The eruptive lesions appear as flat discoloured macules that progress to firm papules on the second day of the rash. These are typically described as 'shotty'. They become clearly identifiable as vesicles on the fourth or fifth day of the rash and progress to pustules on day seven.

Day ten commonly sees the pustules at maximal size and the lesions then commence to flatten. Approximately 14 days after rash onset the pustules begin to dry up and crust. Most pustules begin to scab and separate at day 19. Lesions on the palms and soles separate last and typically leave pitted scars.

A patient is no longer infectious once all the scabs have separated from the skin which is usually three to four weeks after the onset of the rash. Recovery results in the complete clearing of the virus from the body and prolonged immunity.

The major differential diagnosis is chickenpox and disseminated herpes simplex infections.

Smallpox may be complicated by secondary bacterial skin infection, corneal scaring, keratitis, arthritis, osteomyelitis, bronchitis, pneumonitis, pulmonary oedema and encephalitis.

Method of diagnosis

The diagnosis of smallpox will be made on the basis of a consistent clinical presentation combined with the results of electron microscopy and PCR testing which will be performed at the National High Security Quarantine Laboratory at Victorian Infectious Diseases Reference Laboratory in Melbourne.

Incubation period

The incubation period is regarded to be seven to 17 days, with a median of 12 days.

Public health significance and occurrence

In 1980 the World Health Organization (WHO) declared smallpox the first communicable disease ever to be globally eradicated. This was a direct consequence of the Global Smallpox Eradication Program which was achieved by a population based smallpox vaccination strategy.

The virus has been retained legally under strict security in two World Health Organization collaborating centres in the USA and the Russian Federation.

The virus is believed to have been part of the bio-weapons research of certain countries and there have been recent concerns that non-state actors may obtain access to the virus for deliberate release.

A single confirmed case of smallpox would prompt a global public health alert from the World Health Organization and would raise the spectre of an intentional release.

Historically variola major has a significant mortality and it would be reasonable to expect a greater impact upon today's unimmunised and older populations. It is clear that an outbreak would be of extreme public concern requiring action at the highest level of government and involving the mobilisation of significant resources.

Reservoir

Smallpox is a disease only of humans and there are no non-human hosts.

Mode of transmission

The variola virus is most frequently transmitted from an infectious person via direct deposition of large, infective, airborne droplets of saliva onto the nasal, oral or pharyngeal mucosal membranes during close, face to face contact with a susceptible individual.

The generation of infectious fine-particle aerosols provides a possible albeit less common means of smallpox transmission. This may result in the infection of persons involved in non-face to face contact with the case, with the virus carried in aerosols spread by drafts and air-conditioning systems. Such spread is most likely in instances where the case has a significant cough.

Cases may contaminate objects in their environment including their clothing and linen with the large droplets or aerosols during sneezing or coughing and these fomites may serve as a further route of transmission.

Physical contact with a smallpox pustule or crusted scab may also transmit the virus. The virus has been found to survive in scabs for many years, however encased in this form it is not considered to represent a significant infectious risk.

Body fluids are also infectious and care is needed for the disposal of clinical waste.

The variola virus is thought to be unlikely to survive on its own for more than 48 hours when exposed to normal environmental conditions (ambient temperature, usual humidity and sunlight exposure).

During the smallpox era the disease had secondary household or close contact attack rates of up to 80%.

Period of communicability

Patients are not infectious during the asymptomatic incubation period. They become increasingly infectious after onset of fever and this usually results from the release of virus from oropharyngeal lesions.

For the purpose of contact tracing, cases are regarded as infectious 24 hours prior to the recognition of fever, and any contacts identified from this time on need suitable management.

Susceptibility and resistance

Resolved infection confers lifetime immunity.

Pregnant women and those who are immunocompromised are more susceptible to variant forms of smallpox.

It is unclear how long the smallpox vaccine will provide effective immunity but it is unlikely to be more than 10 years. As a result essentially all persons in Victoria and Australia will be regarded as susceptible to smallpox.

Control measures

Preventive measures

The Australian Government Department of Health and Ageing has stockpiled a certain amount of smallpox vaccine which will be accessed under appropriate situations.

In the event of an outbreak, there will be a stepwise process to vaccinate persons who will be required to assist in its containment such as doctors, nurses and ambulance personnel.

All others will be offered vaccination only if they have had contact with a case or form part of a 'ring vaccination' control strategy.

Control of case

Any patient that raises a concern of smallpox must be notified to the Communicable Diseases Section of the Department of Human Services as soon as possible such that a mobile smallpox care team can be dispatched to provide a swift and expert provisional diagnosis, and to implement suitable patient care and public health management.

All such patients (and their possessions) should be placed in the best available form of isolation as soon as possible. They should have limited contact with any persons other than those directly involved in their care, who must wear personal protective equipment. Any air conditioning should be turned off immediately.

All persons in contact with the case or those sharing the same airspace (hospital or practice staff, other patients etc) should be requested to remain in a safe area until the smallpox care team arrives and makes an assessment. They may need to be given access to smallpox immunisation in the short term and their details, including contact numbers, will be essential to collect. This should be commenced as soon as practicable. The smallpox care team will advise about infection control matters including disinfection and provide information to those present.

Cases will be categorised as possible, suspected, probable or confirmed, depending upon the epidemiology, clinical presentation and the results of electron microscopy and PCR testing of vesicular fluid.

All confirmed and probable cases will be managed in the treatment ward of the smallpox care centre where they will receive optimal health care by staff who have been successfully immunised, whilst maintaining appropriate isolation precautions for the community.

Those who meet the possible or suspected criteria will be placed in the observation ward of the smallpox care centre.

Control of contacts

The strategy of ring vaccination will be used in the control of any smallpox outbreak. This means that all contacts of a case will be immunised (category A and B, see below) and as an extra precaution, those persons with ongoing household contact with category A contacts, during the formal monitoring period, will also be offered access to the vaccine.

Category A, primary contacts

These are persons who are likely to be exposed to the virus through large droplets or contaminated fomites. They include:

- Household contacts. All usual residents and any visitors who had spent more that one hour at the address during the infectious period.
- Face to face contacts (within two metres) during the infectious period. This will include work and social settings as well as unvaccinated health care and emergency services personnel.
- Fomite contact. All persons with direct contact with clothing or articles that have been used by infectious cases of smallpox.

Actions required

- Urgent vaccination, preferably before day three but up to day seven.
- Active surveillance for 17 days after the last exposure
  - Daily reporting of the contact by phone to the Department of Human Services will be required
  - Details of oral temperature and presence of constitutional symptoms
  - If there is failure to contact, the Department will actively follow up cases by phone or in person.
- Restriction of movement from seven days after first exposure until 17 days after last exposure.
  - Avoid contact with unvaccinated persons
  - During this time contact must stay away from school and work
  - Remain within local area as defined by the Department
- If symptomatic, category A cases need to stay at home and immediately contact the Department.
  - A category A contact who develops fever will be regarded as a **possible** case and transferred immediately to the observation ward of the smallpox care centre
  - A category A contact who develops fever and rash will be regarded as a **probable** case and transferred immediately to the treatment ward of the smallpox care centre.
- Outside the restricted period category A contacts will need to stay within the local area until their vaccination site is completely healed and their formal monitoring period is over.

Category B, primary contacts
These contacts are less likely to have been exposed to the virus than Category A contacts. They include:

- All persons who have shared a room or other enclosed spaces with the case whilst infective and not meeting the criteria of category A contacts
  - These may include those who have visited the same premises or travelled on the same public transport (trains, planes or buses) or who have shared the same floors of buildings or the same air conditioning space with an infectious case.

Action required for category B contacts

- Vaccinate unless contraindicated
- Commence passive surveillance:
  - Details will be taken by the Department of Human Services and information provided as to nature of smallpox and actions to be taken if symptoms develop (fever, rash or constitutional symptoms).
  - If symptoms develop they must immediately contact the Department and remain at home, avoiding contact with all unvaccinated persons.
  - Surveillance will continue until 17 days after the last exposure to the virus.
- Restricted movement:
  - Category B contacts will not be allowed to travel abroad until their vaccination site is completely healed and their formal surveillance period is over.
  - No other restrictions of activity are required unless the case is unwell.
- If symptomatic they will be admitted to the smallpox care centre:
  - Category B contacts who develop fever will be classified as possible cases and transferred to observation ward of the smallpox care centre.
  - Category B contacts who develop fever and vesicular rash will be classified as probable cases and transferred to the treatment ward.

Secondary contacts
These are persons who will have ongoing household contact with category A contacts during the formal monitoring period. As such they are at risk of exposure to the virus if the primary contact becomes symptomatic. Secondary contacts would be expected to include usual household residence of category A contacts, together with any visitors to the household who expect to spend extended periods of time there, during the formal monitoring period.

Actions required for secondary contacts

- Vaccinate unless contraindicated:
  - If immunisation is contraindicated then the secondary contact will need to avoid contact with the primary contact until the vaccination site is completely healed.
- Passive surveillance and no restriction of movement:
  - There are no monitoring or restriction requirements necessary unless the primary contact becomes symptomatic. If they become confirmed with smallpox, then the secondary contacts will be reclassified as a category A contact and will need to be managed accordingly.

Unimmunised primary contacts
Definition
These are primary contacts (both category A and B) who fail to respond to the vaccine after three days, who are vaccinated later than three days after first exposure to the virus, or who refuse to be vaccinated.

Action

- Limited options are available for the pharmacological management of persons vaccinated late. The smallpox response team's infectious disease specialist may suggest the use of vaccinia immune globulin or cidofovir in very limited circumstances.
- Category A primary contacts who are categorised as unimmunised, will be required to remain in isolation accommodation until the incubation period has elapsed.
- Category B primary contacts will be managed as if they were a category A contact (active surveillance, restricted movement from day seven after first exposure to day 17 after last exposure, see above).

Control of environment

All persons in contact with a case of smallpox must wear the appropriate personal protective equipment (PPE) and in order to limit any further spread, this will be removed and the person required to shower on leaving the infected area. This PPE includes gloves, theatre cottons with head cover, disposable apron, eye protection, foot wear such as overshoes, and a P2 respiratory mask.

Until the smallpox care team arrives, the possibly infected area should be cordoned off and access limited to those already present and those required for urgent medial care. The case of concern should be isolated as best as possible and all others should remain within a safe distance of the cordoned off area. Information and all care should be afforded all persons involved, with particular attention being made to advise that the earliest possible access to the vaccine will provide the best possible outcome if the case in fact proves to be smallpox.

The smallpox care team will advise on suitable decontamination processes and the disposal of possibly infectious materials. This will be in accordance with the *Guidelines for the smallpox outbreak, preparedness, response and management*.

As the virus is transmitted through infectious respiratory droplets and bodily fluids or contaminated clothing, dressings, linen, towels or clinical waste, every effort must be made by relevant staff to limit spread through these routes. Air conditioning must be isolated or turned off and the time documented. Any object that enters the infected area must remain there until disinfected or disposed of appropriately. This includes all linen, dressings and disposable eating utensils and medical notes.

Outbreak measures

Smallpox management will be framed in one of the five Australia response codes detailed below.

Australian response codes for smallpox

- Response code 0: Smallpox remains eradicated – no credible threat of a release
- Response code 1: Imminent threat or a case overseas
- Response code 2: One case or a cluster of related cases in Australia
- Response code 3: Unrelated cases or unrelated clusters occurring in Australia
- Response code 4: Outbreak controlled – no further cases occurring

Emergency plans would be activated in sequence with these codes as outlined in the Australian Government's *Guidelines for the smallpox outbreak, preparedness, response and management*. This would include alerts to the community and health providers, roll out of the smallpox vaccination strategy, mobilisation and augmentation of the smallpox care teams, and commissioning of the smallpox care centre.

Special settings

General hospital wards and their emergency department (ED) may be at increased risk of attending to smallpox cases. In order to limit this, all community concerns regarding smallpox need to be notified to the Department of Human Services immediately. The Department will dispatch a smallpox care team to make an urgent assessment. In this way, cases will be diverted to smallpox care centres without disrupting the working of any hospitals.

However if a case does present to an ED, then activation of the ED infection control procedures should be instituted, such that appropriate action is taken to limit any spread into the broader hospital.

International measures

If there are smallpox cases overseas then the Australian Government may divert all aircraft from that country, to a limited number of airports where screening, immunisation and the appropriate isolation and quarantine measures will be applied as required.

Additional sources of information

- Australian Government Department of Health and Ageing 2004, *Guidelines for smallpox outbreak, preparedness, response and management*.
- Fenner, F 1988, *Smallpox and its eradication*, World Health Organization.

Staphylococcal infections

Victorian statutory requirement

Notification is not required.

School exclusion: for impetigo due to staphylococcal infection exclude until appropriate treatment has commenced.

Infectious agent

There are nineteen species of staphylococci. The most significant human pathogens are *Staphylococcus aureus* and *Staphylococcus epidermidis*. Methicillin resistant *S. aureus* (MRSA) and vancomycin-resistant *S. aureus* (h-VISA, VISA and VRSA) are significant pathogens in hospital-acquired disease. Virulence varies greatly amongst the bacterial strains.

Identification

Clinical features

Staphylococcal infection presents with a variety of different clinical and epidemiological patterns amongst the general community, newborns, hospitalised patients and menstruating women. It may cause:

- purulent skin infections such as a boils, abscesses, styes, impetigo and scalded skin syndrome
- systemic infections such as pneumonia, osteomyelitis or endocarditis
- urinary tract infections due to *S. saprophyticus* in young women or *S. epidermidis* with indwelling catheters
- hospital-acquired (nosocomial) infection of surgical wounds or treatment lines
- food poisoning by releasing toxins into food
- toxic shock syndrome by releasing toxins into the blood stream.

Method of diagnosis

Diagnosis is confirmed by isolation of the organism from relevant specimens. Their antibiotic resistance profile is important in management.

Incubation period

The incubation period is variable and indefinite. It is most commonly four to ten days.

Public health significance and occurrence

Staphylococcal infections are frequent but are usually contained by immune mechanisms to the site of entry. Approximately 20–30% of the population are colonised with S. aureus in the anterior nasal passages. The highest incidence of disease usually occurs in people with poor personal hygiene, overcrowding and in children. However anyone can develop a serious staphylococcal infection including fit young people.

Since the late 1970s MRSA strains have been identified in Victoria as a major cause of nosocomial infections and outbreaks. MRSA accounts for approximately 30–50% of hospital-acquired S. aureus isolated from normally sterile sites. Vancomycin resistant strains have been reported. Health care employees and other carers may develop intermittent colonisation with MRSA. These workers rarely develop infection.

Reservoir

Human carriers are a major source of infection. Staphylococci have prolonged survival in the hospital environment due to resistance to antiseptics and disinfectants.

Mode of transmission

Staphylococci are most often transmitted by direct or indirect contact with a person who has a discharging would, a clinical infection of the respiratory or urinary tract, or one who is colonised with the organism. MRSA can be carried on the hands of healthcare personnel and is a likely mode of transmission between patients and staff. Contaminated surfaces and medical equipment are also possible sources of MRSA.

Period of communicability

Communicability exists as long as purulent lesions continue to drain, or the carrier state persists.

Susceptibility and resistance

People who are most susceptible to infection are the chronically ill and newborns.

Mechanisms of immunity are not well understood. An experimental vaccine with a short duration of immunity has been developed to assist patients with end-stage renal disease.

Resistance to penicillin-related antibiotics in the hospital setting is common and includes MRSA. Two specific types of vancomycin antimicrobial resistant *S. aureus* called VISA and VRSA have recently emerged.

Control measures

Preventive measures

General measures:

- maintain good hygiene through public education in relation to hand washing, food preparation and avoiding sharing toilet articles
- cover purulent lesions with a waterproof dressing.

In the health care setting:

- educate hospital staff regarding the importance of hand washing
- use common narrow spectrum antibiotics where possible.

Control of case

Advise isolation until treatment of the infection has commenced. Search for and cover draining lesions. Infected persons should avoid contact with infants and chronically ill patients. Added infection control precautions may be recommended for cases with infections due to multi-resistant organisms.

Control of contacts

Routine contact tracing is not usually required.

Determining the carrier status amongst family members of a pathogenic strain may be occasionally useful, in which carriers might be recommended antibiotics to eliminate the bacteria such as mupirocin.

Control of environment

Encourage hand washing, especially in the hospital setting.

Outbreak measures

The Department of Human Services may investigate unusual clusters of staphylococcal infection in the community, particularly those associated with antibiotic resistant strains.

This may include:

- investigation of the source of infection including microbiological screening of contacts
- advising on added infection control precautions for cases and carriers
- treatment recommendations for cases and carriers.

Special settings

Hospital nursery workers with minor lesions such as boils or abscesses should not have direct contact with infants until the lesion has healed.

All known or suspected cases in a nursery should be isolated.

In school settings, the child should be excluded from school until specific treatment begins. Lesions must be covered with a watertight dressing. Contacts do not need to be excluded.

Streptococcal disease
(Group A beta-haemolytic streptococcus)

Victorian statutory requirement
Notification is not required.

School exclusion: exclude until the child has received antibiotic treatment for at least 24 hours and the child feels well.

Infectious agent
Streptococcus pyogenes or Group A streptococci (GAS) has approximately 80 serologically distinct types. Those producing skin infections are usually of different serological types to those that cause throat infections.

Identification
Clinical features

The spectrum of disease caused by GAS includes:

- superficial infections such as pharyngitis, impetigo and pyoderma
- scarlet and puerperal fever
- severe invasive disease such as necrotising fasciitis, toxic shock syndrome and septicaemia
- post-streptococcal immunological sequelae include acute rheumatic fever and acute glomerulonephritis.

Method of diagnosis

Superficial infection is diagnosed by isolation of the organism from infected tissues. Invasive infection can be confirmed by isolation of the organism from a normally sterile site such as blood. Throat swabs are of limited value due to the frequency of inapparent Streptococcal carriage. Definitive identification depends on specific serogrouping procedures.

Antigen detection tests are available for rapid identification. A rise in serum antibody titres (anti-streptolysin O, anti-hyaluronidase, anti-DNAase B) may also be demonstrated in sera taken in the acute and convalescent phases of the disease.

Incubation period
The incubation period is usually one to three days.

Public health significance and occurrence
The incidence of GAS infections and their sequelae are not well documented in Australia except in Aboriginal communities in northern Australia. In the USA, acute pharyngitis is one of the most common reasons for seeking medical advice and GAS is thought to be responsible for 15–30% of pharyngitis in children and 5–10% in adults. The community burden of pyoderma in industrialised countries in not well documented.

Preliminary data from a voluntary surveillance system implemented in Victoria in 2002 suggests the incidence of invasive GAS disease may be greater than 4 per 100 000 per year, with a case-fatality rate of approximately 11%.

Outbreaks occur in childcare settings, institutions, and in remote communities in northern and central Australia.

Reservoir
Humans.

Mode of transmission
GAS is usually transmitted via large respiratory droplets or direct contact with infected persons or carriers. It is rarely transmitted by indirect contact through objects. Outbreaks of streptococcal infection may occur as a result of ingestion of contaminated foods such as milk, milk products and eggs.

Period of communicability
With appropriate antibiotic therapy GAS is communicable for 24–48 hours. In untreated uncomplicated cases communicability can last for 10–21 days. Communicability can be prolonged in untreated complicated cases.

Susceptibility and resistance
Pharyngitis and tonsillitis are common in children aged 5–15 years, whereas pyoderma occurs more frequently in children aged less than five years. Most people in their lifetime will develop a GAS throat or skin infection and many of the throat infections may be subclinical. People with chronic illnesses like cancer and diabetes, those on kidney dialysis, or those who use medications such as steroids, have a higher risk than healthy persons. There is an increased risk of infection in varicella (chickenpox).

Control measures
Preventive measures

There are currently no vaccines available but candidate vaccines are being used in clinical trials. Food-borne disease can be prevented by pasteurising milk and milk products and careful preparation and storage of high risk foods, particularly eggs.

Control of case

Treatment is dependent on the clinical presentation and severity of disease. Evidence has accumulated that antibiotics may not always be indicated in pharyngitis or tonsillitis. The current version of *Therapeutic guidelines: antibiotics* (Therapeutic Guidelines Limited) should be consulted prior to treatment.

Infected children should be excluded from schools and children's services centres until they have received antibiotics for at least 24 hours and the child feels well. People with skin lesions should be excluded from food handling until infection has resolved.

Control of contacts

Consider the diagnosis in symptomatic contacts. Few people who come in contact with GAS will develop invasive GAS disease. At present, the role of antibiotic prophylaxis for close contacts of cases of invasive GAS infection is uncertain. However in certain circumstances, antibiotic therapy may be appropriate for those at higher risk of infection.

Control of environment

Standard infection control procedures should be applied.

Outbreak measures

Outbreak management is dependent on the setting and specific disease. Seek advice from the Department of Human Services.

Additional sources of information

- Passmore, J, Kelpie, L & Carapetis, J 2003, 'Surveillance for invasive group A streptococcal disease in Victoria-the first 12 months', *Vic Infect Dis Bulletin*, vol. 6, no. 2, p. 30, www.health.vic.gov.au/ideas

Syphilis

Victorian statutory requirement

Syphilis (Group C disease) must be notified in writing within five days of diagnosis.

Specific information must be notified under the Health (Infectious Diseases) Regulations 2001. To maintain confidentiality, only the name code (first two letters of the surname followed by the first two letters of first name) is required. A questionnaire is sent to the diagnosing doctor to collect additional information on the case that is essential for detecting disease trends and informing policy development.

Medical practitioners have a statutory obligation under the *Children and Young Person's Act 1989* to notify the Department of Human Services' Child Protection service if they believe that a child is in need of protection on the basis of sexual abuse.

Infectious agent

The spirochaete *Treponema pallidum,* subspecies *pallidum* is the infective agent.

Identification

Clinical features

Syphilis is characterised by a primary lesion, a secondary eruption involving skin and mucous membranes, long periods of latency, and late lesions of skin, bone, viscera, cardiovascular and central nervous systems.

The stages of syphilis can be divided into:

- early syphilis where primary (chancre), secondary (rash or condylomata lata) or latent syphilis (asymptomatic) of less than two years duration exist based on serology results
- late latent syphilis where latent syphilis has existed for two or more years or of indeterminate duration, in the absence of neurosyphilis and other symptoms and signs of disease
- tertiary syphilis where cardiovascular involvement and neurosyphilis is present.

Syphilis in pregnancy

Foetal infection may result in abortion, stillbirth, premature delivery and perinatal death. In congenital syphilis, generalised systemic disease in a live born infant is present.

Method of diagnosis

Syphilis can be diagnosed by the demonstration of spirochaetes in the exudate from primary chancres or from the mucous membrane lesions of secondary syphilis, using dark field microscopy or immunofluorescence.

Dark field microscopy is a difficult technique and requires an experienced operator for reliable results. The test is unreliable on mucous membrane lesions due to the presence of morphologically similar saprophytic spirochaetes. Dark field is also best done on site, as drying of the exudate during transport to the laboratory renders the specimen unsuitable for microscopy. Immunofluorescence is more sensitive and does not have to be performed immediately. It is suitable for use with mucous membrane lesions but it is not currently performed by the Victorian Infectious Diseases Reference Laboratory.

More commonly syphilis is diagnosed using a combination of treponemal and non-treponemal serological tests:

- Treponemal tests measure specific treponemal antibodies in serum. These include *Treponema pallidum* particle agglutination, enzyme immunoassay and fluorescent Treponemal antibody absorption tests. Once these tests are reactive they usually remain so for life and give no indication of current disease activity. Enzyme immunoassays with highly purified *Treponema pallidum* antigens are becoming more commonly used for screening for syphilis. These assays have a high specificity and sensitivity. IgM enzyme immunoassay for the detection of IgM antibodies to *Treponema pallidum* is a useful assay for the diagnosis of congenital syphilis.
- Non-treponemal tests such as rapid plasma reagin (RPR) and venereal diseases research laboratory test measure antibodies that are produced in response to syphilis and also to a relatively large number of other conditions. This results in biological false positives. The non-treponemal test gives an indication of current disease activity.

All sera showing reactive serology on screening tests should be forwarded to a reference laboratory for confirmatory testing.

It is necessary to interpret syphilis serology in the context of:

- clinical history and examination
- serial RPR titres tested in parallel where possible (results obtained from different laboratories are not directly comparable)
- treponemal test results
- a past record of treatment.

It is essential that all cases of syphilis receive close clinical and laboratory follow-up.

Lumbar puncture is advised when there are:

- neurological or ophthalmic signs or symptoms
- evidence of active tertiary syphilis
- treatment failure
- HIV infection with late latent syphilis or syphilis of unknown duration.

Note that other sexually transmissible infections may be present in addition to syphilis. Patients in whom syphilis is diagnosed should be encouraged to be tested for HIV infection.

Incubation period

The incubation period is from ten days to three months and is usually three weeks.

Public health significance and occurrence

This is a complex disease. Sequelae include neurosyphilis, cardiovascular syphilis and congenital infection with foetal death, stillbirth and abortion. Testing and effective treatment are available to facilitate the interruption of disease transmission. Immune responses are only partially protective and reinfection may occur following treatment. Syphilis enhances the transmission of HIV, like other diseases that cause genital ulcers.

The number of notifications of infectious syphilis in Victoria is currently relatively small. However, syphilis occurs worldwide and has a high incidence in other parts of Australia. Other developed countries have experienced recent outbreaks. Imported infectious cases could result in syphilis re-emerging as a significant public health issue.

Reservoir

Humans are the only reservoir.

Mode of transmission

Transmission occurs primarily by sexual contact. Transplacental infection of a foetus may occur during the pregnancy of an infected woman. Foetal infection occurs with high frequency in untreated early infections of pregnant women and with lower frequency later in the disease or in late latency. Syphilis may also be transmitted by transfusion of blood from infected individuals, however this risk is minimised by the screening of all donated blood in Australia.

Period of communicability

A case is considered sexually infectious until the end of the early latent period which is approximately two years after infection. Infectious moist mucocutaneous lesions are present in the primary and secondary stages of syphilis and may reoccur intermittently in the early latent period. These lesions may not be apparent to the infected individual.

Susceptibility and resistance

Everyone is susceptible to infection.

Control measures

Preventive measures

Education about safe sex practices including the use of condoms and early detection of infection by testing of people at risk are the main prevention measures.

Control of case

Penicillin is the drug of choice to treat syphilis.

Further information on the clinical management of patients with syphilis can be found in the latest editions of the *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited) and the National management guidelines for sexually transmissible infections (Venereology Society of Victoria, 2002).

The necessity for long term follow-up with repeat serology and the frequent presence of complicating factors makes it desirable to seek specialist advice.

Control of contacts

Sexual contacts should be identified. The extent of contact tracing depends on the clinical stage of infection.

For primary syphilis, all persons having sexual contact with the index case during the three months preceding onset should be evaluated. Such contacts should be treated as for the case, even if their serology is negative.

For secondary syphilis, this period should be extended to six months and for early latent syphilis, to twelve months.

For late latent syphilis, any sexual partners and also children of infected women should be evaluated.

For congenital syphilis, all members of the immediate family should be evaluated.

Contact tracing assistance can be provided by the Department's partner notification officers (phone 9347 1899).

All newborns of mothers with syphilis should be investigated and treated in consultation with a specialist.

Control of environment
Not applicable.

Outbreak measures
Not applicable.

Additional sources of information

- Australian Government Department of Health and Family Services 1997, *Contact tracing manual – a practical handbook for health care providers managing people with HIV, viral hepatitis, other STDs and HIV-related tuberculosis*, Australian Government Department of Health and Family Services.
- Centers for Disease Control and Prevention 2002, 'Sexually transmitted diseases treatment guidelines 2002', *Morbidity and Mortality Weekly Report*, vol. 51, RR06, pp.1–80. http://www.cdc.gov/mmwr
- Egglestone, SI et al 2000, 'Serological diagnosis of syphilis,' *Communicable Diseases & Public Health*, vol. 3, no. 3, pp. 158–62.
- Larsen, SA et al 1995, 'Laboratory diagnosis and interpretation of tests for syphilis', *Clin Micro Revs*, vol. 8, no. 1, pp. 1–21.
- Nganampa Health Council 1999, *How to interpret syphilis results – A manual for nursing and medical staff in remote area clinics*, 2nd ed.
- Singh, AE et al 1999, 'Syphilis: Review with emphasis on clinical, epidemiologic and some biologic features', *Clin Micro reviews* vol. 12, no. 2, pp. 187–209.
- Venereology Society of Victoria 2002, *National management guidelines for sexually transmissible infections*, http://www.mshc.org.au

Taeniasis

Victorian statutory requirement

Notification and school exclusion are not required.

Infectious agent

Taenia solium (pork tapeworm) causes both intestinal infection with the adult tapeworm and somatic infections with the larvae (cysticerci).

Taenia saginata (beef tapeworm) causes only intestinal infection with the adult tapeworm in humans.

Identification

Clinical features

Taenia saginata infections are often asymptomatic apart from the anal passage of tapeworm segments. Infection may be associated with epigastric pain, diarrhoea and weight loss.

Taenia solium adult worm infections are also usually asymptomatic. Many tissues and organs may be infected by the larval form (cysticercosis). Neurocysticercosis is a serious but rarely fatal complication which may be manifest as headaches, epileptiform seizures and visual or psychiatric disturbances.

Method of diagnosis

Infection with an adult tapeworm can be diagnosed through the identification of segments, eggs or the head of the parasite in faeces or perianal swabs. Microscopic examination of the eggs cannot differentiate between the two species.

Specific serological tests are available to support the clinical diagnosis of taeniasis.

Subcutaneous cysticerci may be visible or palpable. Calcified cysticerci may be visualised using ultrasound, CT scan or MRI.

Incubation period

Symptoms of cysticercosis may appear from weeks to years after infection. Eggs appear in the faeces 8–12 weeks after infection with the adult *T. saginata* tapeworm and after 10–14 weeks with *T. solium*.

Public health significance and occurrence

Taeniasis occurs worldwide. It is commonly seen in parts of Latin America, Africa, South East Asia and Eastern Europe. Both forms are usually imported to Australia but sporadic locally acquired cases of *T. saginata* infection have been reported.

Many infections are largely asymptomatic, but the larval stage of *T. solium* may cause fatal cysticercosis. Chronic tapeworm infections contribute to malnutrition for developing communities in many parts of the world.

Reservoir

Humans are the definitive host for both species. Cattle are the intermediate host for *T. saginata* and pigs for *T. solium*.

Mode of transmission

Eggs of *T. saginata* passed in the faeces of an infected person are only infectious to cattle. Humans are infected by ingestion of raw or undercooked beef infected with cysticerci bovis, the larval stage of *T. saginata*. In humans the adult tapeworm develops in the intestine over two to three months. The cycle of infection repeats when infectious eggs are passed in the faeces and later ingested by cattle, slowly migrating into the flesh and transforming into the larval stage.

Infections by *T. solium* may follow a similar cycle with consumption of infected pork leading to the subsequent development of adult tapeworms. However human infection may also occur through the consumption of *T. solium* eggs. This occurs by direct transfer from the faeces of an infected person or through the ingestion of contaminated food or water. When the eggs of *T. solium* are ingested by either humans or pigs the embryos escape the shells and penetrate the intestinal wall, with subsequent spread of larvae to various tissues to produce cysticercosis.

Period of communicability

T. saginata is not directly transmissible from person to person although *T. solium* may be. Adult tapeworms may persist in the intestines for up to 30 years and are able to disseminate eggs for all of this time. Eggs may remain viable in the environment for months.

Susceptibility and resistance

Everyone is susceptible to infection. Infection does not appear to confer immunity.

Control measures

Preventive measures

The public should be advised to avoid faecal contamination of soil, and human and animal food; avoid the use of raw sewage for irrigation of pasture soil; and to cook beef and pork thoroughly.

Beef and pork, should be adequately cooked, for example at 60°C for five minutes.

Freezing meat below –5°C for more than four days will kill cysticerci.

Meat should be routinely inspected for evidence of taeniasis at slaughter.

Control of case

Praziquantel or niclosamide are used for treatment of beef and pork tapeworm infections. Consult the current version of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Persons harbouring adult *T. solium* should be immediately identified and treated to prevent human cysticercosis. For cysticercosis surgical intervention may relieve symptoms. For CNS cysticercosis, praziquantel or albendazole may be used, with corticosteroids if indicated.

Isolation is not required. The case and relevant caregivers should be advised that the case's faeces may be infectious and advised on sanitary disposal of wastes.

Control of contacts

Symptomatic patients exposed to a suspected source of infection should be evaluated for evidence of taeniasis.

Control of environment

If the history is consistent with local infection the source of the infection should be investigated, often with the assistance of the local government.

Outbreak measures

Not applicable.

Additional sources of information

- Centers for Disease Control and Prevention, http://www.dpd.cdc.gov

Tetanus

Victorian statutory requirement

Tetanus (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion is not applicable.

Infectious agent

Clostridium tetani, the tetanus bacillus is the causative agent.

Identification

Clinical features

Tetanus is an acute, potentially fatal disease caused by tetanus bacilli multiplying at the site of an injury. These produce an exotoxin that reaches the central nervous system and causes muscle stimulation.

Initial features are increased muscle rigidity. This may be restricted to and most pronounced in muscles near the injury (localised tetanus). Depending on severity, muscle rigidity usually affects most parts of the body and is associated with hyperreflexia. As a result, features such as neck, back and limb stiffness, stiff jaw or 'lock jaw' (trismus) and a sardonic smile (risus sardonicus) may occur.

With progression, superimposed painful muscle spasms can appear anywhere or involve most body muscles simultaneously. Opisthotonos can result. This is when spasm is most marked in the back muscles causing the head and heels to bend backward and the body to bow forward. Painful spasms may become very frequent and together with background rigidity cause life-threatening interference with respiration.

Laryngeal spasm is a very serious complication which may occur at any stage and can cause sudden asphyxia. Exhaustion and inability to swallow are also associated with severe disease.

Case fatality rates vary from 10–90% and are highest in infants and the elderly.

Method of diagnosis

Clinical features of severe classical tetanus are virtually diagnostic.

Laboratory confirmation of tetanus infection is often difficult.

C. tetani antibodies are sometimes detectable in serum samples but may result from waning past immunisation. Cultures from the site of infection should be attempted although the organism is often not recovered.

Incubation period

The incubation period is usually three to 21 days although it may range from one day to several months depending upon the nature of exposure. Most cases occur within14 days.

Cases with shorter incubation periods tend to have more severe disease and thereby a greater risk of death.

Public health significance and occurrence

Tetanus occurs worldwide but is now rare in developed countries due to high immunisation rates. Infection is most likely in older people who have never been immunised or who have waning and inadequate immunity.

Tetanus is still common in developing countries with low immunisation rates and where contact with animal excreta is more common. Tetanus, particularly neonatal tetanus, is a significant cause of death in these settings.

Intravenous drug use is an independent risk factor for tetanus in the absence of acute injuries and may be linked to localised case clusters.

Reservoir

C. tetani is widely distributed in cultivated soil and in the gut of humans and animals. Spores can usually be found wherever there is contamination with soil.

Mode of transmission

Tetanus is not directly transmitted from person to person.

Spores may be introduced through contaminated puncture wounds, lacerations, burns or contaminated injected 'street drugs'. Tetanus can result from minor wounds considered too trivial for medical consultation.

The presence of necrotic tissue or foreign bodies encourages the growth of anaerobic organism such as *C. tetani*. Tetanus rarely follows surgical procedures today.

Period of communicability

Spores may remain viable for many years in the environment.

Susceptibility and resistance

Active immunity is produced by immunisation with tetanus toxoid and persists for at least ten years after full immunisation.

Transient passive immunity follows injection of tetanus immune globulin (TIG) or tetanus antitoxin.

Recovery from tetanus is not necessarily associated with immunity.

Control Measures

Preventive measures

Tetanus toxoid is part of the Australian Standard Vaccination Schedule. Primary immunisation for children begins at two months of age and requires three doses of tetanus toxoid-containing vaccine at two-monthly intervals. Children should be given a booster at four years of age. A further booster dose is given prior to leaving school (15–17 years of age) and again at 50 years of age.

For further information on tetanus vaccination, particularly with respect to the management of children who have missed doses, consult the current edition of *The Australian immunisation handbook* (National Health and Medical Research Council).

The use of tetanus toxoid in the management of wounds, with or without tetanus immunoglobulin, is determined by considering the vaccination history of the person and the nature of the wound. For further information on the management of bites and other tetanus-prone wounds, consult the current edition of *Therapeutic guidelines: antibiotic* (Therapeutic Guidelines Limited).

Control of case

Refer the patient immediately to a specialised centre with intensive care facilities. The principles of treatment include:

- tetanus immunoglobulin (TIG) by intramuscular injection
- IV penicillin in large doses for 10 to 14 days. Intravenous metronidazole is a reasonable alternative for patients with immediate penicillin hypersensitivity
- adequate wound debridement
- careful attention to provide an adequate airway and to control muscle spasm
- case investigation to determine the circumstances of injury
- completion of course of active immunisation after recovery.

Control of contacts

Not required.

Control of environment

Not required.

Outbreak measures

Not applicable.

Toxoplasmosis

Victorian statutory requirement

Notification and school exclusion are not required.

Infectious agent

Toxoplasma gondii is a protozoal disease.

Identification

Clinical features

Toxoplasmosis infection is asymptomatic in 80% of people. The most common sign in symptomatic patients is enlarged lymph nodes, especially around the neck. The illness may mimic glandular fever with other symptoms of muscle pain, intermittent fever and malaise.

Dormant infection persists for life and can reactivate in the immunosuppressed person.

More serious disease can develop or reactivate in immunosuppressed patients with brain, heart or eye involvement, pneumonia and occasionally death.

Cerebral toxoplasmosis or chorioretinitis are frequent complications of AIDS when the lymphocyte CD4 cell count drops below 100 / cu mm.

Acute toxoplasmosis in pregnant women can affect the unborn child. In early pregnancy brain damage as well as liver, spleen and eye disorders may occur. Infection in late pregnancy may result in persistent eye infection through life. Toxoplasmosis acquired after birth usually results in no symptoms or only a mild illness.

Method of diagnosis

Infection may be diagnosed by visualisation of the protozoa in biopsy material or serology.

Serological results require careful interpretation and should preferably be performed and discussed with a reference laboratory. In general, *toxoplasma*-specific IgG antibody appears two to three weeks after acute infection, peaks in six to eight weeks and often persists lifelong.

Presence of *toxoplasma*-specific IgM antibody suggests infection within the last two years. False positive IgM results are common and should always be repeated before final interpretation. They are common in autoimmune disease.

Presence of IgA antibodies is said to correlate with acute infection.

Testing paired sera taken two weeks apart is often helpful as is IgG antibody avidity testing.

A specific PCR performed on amniotic fluid may determine if a foetus has become infected.

Incubation period

The incubation period is uncertain but probably ranges from 5–23 days.

Public health significance and occurrence

Toxoplasmosis occurs worldwide in mammals and birds. Infection in humans is common.

Infections during pregnancy may lead to severe complications for the foetus. Primary or reactivated lesions may lead to severe complications in immunosuppressed patients.

Reservoir

The main host in Australia is the domestic cat. Cats acquire the infection mainly through eating small infected mammals including rodents and birds, and rarely from the ingestion of infected cat faeces. Only young felines harbor the parasite in the intestinal tract, where the sexual stage of the life-cycle takes place resulting in the excretion of oocysts in faeces for 10–20 days.

Many other intermediate hosts including sheep, goats, rodents, cattle, swine, chicken and birds may carry an infective stage of T. gondii encysted in their tissues. This occurs more commonly in muscle and brain. Tissue cysts remain viable for long periods.

Mode of transmission

Adults most commonly acquire toxoplasmosis by eating raw or undercooked meat infected with tissue cysts. Consumption of contaminated, unpasteurised milk has also been implicated.

Children may become infected by ingestion of oocysts in dirt or sandpit sand after faecal contamination by cats, particularly kittens, or other animals.

The infection may also be transmitted through blood transfusion and organ transplantation. Transplacental transmission may occur when a woman has a primary infection during pregnancy.

Period of communicability

Toxoplasmosis is not transmitted from person to person spread except in-utero.

Oocysts spread by cats sporulate and become infective one to five days later. They may remain infective in water or moist soil for over a year.

Tissue cysts in meat remain infective for as long as the meat is edible and under-cooked.

Susceptibility and resistance

Everyone is susceptible to infection. About 75% of women of childbearing age are susceptible.

Immunity is thought to be life long however patients undergoing immunosuppressive therapy, in particular for haematological malignancies, or patients with AIDS, are at high risk of developing illness from reactivated infection.

Control measures

Preventive measures

No immunisation is available.

Pregnant women and immunosuppressed people should be advised to:

- cook meat thoroughly (until no longer pink) and avoid uncooked cured meat products
- not consume unpasteurised milk or its products
- wash all raw fruit and vegetables carefully before eating
- wash hands thoroughly before meals and after handling raw meat
- delegate the cleaning of cat litter trays to others wherever possible and if this is not possible, gloves should be worn during cleaning and hands washed well afterward
- cat litter trays should be emptied daily and regularly disinfected with boiling water to dispose of the oocysts before they become infective.

Cats should only be fed with dry, canned or boiled food and should be discouraged from hunting and scavenging. However, direct contact with cats is rarely the cause of infection. Cats are generally infected as kittens and only excrete the oocysts for two weeks after their original infection.

Sandpits should be covered when not in use to stop cats defecating in the pit.

Control of case

Isolation of patient is not required.

Specific anti-protozoal treatment may be indicated in immunosuppressed persons, infections during pregnancy, or where there is eye or other organ involvement. Specialist advice should be sought. Immunosuppressed persons may also require prophylactic treatment for the duration of their immunosuppression.

Infants who acquire an infection before birth may require prolonged treatment to reduce the risk of ongoing active infection.

Control of contacts

Not applicable.

Special settings

Pregnancy

Children of mothers with evidence of previous immunity more than six months prior to conception are not at risk. Primary infection in pregnancy is rare although up to one third of these infections result in transplacental spread to the developing foetus.

Primary infection in pregnancy can cause serious foetal disease. Infection in the first trimester results in a low foetal infection rate (15%) but a higher risk of serious disease. Infection later in pregnancy results in a higher infection rate but generally less severe disease.

Diagnosis and treatment during pregnancy appears to reduce the effects on the baby.

False positive IgM antibody test (and less commonly IgG) results do occur and treatment should never begin without further testing. Where infection of the mother is confirmed, treatment is indicated.

Amniocentesis with PCR testing can be carried out to determine whether transmission to the foetus has occurred.

Newborns

Newborns of mothers with primary infection during pregnancy or active infection, and immunosuppressed patients are treated empirically until congenital disease is ruled out. Where infection is confirmed, treatment is continued for 12 months to help reduce long term effects.

Outbreak measures

Not applicable.

Additional sources of information

- Gilbert, G 2002, 'Infections in pregnant women' *MJA*, vol. 176, pp. 229–236.

Typhoid and paratyphoid fever

Victorian statutory requirement

Salmonella (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion: exclude until approved to return by the Department of Human Services.

Work exclusion: exclusions apply to food-handlers and some health care workers (see below).

Infectious agent

Salmonella typhi, the typhoid bacillus and *Salmonella paratyphi*, with three recognised serovars A, B and C are the infectious agents.

Identification

Clinical features

Typhoid fever (enteric fever) is a septicaemic illness characterised initially by fever, bradycardia, splenomegaly, abdominal symptoms and 'rose spots' which are clusters of pink macules on the skin.

Complications such as intestinal haemorrhage or perforation can develop in untreated patients or when treatment is delayed.

Paratyphoid fever presents a similar clinical picture but is usually milder, shorter in duration and with fewer complications.

Method of diagnosis

Diagnosis is made by culture of typhoid or paratyphoid bacilli from the blood, urine or faeces. Repeated sampling may be necessary. Serology in the form of the Widal test is no longer routinely used.

Phage typing is used for characterising *S. typhi* and *S. paratyphi* isolates for epidemiological purposes and in outbreak settings. A number of phage types are recognised.

Incubation period

The incubation differs for typhoid and paratyphoid fever:

- typhoid fever is usually 8–14 days but this depends on the infective dose and can vary from three days to one month
- paratyphoid fever is usually one to ten days.

Public health significance and occurrence

Typhoid and paratyphoid infections occur worldwide. Outbreaks occur in areas with poor sanitation and inadequate sewerage systems. Approximately 30–35 cases of enteric fever occur in Victoria each year. The majority of these are returned travellers, especially from the Indian subcontinent.

Reservoir

Reservoirs for typhoid and paratyphoid fever are:

- typhoid fever: Human gallbladder carriers and rarely human urinary carriers
- paratyphoid fever: Humans and rarely domestic animals.

Mode of transmission

Salmonella is transmitted by contaminated water and food and rarely by direct contact. Water, ice (if unboiled water used), raw vegetables, salads and shellfish are important sources for travellers. The disease commonly occurs in association with poor standards of hygiene in food preparation and handling.

Period of communicability

It is communicable as long as typhoid or paratyphoid bacilli are present in excreta. Some patients become permanent carriers.

Susceptibility and resistance

Everyone is susceptible to infection. Immunity following clinical disease or immunisation is insufficient to protect against a large infectious dose of organisms.

Control measures

Preventive measures

Vaccination is not routinely recommended, except for travellers who will be exposed to potentially contaminated food and water in countries such as in Asia, the Middle East, Africa, Latin America and the Pacific Islands.

Vaccination should be considered for laboratory workers in potential contact with Salmonella typhi.

Three typhoid vaccines are currently available in Australia. The live oral vaccine and Vi capsular polysaccharide injectable vaccine generally cause few adverse reactions.

A combination hepatitis A and typhoid injectable vaccine is also available. All formulations are equally effective.

Vaccination does not offer full protection from infection and travellers should be advised to exercise care in selecting food and drink.

No vaccine is available against paratyphoid fever.

The community should be educated about personal hygiene, especially thorough hand washing after toilet use and before food preparation.

Control of case

Hospitalisation is usually required for acute infections.

Antibiotic therapy may include one or more to the following agents: ciprofloxacin, ceftriaxone, chloramphenicol, amoxycillin or co-trimoxazole. However, strains resistant to chloramphenicol, amoxycillin and co-trimoxazole are common in south Asia. Failure to respond to ciprofloxacin has been reported from Vietnam. In the UK decreased susceptibility to ciprofloxacin has been exhibited with increasing numbers of treatment failures particularly in patients with a travel history to India and Pakistan. A similar picture is emerging in Victoria with ongoing S. typhi and S. paratyphi after treatment being noted. Consult the current version of Therapeutic guidelines: antibiotic (Therapeutic Guidelines Limited).

Education should be given to the patient regarding the importance of completing the course of antibiotics, the possibility of relapse, persisting excretion, the need for good personal hygiene and precautions in food preparation.

Follow-up of all patients is conducted by the Department of Human Services to identify possible sources of infection, other cases, and to manage ongoing risks.

If the patient is a food-handler or works in a profession that poses a high risk of transferring infection to others, such as health care workers, or child care workers, they should be advised to cease work until advised by the Department.

The Department arranges the collection and testing of weekly faecal specimens for *S. typhi* or *S. paratyphi* to be taken over three consecutive weeks, commencing no sooner than at least 48 hours after cessation of antibiotic treatment. Food handlers and workers in high risk professions are generally excluded from high risk work or patient care until they have had three consecutive negative faecal specimens.

Control of contacts

Contacts should be educated about the disease so as to reduce the risk of transmission and to allow for early identification if they develop symptoms.

The decision to screen contacts of cases is dependent upon the extent of contact and the likely source of the patient's infection. Faecal screening is generally arranged for:

- Household contacts of a case who are food-handlers or in a high risk profession. Screening is more intensive and includes the entire household if the patient has no history of travel to a typhoid-endemic area.
- Fellow travellers.

Use of typhoid vaccine for contacts is not generally recommended. Typhoid vaccination is only recommended for persons with intimate exposure to a documented typhoid fever carrier, such as occurs with continued household contact.

Control of environment

A public health investigation is carried out to determine the most likely source of infection. A history of travel to an endemic area is usually found.

If there is no history of travel, local sources of infection are investigated to identify further cases, asymptomatic carriers, and contaminated food items.

Food industry

If a case is involved in commercial food preparation, the Department will determine the appropriate management of the workplace on an individual basis.

Outbreak measures

All cases are intensively investigated, whether sporadic or part of a cluster. Further actions to reduce the risk of infection during an outbreak may include:

- selective elimination of suspected contaminated food
- ensuring pasteurisation of milk
- ensuring appropriate chlorination or boiling of drinking water prior to consumption
- reviewing the integrity of waste and sewerage systems.

Widespread use of typhoid vaccine is not generally recommended.

International measures

Typhoid vaccination is recommended for prolonged travel to endemic areas.

Additional sources of information

- Skull, SA, Tallis, G 2001, 'An evidence-based review of current guidelines for the public health control of typhoid in Australia: a case for simplification and resource savings', *Aust N Z J Public Health*, vol. 25, no. 6, pp. 539–42.

Verotoxin producing *E. coli* (VTEC)

(Enterohaemorrhagic *E. coli* [ETEC], Shiga-like toxin producing *E. coli* [STEC])

Victorian statutory requirement

Haemolytic uraemic syndrome (Group A disease) must be notified immediately by telephone followed by written notification within five days.

VTEC and STEC (Group B disease) must be notified in writing within five days of diagnosis.

School exclusion: exclude if required by the Secretary and only for the period specified by the Secretary. Contacts are not excluded.

Infectious agent

Escherichia coli serotypes capable of producing toxins (Shiga-like or Vero-toxins) similar to those of *Shigella dysenteriae* type 1 are the causative agents. The most important are *E. coli* O157:H7, E. coli O111:H8 and *E. coli* O26:H11.

Identification

Clinical features

Illness is characterised by severe abdominal pain and cramping and watery diarrhoea which becomes grossly bloody and lasts five to ten days. Fever is usually mild or absent. Asymptomatic infection can occur.

In children aged less than five years and the elderly, infection may lead to haemolytic uraemic syndrome (HUS). This is a disease characterised by renal failure, a high mortality rate and thrombotic thrombocytopaenic purpura (TTP). HUS and TTP are complications of infection with serotype O157:H7.

Method of diagnosis

Diagnosis is confirmed by isolation of the organism from faeces. Other diagnostic methods may be required including:

- demonstrating the presence of Shiga-like toxins
- serotyping
- DNA probes that identify the toxin producing genes or the presence of the VTEC virulence plasmid.

As screening for VTEC is not routine in Victorian laboratories the test should be specifically requested for persons with bloody diarrhoea.

As a negative stool culture is not exclusionary, HUS should be considered in the presence of the following:

- acute microangiopathic anaemia on peripheral blood smear
- acute renal impairment (haematuria, proteinuria or elevated creatinine level)
- thrombocytopaenia, particularly during the first seven days of illness.

Incubation period

The incubation period is two to eight days, with an average of three to four days.

Public health significance and occurrence

Recognition of VTEC as an important cause of food-borne illness is relatively recent. The first outbreaks of O157:H7 were reported in the United Kingdom and United States in the early 1980s.

Since then several large outbreaks have been reported worldwide and over 70,000 cases are reported in the US each year. A particular brand of fermented salami was implicated in a large outbreak in South Australia in 1995.

An average of five cases of VTEC and three cases of HUS are reported in Victoria each year. This is likely to be a significant underestimate of the true burden of disease related to VTEC due to the lack of routine screening of bloody diarrhoea.

Reservoir

The gastrointestinal tracts of cattle and possibly other domesticated animals act as reservoirs. Humans serve as reservoirs for person to person transmission. Prolonged carriage is uncommon.

Mode of transmission

Ingestion of contaminated food and water and person to person and animal to person transmission by the faecal-oral route are responsible for VTEC infection. Undercooked meat, especially ground meat or mince, is a source of infection. Other known food sources have included lettuce, sprouts, salami, unpasteurised milk and fruit juices. The infectious dose necessary to cause disease is thought to be as low as ten organisms.

Period of communicability

VTEC is communicable for as long as the organism is present in faeces which is approximately one week for adults but may be as long as three weeks in children.

Susceptibility and resistance

Everyone is susceptible to infection. Children and the elderly are at higher risk for severe disease. Antibiotic resistance is of increasing concern. A study recently conducted in Melbourne found that 28% of VTEC strains isolated from healthy babies, who had neither contact with antibiotics nor had gastrointestinal symptoms for at least two weeks prior to the specimen being taken, were resistant to one or more of the antibiotics tested.

Control measures

Preventive measures

Avoid ingestion of inadequately cooked meat and meat products, unpasteurised milk and fruit juices, unwashed salad ingredients and untreated water. Hand washing before and after using the toilet and preparing or eating food is critical.

Control of case

Treatment is generally supportive, particularly maintenance of hydration. The role of antibiotics in the management of VTEC is unclear and there is some concern that they may precipitate the onset of HUS. Specialist medical advice should be sought for cases of HUS and TTP. Enteric precautions should be strictly observed in the management of hospitalised cases.

Food handlers, child care workers and health care workers must not work until symptoms have stopped and two consecutive faecal specimens taken at least 24 hours apart are negative for VTEC.

Children must not attend school or child care until diarrhoea has ceased. Any ongoing exclusion is at the discretion of the Secretary of the Department of Human Services.

Control of contacts

The diagnosis should be considered in symptomatic contacts. As for cases, work and school exclusion apply to contacts with diarrhoea.

Control of environment

Environmental surfaces exposed to infectious material should be thoroughly cleaned. Implicated food should be destroyed and contaminated water sources treated.

Particular attention to personal and environmental hygiene should be observed in food premises, institutions and child care centres.

Outbreak measures

A single case of EHEC or HUS is potentially indicative of an outbreak. Search for other cases and identify persons at risk of infection. A source of infection should be sought for all cases of EHEC and HUS. Obtain detailed food and environmental exposure histories from cases. Collect samples of potentially implicated food and send to the Microbiological Diagnostic Unit for analysis. Antibiotic prophylaxis has neither been proven to be efficacious nor safe for the prevention of secondary cases during VTEC outbreaks.

Refer to the Department's *Guidelines for the investigation of gastrointestinal illness* for more detailed on the investigation and management of outbreaks.

Additional sources of information

- Bettelheim, KA, Hornitzky, MA, Djordjevic, SP, Kuzevski, A 2003, 'Antibiotic resistance among verocytotoxigenic Escherichia coli (VTEC) and non-VTEC isolated from domestic animals and humans', *J Med Microbiol*, vol. 52, pt 2, pp. 155–62.
- Karmali, MA 1989, 'Infection by verocytotoxin-producing Escherichia coli', *Clinical Microbiological Review*, vol. 2, no. 1, pp. 15–38.
- Subcommittee of the PHLS Advisory Committee on Gastrointestinal Infections 2000, 'Guidelines for the control of infection with Vero cytotoxin producing *Escherichia coli* (VTEC)', *Commun Dis Public Health*, vol. 3, no. 1, pp. 14–23.

Viral gastroenteritis
(not rotavirus)

Victorian statutory requirement
Isolated cases are not notifiable.

School exclusion: exclude from school or childcare centres until after symptoms have ceased or a medical certificate of recovery is produced.

Infectious agents
Small round structured viruses (SRSVs) including noroviruses and other caliciviruses, astroviruses and adenoviruses are the infective agents.

Identification
Clinical features

Illness is characterised by an acute onset of fever, myalgia, headache, nausea, vomiting, abdominal cramps and watery diarrhoea lasting 12–60 hours. Vomiting is relatively more prevalent among children. Forceful vomiting as a predominant symptom and a significant secondary attack rate in an outbreak of gastroenteritis are suggestive of norovirus infection. Although rare, severe dehydration caused by viral gastroenteritis can be fatal in persons with debilitating health conditions.

Method of diagnosis

Diagnosis is predominantly based on clinical presentation. Virus in stool can be visualised and distinguished by electron microscopy. Nucleic acid hybridisation assays and RT-PCR assays to detect norovirus genome are a sensitive and specific tool for outbreak investigations. Nucleotide sequencing provides a classification of the viruses n observed,and is an important tool in establishing links to contaminated vehicles of infection in outbreak settings.

Incubation period
The incubation period is usually 24–48 hours. The known range for norovirus is 10–50 hours.

Public health significance and occurrence
The endemic burden of gastroenteritis of viral causes is not known however norovirus is recognised as the major cause of outbreaks of non-bacterial gastroenteritis. Explosive outbreaks have occurred in institutions, camps, childcare centres, cruise ships, restaurants and following catered functions. Approximately 50% of gastroenteritis outbreaks investigated each year in Victoria are attributed to viral pathogens. High secondary attack rates result in outbreaks that are often prolonged and difficult to contain.

Disease occurs in all age groups and predominantly affects infants and young children. In Australia, viruses can be detected throughout the year but are more common in the period from late winter to early summer. Norovirus has been reported to account for between 5–17% of cases of diarrhoea in the community and 5–7% of cases requiring treatment by physicians.

Reservoir
The reservoir is thought to be primarily humans.

Mode of transmission
Viral gastroenteritis is predominantly spread via the faecal-oral route. Transmission is facilitated through contaminated food (particularly raw shellfish), water (including ice) and person to person contact. Aerosols are thought to be important in the transmission of norovirus and it is also known to persist on certain contaminated surfaces such as carpets for weeks.

Period of communicability
Communicability continues during the acute phase and for as long as viral shedding persists. Cases should be considered infectious until at least 48 hours after diarrhoea has ceased. Shedding of norovirus in the absence of clinical illness can persist for up to two weeks and is of concern in food-handler related transmission.

Susceptibility and resistance
Everyone is susceptible to infection and infection is not known to confer lifelong immunity.

Control measures
Preventive measures

Prevention is dependent on attention to good food and personal hygiene, particularly hand washing.

Control of case

Treatment is symptomatic and should be focussed on maintaining hydration.

Healthcare workers and food handlers should be excluded from work until at least 48 hours after diarrhoea and vomiting has ceased. Children should be excluded from school or childcare centres until after symptoms have ceased or a medical certificate of recovery is produced. Residents of institutions should be isolated until diarrhoea has ceased.

Control of contacts

Advise case to maintain strict personal hygiene and hand washing in the home.

Determine if others are ill. If so, report to Local Government environmental health officers or the Department's Communicable Diseases Section so that outbreak investigation and control can occur.

Control of environment

The ability of norovirus to survive relatively high levels of chlorine and varying temperatures (from freezing to 60°C) means rigorous attention to clean-up procedures and personal and home hygiene is essential in preventing further transmission.

Outbreak measures

An outbreak is defined as two or more related cases of gastroenteritis. The primary aim is to prevent further disease by identifying the source, cleaning contaminated environments and isolating cases.

Special settings

Specific protocols for the management of outbreaks in special settings are available from the Communicable Diseases Section of the Department of Human Services.

Additional sources of information

Centres for Disease Control and Prevention 2001, 'Norwalk-like viruses: public health consequences and outbreak management', *MMWR*, vol. 50, RR9, pp. 1–18.

Viral haemorrhagic fevers

Victorian statutory requirement

Viral haemorrhagic fever (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

School exclusion: until medical clearance.

Crimean-Congo, Ebola, Lassa and Marburg viral haemorrhagic fevers are subject to Australian quarantine.

Infectious agent

Four viral haemorrhagic fevers (VHFs) are of particular concern because they could be imported into Australia and be transmitted to other people, particularly health care personnel by blood or body fluid inoculation. These quarantinable VHFs are:

- Lassa fever (LF) virus - an arenavirus
- Crimean-Congo haemorrhagic fever (CCHF) virus - a Bunyavirus
- Ebola virus (EV) and Marburg virus (MV) - filoviruses.

Dengue haemorrhagic fever and yellow fever are discussed elsewhere.

Identification

Clinical features

Clinically apparent infections with any of these viruses may present with similar symptoms. Fever is typically insidious in onset and accompanied by severe headache, myalgia and malaise. Other symptoms include retrosternal chest pain, cough, abdominal pain, diarrhoea, conjunctivitis, facial swelling, proteinuria and jaundice. A bleeding diathesis leads to mucosal bleeding, haematemesis, melaena and haematuria. Severe infections are complicated by massive haemorrhage and multi-organ failure.

Case fatality rates vary greatly:

- Lassa fever virus has a case fatality rate of 1% of infected cases but 25% of hospitalised cases.
- Crimean-Congo haemorrhagic fever virus has a case fatality rate of 2–50%.
- Marburg virus has a case fatality rate of 25% and Ebola virus is 50–90%.

Method of diagnosis

The Department of Human Services and the Victorian Infectious Diseases Reference Laboratory (VIDRL) should be consulted prior to the collection and transport of any clinical specimens from suspected VHF patients for diagnostic testing.

All suspected VHF clinical specimens are tested under the highest bio-security level (BSL–4) laboratory conditions. Diagnosis is typically made using specific PCR tests supported by viral isolation and serology. Appropriate specimens are:

- unclotted blood, tissue or nose and throat swabs for viral PCR
- unclotted blood, urine, tissue or nose and throat swabs for virus isolation
- clotted blood for serology.

Incubation period

The incubation period varies according to the causative agent:

- Lassa fever virus is usually 6–21 days
- Crimean-Congo haemorrhagic fever virus is usually one to three days (range 1–12 days)
- Marburg virus is usually three to nine days
- Ebola virus is usually 2–21 days.

Public health significance and occurrence

The term viral hemorrhagic fever (VHF) refers to a group of rare illnesses that are caused by several distinct families of viruses. While some types of hemorrhagic fever viruses can cause relatively mild illnesses, many of these viruses cause severe life-threatening disease.

Lassa, Marburg and Ebola viruses are restricted to sub-Saharan Africa. Crimean-Congo haemorrhagic fever virus is more widely distributed in Africa, the Mediterranean region, the Middle East, Eastern Europe, Central Asia and China. The origins of the Marburg and Ebola viruses are still unclear but most cases appear to have arisen in Africa.

The high case fatality rate means that it is important that the diagnosis is made and treatment is commenced as early as possible. Viral haemorrhagic fevers should be considered in the differential diagnosis of every patient with an unexplained fever who has been exposed to the infection in an area with endemic VHF during the preceding three weeks.

Reservoir

The reservoir for Lassa fever virus is a rodent known as the multimammate rat of the genus *Mastomys* spp.

The reservoirs for Crimean-Congo haemorrhagic fever virus are hares, birds and Hyalomma spp. of ticks. Domestic animals such as sheep, goats and cattle

may act as amplifying hosts.

The natural reservoir of Ebola virus remains unknown. Current evidence suggests that the virus is zoonotic (animal-borne) and is normally maintained in animal hosts native to the African continent. This could include other primates such as gorillas.

Mode of transmission

Transmission for the viral haemorrhagic fevers depends on the type of virus:

- The *Mastomys* rodents shed Lassa fever virus in urine and droppings. The virus can be transmitted through direct contact with these materials, touching objects or eating food contaminated with these materials, or through cuts or sores. Person to person transmission may occur through sexual contact or inoculation with blood.
- Crimean-Congo haemorrhagic fever virus is transmitted by the bite of infective *Hyalomma* spp. ticks. Ticks are believed to acquire the virus by transovarian transmission or from animal hosts. Nosocomial spread to medical workers in contact with infected blood or secretions has been observed. Slaughtering of infected animals is also linked to some infections.
- The source of infection for the index human for Ebola and Marburg viruses is usually unknown. Secondary human infections occur by person to person spread through direct contact with infected blood or secretions, including semen. Nosocomial transmission has also been reported through contaminated needles and syringes.

Period of communicability

Communicability of viral haemorrhagic fevers depends on the infective agent:

- Lassa fever virus is communicable via person to person spread during the acute febrile phase. Virus is excreted in the urine for up to three to nine weeks from the onset of the illness.
- Crimean-Congo haemorrhagic fever virus communicability is unknown. The virus is highly infectious in the hospital setting where it has been transmitted to health care personnel by accidental needle stick injury.
- Marburg and Ebola virus are communicable as long as blood and secretions contain virus. Virus has been isolated in seminal fluid 60 days after the onset of infection.

Susceptibility and resistance

All ages are susceptible. The duration of immunity after infection is unknown.

Control measures

Preventive measures

Not applicable in Australia. No vaccines are available.

Intending travellers to LF and CCHF endemic areas should avoid contact with ticks and rodents.

Control of case

All travellers who arrive in Australia with any risk of contracting quarantinable VHF should be immediately notified to the Department of Human Services.

The Department of Human Services state chief quarantine officer will make any decisions concerning patient's assessment, transport and quarantine.

All patients should be cared for at the hospital where they are first seen (if possible), or transferred to the Victorian Infectious Diseases Service at the Royal Melbourne Hospital, the designated VHF treatment centre for Victoria, or similarly equipped tertiary hospital.

Intravenous ribavirin may be useful for treatment purposes; a stock of this drug is maintained at a number of tertiary hospitals including the Royal Melbourne Hospital.

Cases should be cared for in an isolation room, preferably with negative pressure ventilation, and non-essential staff and visitors should be restricted. The highest level of barrier infection control precautions should be instituted including gowns, gloves, face shields and masks.

No airborne transmission has been reported so personal and room HEPA filtration is not an absolute requirement but should be used if available. An anteroom for putting on and discarding clothing and storing supplies is advisable.

The obligatory period of isolation for a proven case of viral haemorrhagic fever is a minimum of two days without fever and a total of 21 days from onset of illness.

Convalescent patients and their contacts should be informed that VHF viruses may be excreted for many weeks in semen (MV and EV) and in urine (LF). Meticulous personal hygiene is necessary. Abstinence from sexual intercourse is advised until genital fluids have been shown to be free of the virus.

Post-mortem is discouraged. Bodies of deceased patients should preferably be cremated.

Control of contacts

Active case and contact surveillance is conducted by the Department to identify any fellow cases and all contacts with the patient from the 21 days after the onset of symptoms. A contact is a person who has been exposed to an infected person or to an infected person's secretions or tissues within three weeks of the patient's onset of illness.

Contacts are further classified as casual contacts, close contacts or high risk contacts.

Casual contacts are those people with no direct contact with the patient but who have been in the near vicinity, such as on the same aeroplane or in the same hotel. No special surveillance is required although information on the disease and symptoms may be distributed.

Close contacts are defined as those living with the patient, nursing and hugging the patient, or handling the patient's laboratory specimens. If the diagnosis is confirmed, close contacts are placed under self-surveillance with twice daily recording of body temperature.

High risk contacts are those with a history of either mucous membrane contact with the patient (kissing, sexual intercourse), or needle-stick or other penetrating injuries contaminated with blood or other body fluids from the patient during their infectious period. These contacts should be placed under self-surveillance as soon as VHF is considered to be a likely diagnosis in the index patient.

Any close or high risk contact that develops a fever (> 38°C) or any other symptoms of illness should be immediately isolated and treated as a VHF patient.

Ribavirin may be prescribed as post-exposure prophylaxis for high risk contacts of patients.

Control of environment

All potentially contaminated personal items and items used in the treatment of the patient should be disinfected with an appropriate viricide such as 0.5% hypochlorite or 0.5% phenol with detergent, and as far as possible, subjected to heating by incineration, autoclaving or boiling by appropriately protected staff. Room disinfection should be performed using the same virucidal disinfectants.

These disinfection measures may apply to the patient's place of residence and other environments where the patient has spent a significant period of time while symptomatic, such as aircraft and hotel rooms.

Outbreak measures

A single case of any of these viral haemorrhagic fevers in any setting would constitute an outbreak and requires the clinical and public health control measures as outlined above.

International measures

In the event of a suspected or confirmed case of any of these viral haemorrhagic fevers, the Department would immediately notify the Commonwealth Chief Medical Officer who would in turn notify WHO according to International Health Regulations, as well as notify the source country and other countries who may receive possible exposure by infected travellers.

Close and high risk contacts should be discouraged from travel during their period of surveillance.

Additional sources of information

- Australian Government Department of Health and Ageing, quarantine and travel health information, http://www.health.gov.au
- Centers for Disease Control and Prevention, Atlanta USA, *Public health emergency preparedness and response*, http://www.bt.cdc.gov

Yellow fever

Victorian statutory requirement

Yellow fever virus (Group A disease) must be notified immediately by telephone or fax followed by written notification within five days.

Any health care provider who suspects yellow fever should immediately contact the Chief Quarantine Medical Officer.

School exclusion is not applicable.

Yellow fever is subject to Australian quarantine.

Infectious agent

Yellow fever virus (YFV) is a member of the flavivirus group.

Identification

Clinical features

Yellow fever is an acute viral disease of short duration. In mild cases, the only symptoms may be headache and fever or a 'dengue-like' illness with fever, chills and myalgia. Sometimes the infection may be inapparent.

More severe disease occurs in a small percentage of cases and is characterised by three stages:

- stage one has fever, chills, backache, myalgia, nausea, vomiting and epistaxis. Faget's sign of relative bradycardia with a high temperature with a slow pulse appears on the second day. On the third day the fever falls and the patient enters remission.
- stage two is a remission that may last several hours to several days. Haemorrhages, anuria and delirium may occur without remission.
- stage three shows development of the classic features of jaundice and haemorrhagic manifestations such as epistaxis, haematemesis, melaena and uterine bleeding followed by albuminuria, coma and death two to three days later.

Method of diagnosis

The diagnosis is based on the presence of laboratory, clinical and epidemiological evidence.

Laboratory evidence includes one of the following:

- isolation of YFV from clinical material
- detection of YF viral RNA in clinical material
- seroconversion with a significant rise in IgG level to YFV
- YFV specific IgM detected in the absence of IgM to other flaviviruses.

Yellow fever virus specific IgG on a single specimen confirmed by neutralisation and where cross-reactions with other flaviviruses have been excluded is suggestive of infection and should be viewed in the context of clinical and epidemiological evidence.

All clinical specimens should be transferred immediately to the National High Security Quarantine Laboratory (NHSQL) at Victorian Infectious Diseases Reference Laboratory (VIDRL) as per national quarantine guidelines. VIDRL should be contacted on (03) 9342 2600 to discuss requirements for confirmatory tests or for interpretation of laboratory results. Cross-reactivity with other flaviviruses can occur.

Clinical evidence

Clinical evidence includes acute onset with fever and jaundice and other possible manifestations of the disease.

Epidemiological evidence

Epidemiological evidence includes a history of travel to a yellow fever endemic country in the preceding six days and no history of vaccination with yellow fever vaccine in the preceding two months.

A person with a febrile illness who has been in a yellow fever area within the previous six days is considered a suspected case and should be reported immediately.

Incubation period

The incubation period is three to six days.

Public health significance and occurrence

Yellow fever is endemic in tropical areas of South America and Central Africa. Outbreaks may occur in unaffected areas if mosquitoes are infected by migrating humans or monkeys infected with the virus.

Ae. aegypti is widely distributed in Queensland. The introduction of yellow fever virus to the Australian mosquito population could theoretically result in an urban outbreak of human disease. No other mosquito species in Australia are considered likely to be competent vectors.

Reservoir

In urban areas of endemic countries the reservoirs are humans and Aedes mosquitoes. In forest areas, invertebrates (other than humans), mainly monkeys and possibly marsupials, and forest mosquitoes are the reservoir. The viraemic period in monkeys and man is too short for monkeys to act as a reservoir.

Humans have no essential role in transmission of jungle yellow fever but are the primary amplifying host (in the urban cycle).

Mode of transmission

Yellow fever is transmitted via infected mosquitoes. Mosquitoes become infectious 9–12 days after a blood meal from a viraemic host. Human to human transmission has not been documented.

Period of communicability

Human blood is infective for mosquitoes shortly before the onset of fever and for three to five days after. Mosquitoes require nine to 12 days after a blood meal to become infectious and remain so for life.

Susceptibility and resistance

Mild infections are common in endemic areas. Previous infection with dengue gives some degree of immunity, and passive immunity in infants born to immune mothers may last for six months. Infections confer lifelong immunity.

Control measures

Preventive measures

All travellers to endemic areas in Africa and South America should be immunised. Certification of yellow fever vaccination is required for travellers over one year of age entering Australia within six days of leaving an infected country. A yellow fever vaccination certificate is valid for ten years and begins ten days after vaccination. Vaccine providers in Victoria must be accredited with the Department of Human Services.

Control of case

In Victoria suspected or confirmed cases that require inpatient treatment should be referred to the Victorian Infectious Diseases Service at the Royal Melbourne Hospital where adequate facilities for isolation are available if required. This is of particular concern in suspected cases where the differential diagnosis may include other viral or haemorrhagic fevers with greater potential for person to person spread.

There are no endemic foci of yellow fever vectors in Victoria however infection of Victorian mosquitoes is a theoretical risk. Therefore, the case should be protected from exposure to mosquitoes for greater than five days after onset of infection. The case should be cared for in an isolation room or in a screened room with use of a mosquito net and a suitable knock down spray for mosquitoes if not in hospital.

Control of contacts

If a traveller to Australia is diagnosed with yellow fever and has been potentially exposed to Australian Ae. aegypti mosquitoes during the period of viraemia or if the first recognised case is indigenous, then the following measures should be considered:

Spray inside and around the home of the patient, and all houses within a half a kilometre radius, with an effective insecticide to eliminate vectors. Potential vector breeding sites should be destroyed, emptied or sprayed within this area.

Contacts of the patient who have not previously been immunised should be offered yellow fever vaccine. Other cases of mild febrile illness and any unexplained deaths possibly consistent with yellow fever should be investigated.

Australian Quarantine and Inspection Service officers routinely place travel companions of the case under quarantine surveillance on entry into Australia for six days since last staying over night in a country where yellow fever may be present. During this period they are required to notify the Chief Quarantine Medical Officer if suffering from a febrile illness.

Control of environment

Ae. aegypti mosquitoes should be eliminated near airports. Insect quarantine should be maintained to prevent the introduction of Ae. albopictus, a prevalent Asian species which is capable of transmitting yellow fever.

Outbreak measures

A single case of indigenous transmission constitutes an outbreak. In the event of an epidemic of yellow fever in an urban area, all persons living in the area infested with *Ae. aegypti* should be offered yellow fever vaccine and a wider mosquito spraying and breeding site elimination program implemented.

International measures

Yellow fever must be notified to the World Health Organization under the International Health Regulations (1969).

Additional sources of information

- Australian Government Department of Health and Aged Care 1999, *Guidelines for the management of human quarantine disease in Australia*.
- Victorian Department of Human Services, for information on yellow fever vaccination clinics in Victoria phone the Immunisation Unit 1300 882 008 or go to www.health.vic.gov.au/ideas.
- World Health Organization, http://www.who.int/emc/topics

Appendix 1: Contacts

Organisation	Telephone	Fax	Internet	After hours contact
Department of Human Services, Victoria				
Communicable Diseases Section	1300 651 160 (03) 9637 4126	(03) 9637 4477 general 1300 651 170 for notifications	www.health.vic.gov.au/ideas www.betterhealth.vic.gov.au	132222 pager no. 46870
Melbourne Sexual Health Centre	(03) 9347 0244	(03) 9347 2230	www.mshc.org.au	
Laboratories				
Microbiological Diagnostic Unit	(03) 9344 5713	(03) 9344 7833	www.microbiol.unimelb.edu.au	
Victorian Infectious Diseases Reference Laboratory	(03) 9342 2600	(03) 9342 2666	www.vidrl.org.au	(03) 9342 2600
Interstate health departments				
Australian Capital Territory: Communicable Disease Control	(02) 6205 2155	(02) 6205 0711	www.health.act.gov.au	Pager (02) 6269 0495
New South Wales	(02) 9391 9000	(02) 9391 9101	www.health.nsw.gov.au	
Northern Territory: Disease Control	(08) 8922 8044	(08) 8922 8310	www.nt.gov.au/health	Hospital switchboard (08) 8922 8888
Queensland: Communicable Diseases Unit	(07) 3234 1155	(07) 3234 0057	www.health.qld.gov.au	
South Australia: Communicable Disease Control	(08) 8226 7177	(08) 8226 7187	www.health.sa.gov.au	(08) 8226 7177
Tasmania: Public and Environmental Health Services	1800 671 738 (03) 6233 3203	(03) 6222 7407	www.dhhs.tas.gov.au	1800 671 738
Western Australia: Communicable Disease Control	(08) 9388 4999	(08) 9388 4888	www.population.health.wa.gov.au	(08) 9388 4999
Other government departments				
Food Standards Australia New Zealand	(02) 6271 2222	(02) 6271 2278	www.foodstandards.gov.au	
Australian Government Department of Health and Ageing	(02) 6289 1555	(02) 6281 6946	www.health.gov.au	(02) 6122 2747
Victorian Department of Primary Industries	136 186		www.dpi.vic.gov.au	

Appendix 2: Glossary

airborne transmission
Transmission by air of infectious agents from respiratory secretions.

asymptomatic infection
Infection that does not display any clinical symptoms, but may still be capable of transmitting disease.

carrier
A person or animal that harbours a specific infectious agent in the absence of clinical disease and serves as a potential source of infection.

communicable disease
A disease capable of being transmitted from an infected person or species to a susceptible host, either directly or indirectly.

concurrent disinfection
Immediate disinfection and disposal of discharges and infective matter all through the course of a disease.

contact
A person or animal that has associated with an infected person or animal that might provide an opportunity to acquire the infection.

disinfection
Killing of infectious agents outside the body by direct exposure to chemical or physical agents. High level disinfection refers to the inactivation of all microorganisms except some bacterial spores.

drainage/secretion precautions
Precautions used to prevent infections transmitted by direct or indirect contact with purulent material or drainage from an infected body site.

droplet transmission
Transmission of infectious agents in droplets from respiratory secretions.

endemic
The constant presence of a disease or infectious agent within a given geographic area.

epidemic
The occurrence of a number of cases of a disease (or condition) in excess of a number expected in a given time and place. In some instances a single case will constitute such an unusual occurrence.

fomes (plural fomites)
An object such as a book, wooden object, or an article of clothing that is not harmful in itself, but is able to harbour pathogenic microorganisms and thus may serve as an agent of transmission of an infection.

immunity
The protection against infectious disease generated by immunisation, previous infection or by other nonimmunologic factors.

inapparent infection
The presence of infection in a host without recognisable clinical signs or symptoms.

incubation period
The time interval between initial contact with an infectious agent and the appearance of clinical signs and symptoms.

infection
Invasion and multiplication of micro-organisms in body tissues.

infectious agent
An organism that is capable of producing infection or infectious disease.

infestation
The lodgement, development and reproduction of arthropods on the surface of the body of persons or animals or in clothing.

isolation
Represents separation for the period of communicability, of infected persons or animals from others in such places and under such conditions as to prevent or limit the direct or indirect transmission of the infectious agent. Categories of isolation include:

- *strict isolation:* for highly contagious infections spread by air and contact
- *contact isolation:* for diseases spread primarily by close or direct contact
- *respiratory isolation:* to prevent transmission over short distances through the air.

For drainage/secretion precautions see separate entry. For blood and body substance precautions, see appendix 3.

nosocomial infection
Hospital-acquired infection.

notifiable disease
Disease or condition that is required by law to be notified to the State health department.

notification
The process of reporting a notifiable infectious disease.

outbreak
See epidemic.

period of communicability
The time during which an infectious agent may be transferred directly or indirectly from an infected person or animal to a susceptible host.

personal hygiene
The protective measures within the responsibility of the individual that limit the spread of infectious diseases.

personal protective equipment
The equipment to be worn when performing duties that may involve possible occupational exposure to blood, splashing or aerosols from cleaning processes – for example, masks, goggles, gloves and aprons.

quarantine
The restriction of freedom of movement of apparently healthy individuals who have been exposed to infectious disease.

reservoir of infectious agents
Any person, animal, or substance in which an infectious agent normally lives and multiplies in such a manner that it can be transmitted to a susceptible host.

resistance
The natural ability of an organism to resist micro-organisms or toxins produced in disease.

school exclusion
Exclusion from school or children's services centre under Health (Infectious Diseases) Regulations 2001.

source of infection
The person, animal or substance from which an infectious agent passes to a host.

surveillance
Personal surveillance is the practice of close medical or other supervision of contacts to permit prompt recognition of infection or illness but without restricting the movements of the individual.

susceptibility
Lack of resistance to a particular pathogenic agent.

transmission
In terms of infection, it relates to any mechanism by which an infectious agent is spread from a source or reservoir to a person. This may be direct or indirect (that is, vehicle-borne, vector-borne, or airborne).

standard precautions
Work practices that require everyone to assume that all blood and body fluids are potential sources of infection, independent of perceived risk. Such precautions involve the use of safe work practices and protective barriers, and the safe disposal of body substances and soiled material. See appendix 3.

vector
A carrier, especially the animal (usually an arthropod) that transfers an infective agent from one host to another.

zoonosis
A disease of animals that may be transmitted to humans under natural conditions.

Appendix 3: Standard and additional precautions

General

Infection control and prevention uses a risk management approach to minimise or prevent the transmission of infection. Standard and additional precautions principles and practice are based on the mode of transmission of an infectious agent.

Standard precautions are work practices required for the basic level of infection control. They include good hygiene practices, particularly washing and drying hands before and after patient contact, the use of protective barriers which may include gloves, gowns, plastic aprons, masks, eye shields or goggles, appropriate handling and disposal of sharps and other contaminated or clinical (infectious) waste, and use of aseptic techniques.

Standard precautions apply to all patients regardless of their diagnosis or presumed infection status, and in the handling of:

- blood
- all other body fluids, secretions and excretions (except sweat), regardless of whether they contain visible blood
- non-intact skin
- mucous membranes (mouth and eyes)
- standard precautions also apply to dried blood and other body substances, including saliva.

Standard precautions should be considered minimum requirements for infection control. Implementing standard precautions minimises the risk of transmission of infection from person to person even in high-risk situations.

Standard precautions should be implemented at all times particularly when patients are undergoing invasive procedures, including catheterisation, cannulation or intubation. Health services that offer these procedures should provide detailed protocols for patient management in their infection control procedures manuals.

Additional precautions are work practices that should be applied in a health care setting for patients known, or suspected, to be infected or colonised with infectious agents that may not be contained using standard precautions alone.

Standard precautions

The use of standard precautions is essential as the **primary** strategy for the successful minimisation of transmission of health care associated infection because:

- infectious patients may not show any signs or symptoms of infection that may be detected in a routine history and medical assessment
- a patient's infectious status is often determined by laboratory tests that may not be completed in time to provide emergency care
- patients may be infectious before laboratory tests are positive or symptoms of disease are recognised (the window period of disease)
- people may be placed at risk of infection from those who are asymptomatic but infectious.

Standard precautions for infection control in health care settings consist of the following work practices:

- aseptic technique for all invasive procedures, including appropriate use of skin disinfectants
- personal hygiene practices, particularly hand washing and drying before and after all significant patient contacts
- the use of 70% alcohol-based chlorhexidine (0.5%) hand rub solutions as an adjunct to hand washing
- use of personal protective equipment, which may include gloves, impermeable gowns, plastic aprons, masks/face shields and eye protection
- appropriate handling and disposal of sharps and other clinical (infectious) waste
- appropriate reprocessing of reusable equipment and instruments, including appropriate use of disinfectants
- environmental controls, including design and maintenance of premises, cleaning and spills management including appropriate use of disinfectants
- appropriate provision of support services such as laundry and food services.

Additional precautions

Additional Precautions are used for patients known or suspected to be infected or colonised with epidemiologically important or highly transmissible pathogens that can transmit/cause infection by the following means:

- *airborne transmission* (e.g. pulmonary tuberculosis, chickenpox, measles)
- *droplet transmission* of respiratory secretions (e.g. rubella, pertussis, influenza)
- *contact transmission* (direct or indirect) with patients who may be disseminators of infectious agents of special concern (e.g. the dry skin of those colonised with Multi-resistant Staphylococcus aureus [MRSA], faecal contamination from carriers of vancomycin-resistant enterococci [VRE] or contaminated surfaces)
- *inherent resistance* to standard sterilisation procedures or other disease-specific means of transmission where standard precautions are not sufficient (e.g. patients with known or suspected Creutzfeldt-Jakob disease)
- *any combination* of these routes.

Additional precautions are designed to interrupt transmission of infection by these routes and should be used, in addition to standard precautions, when standard precautions alone might not contain transmission of infection. Additional precautions may be specific to the situation for which they are required, or may be combined where microorganisms have multiple routes of transmission.

Additional precautions should be tailored to the particular infectious agent involved and the mode of transmission, and may include one or any combination of the following:

- allocation of a single room with ensuite facilities
- a dedicated toilet (to prevent transmission of infections that are transmitted primarily by contact with faecal material, such as for patients with infectious diarrhoea or gastroenteritis caused by enteric bacteria or viruses)
- cohorting (room sharing by people with the same infection) may be an alternative if single rooms are not available
- special ventilation requirements (e.g. monitored negative air pressure in relation to surrounding areas)
- additional use of personal protective equipment (e.g. health care workers attending to patients in respiratory isolation should wear a well-fitting mask: a 0.3-mm particulate filter mask (P2 or N95 mask) is recommended for tuberculosis)
- rostering of immune health care workers to care for certain classes of infectious patients (eg chickenpox)
- dedicated patient equipment
- restricted movement of both patients and health care workers.

Additional precautions are not required for patients with bloodborne viruses, such as HIV, hepatitis B virus or hepatitis C virus, unless there are complicating infections, such as pulmonary tuberculosis.

To minimise the exposure time of other people in office practices or hospital waiting rooms, people identified as ‘at risk’ of transmitting droplet or airborne diseases (e.g. a child with suspected chicken pox) should be subject to additional precautions including isolation and should be attended to before other people waiting for treatment.

An outline of the application of additional precautions for infections with airborne, droplet or contact transmission is shown in the following table.

Outline of requirements for specified categories of additional precautions

Requirement	Additional precautions by transmission route		
	Airborne	**Droplet**	**Contact**
Gloves	Nil	Nil	For all manual contact with patient, associated devices and immediate environmental surfaces
Impermeable apron/gown	Nil	Nil	When health care worker's clothing is in substantial contact with the patient, items in contact with the patient, and their immediate environment
Respirator or mask. Refer to AS 4381:2000 for additional information	P2/N95 particulate respirator for tuberculosis only. All others, use face mask suited to the purpose such as a mask that filters to o.1 microns and has a splash resistant shield.	Yes –mask*	Protect face if splash likely
Goggles/face-shields	Protect face if splash likely	Protect face if splash likely	Protect face if splash likely
Special handling of equipment	As per standard precautions	As per standard precautions	Single use or reprocess before reuse on next patient (includes all equipment in contact with patient)
Single room	Yes or Cohort patients with same infection. Door closed.	Yes or Cohort patients with same infection. Door closed.	If possible, or cohort with patient with the same infection (eg methicillin-resistant Staphylococcus aureus)
Negative pressure	Essential for pulmonary TB	No	No
Transport of patients	Appropriate mask* for patient Notify area receiving patient	Appropriate mask* for patient Notify area receiving patient	Notify area receiving patient
Other	Encourage patients to cover nose and mouth when coughing or sneezing and wash their hands after blowing nose. Provide one metre of separation between patients in ward accommodation	Provide one metre of separation between patients in ward accommodation	Remove gloves and gown, and wash hands before leaving patient's room

* Refer to Australian Standards:
AS 4381:2000 Single-use face masks for use in health care, for additional information
AS/NZS 1715 Selection, use and maintenance of respiratory protective devices
AS/NZS 1716:1994 Respiratory protective devices

Handwashing

- Wash and dry hands after touching blood, body fluids, secretions, excretions and contaminated items such as equipment or instruments, regardless of whether gloves are worn or not.
- Wash and dry hands immediately after gloves are removed, after significant patient contact such as contact with or physical examination, emptying drainage bags, undertaking venepuncture or delivery of an injection or going to the toilet.
- Wash and dry hands following any activities that may transfer microorganisms to other patients or environments.
- Use plain liquid soap for routine hand washing. Antimicrobial liquid soap solutions are required for invasive procedures and in some situations such as those patients with VRE and MRSA.
- A 70% alcohol-based chlorhexidine (0.5%) hand rub solution may be used as an adjunct to handwashing and in situations where water is not readily available.

Personal protective equipment

The use of personal protective equipment (PPE) protects the health care worker and others from exposure to blood and body fluids/substances. PPE that complies with relevant Australian Standards should be readily available and accessible in all health services.

Gloves

- Wear gloves (clean non sterile gloves are adequate) when touching blood, body fluids, secretions, excretions and contaminated items; put on clean gloves just before touching mucous membrane and non-intact skin. Sterile gloves are required for invasive procedures.
- Change gloves between tasks and procedures on the same patient after contact with material that may contain a high concentration of microorganisms.
- Remove gloves promptly after use, before touching non-contaminated items and environmental surfaces and before going to another patient. Dispose of gloves in the clinical (infectious) waste or place in a plastic bag and tie before disposing of it in the general household waste.
- Wash and dry hands immediately after removing gloves to avoid transfer of microorganisms to other patients or environments.

Gowns

- Wear a gown (a clean non-sterile gown is adequate) to protect skin and prevent soiling of clothing during procedures and patient care activities that are likely to generate splashing or sprays of blood, body fluids, secretions, or excretions or cause soiling of clothing.
- Select a gown (long- or short –sleeved) that is appropriate for the activity and the amount of fluid likely to be encountered.
- Remove the used gown as promptly as possible using gloved hands, roll up carefully and place in a linen receptacle for laundering.
- Wash and dry hands to avoid transfer of microorganisms to other patients and environments.

Masks, eye protection, faceshields

Wear a mask and eye protection or a faceshield to protect mucous membranes of the eyes, nose and mouth:

- during procedures and patient-care activities that are likely to generate splashes or sprays of blood, body fluids, secretions and excretions
- during cleaning activities.

Remove the mask by holding the ties only and dispose of the mask into a clinical waste bin.

Reusable face shields or goggles should be removed carefully and placed in a receptacle for cleaning.

Waterproof aprons

Wear waterproof aprons when splashes or sprays of blood or body fluids/ substances are likely such as during cleaning activities.

Remove the used apron as promptly as possible using gloved hands, roll up carefully and place in a clinical waste bin.

Environmental control

Ensure that the health service has adequate procedures for the routine care, cleaning, and disinfection of environmental surfaces, beds, bedrails, bedside equipment, and other frequently touched surfaces and that these procedures are being followed. See Appendix 6 for cleaning and waste disposal.

Further information

- Australian Government Department of Health and Ageing 2004, *Infection control guidelines for the prevention of transmission of infectious diseases in the health care setting*, http://www.icg.health.gov.au/
- Victorian Department of Human Services 2000, *Sure protection against infection*, www.health.vic.gov.au/ideas

Appendix 4: Procedure for managing an exposure to blood/body fluids/substances

These include sharps injuries (including needlestick) and splashes into/onto mucous membranes or bare intact skin.

Occupational hazards for health care workers from sharps injuries (including needlestick injury) and other blood or body fluid incidents include human immunodeficiency virus (HIV), hepatitis B virus (HBV) and hepatitis C virus.

Exposure is an injury that involves direct skin contact with a body fluid listed above and there is compromised skin integrity such as an open wound, abrasion or dermatitis, or if there is direct mucous membrane contact. For exposure to skin, the larger the area of skin exposed and the longer the time of contact, the more important it is to verify that all the relevant skin area is intact.

Exposure to blood and body fluids/substances

The following body fluids pose a risk for bloodborne virus transmission:

- blood, serum, plasma and all biological fluids visibly contaminated with blood
- laboratory specimens that contain concentrated virus
- pleural, amniotic, pericardial, peritoneal, synovial and cerebrospinal fluids
- uterine/vaginal secretions or semen.

Infection control protocols

All health services must develop their own infection control protocols for communicable diseases, including clear written instructions on the appropriate action to take in the event of an exposure to blood or body fluids/substances including needlestick injuries and other blood or body fluid incidents involving either patients or health care workers, including:

- the physician, medical officer or other suitably qualified professional to be contacted
- the laboratory which will process emergency specimens
- the pharmacy which stocks prophylactic medication
- procedures for investigating the circumstances of the incident and measures to prevent recurrence (this may include changes to work practices, changes to equipment, and/or training)
- details for prompt reporting, evaluation, counselling, treatment and follow-up of occupational exposures to bloodborne viruses.

Immediate action

Treatment protocols should include removal of contaminated clothing and thorough washing of the injured area with soap and water. Affected mucous membranes should be flushed with large amounts of water. Eyes should be flushed gently.

The exposed person must report any occupational exposures immediately.

The exposed person should have a medical evaluation, including information about medications they are taking and underlying medical conditions or circumstances. Postexposure prophylaxis (PEP and N-PEP) and counselling should be available and offered. Treatment should be available during all working hours, and on call after hours (e.g. through an on-call infectious diseases physician).

Patients or others exposed to blood or other body fluids/substances must be informed of the exposure by a designated professional, while maintaining confidentiality about the source of the blood. Baseline serum should be collected from the patient and expert counselling provided on the implications of what has happened. Postexposure prophylaxis (PEP and N-PEP) and appropriate long-term follow-up should be offered where applicable. Patient or source refusal for testing and serum storage should be documented.

Australian National Council for AIDS, Hepatitis C and Related Diseases (ANCAHRD) has published a comprehensive bulletin entitled *Management of exposure to blood/body fluids in a health care setting*, available at http://www.ancahrd.org/.

Document the incident and include:

- date, time and type of exposure
- how the incident occurred
- name of the source individual (if known)

Exposure incidents that do not occur in a health service should be reported to a general medical practitioner or the Emergency Department at the nearest hospital.

Management of the source individual

The person whose blood or body fluids are the source of an occupational/non-occupational exposure or other injury should be evaluated for infection with HIV, HBV and HCV. Information available in the medical record or from the source person may suggest or rule

out infection with each virus. If the source is known to have HIV infection, then information on stage of infection and current and previous antiretroviral therapy should be gathered and used in deciding the most appropriate regimen of post exposure prophylaxis (PEP).

If the HIV, HBV or HCV status of the source person is unknown, then the source person should be informed of the incident, and their consent sought to test for these viruses, with appropriate pre- and post-test counselling. Their consent to having the information in their patient record used should be sought also. If consent cannot be obtained, for example if the patient is unconscious or unwilling to consent, then procedures should be followed which comply with legislation in Victoria.

The source individual should be tested as follows at the time of injury:

- HIV antibody
- HBsAg
- HCV antibody.

If the HCV antibody test is positive, then HCV polymerase chain reaction (PCR) should be performed to test for HCV RNA. Transmission is much less likely to occur from a source who is PCR negative. The status of the source individual may be known at the time of the incident. In this case the affected person should be managed as described below under 'immediate management'. If the source is unknown, the case should be managed as described below.

Source individual unknown

Reasonable efforts should be made to identify the source. If the source remains unknown, appropriate follow-up should be determined on an individual basis depending on:

- the type of exposure
- the likelihood of the source being positive for a blood pathogen
- the prevalence of HIV, HBV and HCV in the community of the likely source on whom the instrument or needle was used.

Management of the exposed person

Immediate care of the exposure site

Contaminated clothing should be removed, and the injured area should be washed well with soap and water (an antiseptic could also be applied). Any affected mucous membranes should be flushed with large amounts of water. If the eyes are contaminated, they should be rinsed gently but thoroughly with water or normal saline, while kept open.

Evaluation of the exposure

The exposed person should be examined to confirm the nature of exposure and counselled about the possibility of transmission of bloodborne disease.

Evaluation and testing of the exposed person

The exposed person should have a medical evaluation, including information about medications they are taking and underlying medical conditions or circumstances. All exposed people should be assessed to determine the risk of tetanus.

Depending on the circumstances of the exposure, the following may need to be considered:

- tetanus immunoglobulin
- a course of adsorbed diphtheria tetanus vaccine, adult formulation (Td) vaccine
- Td booster.

The current edition of *The Australian immunisation handbook* should be consulted for further details.

The exposed person would normally be tested for HIV antibody, HCV antibody and antibody to HBV surface antigen (HBsAg) at the time of the injury, to establish their serostatus at the time of the exposure. Expert counselling on the implications of the event, PEP (post exposure prophylaxis) and appropriate long-term follow-up should be offered.

An option that may be offered to health care workers who do not wish to undergo testing at the time of the exposure is to have blood collected and stored but not tested. Blood that is collected and stored for this purpose must be retained for a minimum period of 12 months.

If the source person is found to be HIV, HBV and HCV negative, no further follow-up of the exposed person is generally necessary, unless there is reason to suspect the source person is seroconverting to one of these viruses, or was at high risk of bloodborne viral infection at the time of the exposure. If source is positive for one of these viruses, pregnancy testing should be offered to women of child-bearing age who have been exposed and whose pregnancy status is unknown.

Postexposure counselling

A specialist with knowledge of bloodborne infections should undertake follow-up. If it is demonstrated that a person has been exposed to a bloodborne pathogen, they should not donate blood, semen, organs or tissue for six months, and should not share implements that may be contaminated with even a small amount of blood (e.g. razors or toothbrushes).

For HIV and HBV, they should be informed of the risk of transmission to sexual and injecting partners for a six-month period, and be counselled about issues of safe sex and safe injecting. If PEP is indicated, or there is a risk of acute infection with HIV, HCV or HBV, advice should be offered on pregnancy and breastfeeding based on an individual risk assessment. In the case of HIV, patients should be advised of the remote risk of seroconversion up to 12 months post-exposure, particularly if specific PEP was undertaken.

Follow-up for the exposed person

If the source person is seronegative for HIV, HbsAg and HCV, baseline testing or further follow-up of the health care worker normally is not necessary. If the source person has recently engaged in behaviours that are associated with a risk for transmission of these viruses, baseline and follow-up HIV-antibody testing of the health care worker should be considered.

Management of exposure to blood/body fluids summary table

When	What
Immediately after exposure	First aid Relief from duty Risk assessment Post exposure prophylaxis (PEP) – if significant injury
As soon as possible (same day)	Source assessment Documentation of exposure Prevention of transmission and exposure/pre-test counselling Baseline serology if agreed to Referral to specialist physician - if PEP commenced Support of significant others
1-3 weeks	Post-test counselling with results of baseline serology Occupational health and safety review
3 months	Pre HIV test counselling Follow up serology – HIV, HBV, HCV
6 months	Follow up serology – HBV, HCV – HIV (if PEP taken)

Appendix 5: Procedure for managing spills of blood and body fluids/substances

Health services should have management systems in place for dealing with blood and body substance spills and protocols should be included in procedural manuals and emphasised in ongoing education or training programs. The basic principles of blood and body fluid/substance spills management are:

- standard precautions apply, including use of personal protective equipment (PPE) as applicable
- spills should be cleared up before the area is cleaned (adding cleaning liquids to spills increases the size of the spill and should be avoided)
- generation of aerosols from spilled material should be avoided.

Using these basic principles, the management of spills should be flexible enough to cope with different types of spills, taking into account the following factors:

- the nature (type) of the spill (e.g. sputum, vomit, faeces, urine, blood or laboratory culture)
- the pathogens most likely to be involved in these different types of spills (e.g. stool samples may contain viruses, bacteria or protozoan pathogens whereas sputum may contain Mycobacterium tuberculosis)
- the size of the spill (e.g. spot [few drops], small [<10cm] or large [>10cm])
- the type of surface (e.g. carpet or impervious flooring)
- the location involved i.e. whether the spill occurs in a contained area such as a microbiology laboratory or in a public or clinical area of a health service, in a public location or within a community premises
- whether there is any likelihood of bare skin contact with the soiled (contaminated) surface.

Equipment

Standard cleaning equipment, including a mop and cleaning bucket and cleaning agents, should be readily available for spills management and should be stored in an area known to all. This is particularly important in clinical areas. To facilitate the management of spills in areas where cleaning materials may not be readily available, a disposable 'spills kit' could be used, containing the following items:

- a large (10 L) reusable plastic container or bucket with fitted lid, containing the following items
- appropriate leak proof bags and containers for disposal of waste material
- a designated, sturdy scraper and pan for spills (similar to a 'pooper scooper')
- about five sachets of a granular formulation containing 10,000 ppm available chlorine or equivalent (each sachet should contain sufficient granules to cover a 10-cm diameter spill)
- disposable rubber gloves suitable for cleaning (vinyl gloves are not recommended for handling blood)
- eye protection (disposable or reusable)
- a plastic apron
- a respiratory protection device (for protection against inhalation of powder from the disinfectant granules, or aerosols, which may be generated from high-risk spills during the cleaning process).

Single-use items in the spills kit should be replaced after each use of the spills kit.

With all spills management protocols, it is essential that the affected area is left clean and dry.

Sodium hydroxide (caustic soda) spills kits should be available for areas at risk for higher-risk CJD spills, such as neurosurgery units, mortuaries and laboratories.

Procedures

In clinical areas blood and body fluid/substance spills should be dealt with as soon as possible. In operating rooms, or in circumstances where medical procedures are under way, spills should be attended to as soon as it is safe to do so.

Care should be taken to thoroughly clean and dry areas where there is any possibility of bare skin contact with the surface (e.g. on an examination couch).

Personal protective equipment (PPE) should be used for all cleaning procedures and disposed of or sent for cleaning after use. Hands should be washed and dried after cleaning.

Where a spill occurs on a carpet, shampoo as soon as possible. Do not use disinfectant. Steam cleaning may be used instead.

Wash hands thoroughly after cleaning is completed.

Spots or small spills

Spots or drops of blood or other small spills (up to 10cms) can easily be managed by wiping the area immediately with paper towelling and then cleaning with warm water and detergent followed by rinsing and drying the area. Dry the area as wet areas attract contaminants.

A hospital grade disinfectant can be used on the spill area after cleaning.

Large spills

Where large spills (over 10cms) have occurred in a 'wet' area, such as a bathroom or toilet area, the spill should be carefully washed off into the sewerage system using copious amounts of water and the area flushed with warm water and detergent.

Large blood spills that have occurred in 'dry' areas (such as clinical areas) should be contained and generation of aerosols should be avoided.

Granular formulations that produce high available chlorine concentrations can contain the spilled material and are useful for preventing aerosols. A scraper and pan should be used to remove the absorbed material. The area of the spill should then be cleaned with a mop and bucket of warm water and detergent. The bucket and mop should be thoroughly cleaned after use and stored dry.

Use of sodium hypochlorite (bleach)

It is generally unnecessary to use sodium hypochlorite for managing spills but it may be used in specific circumstances. It is recognised, however, that some health care workers/members of the public may feel more reassured that the risk of infection is reduced if sodium hypochlorite is used. Health care workers and members of the public should be aware that there is **no** evidence of benefit from an infection control perspective.

Hypochlorites are corrosive to metals and must be rinsed off after 10 minutes and the area dried.

Creutzfeldt–Jakob disease (CJD)

If a spill of tissue (potentially) infected with Creutzfeldt–Jakob disease (CJD) occurs (eg brain tissue), the contaminated item should either be destroyed by incineration or immersed in either sodium hydroxide or sodium hypochlorite for one hour, rinsed and placed in a pan of clean water and sterilised on an eighteen minute cycle. The items should then be cleaned following routine cleaning and sterilisation procedures.

Surface spills should be cleaned up using paper towels before the surface is wiped over with either sodium hydroxide or sodium hypochlorite, left for one hour (if possible or as long as possible, with the area cordoned off), the solution wiped off and the surface cleaned by following routine cleaning procedures.

Appendix 6: Cleaning and waste disposal procedures

Cleaning

General

Cleaning is important particularly in work areas because deposits of dust, soil and microbes on surfaces can transmit infection. Contaminated areas such as operating rooms or isolation rooms must be cleaned after each session and spot cleaned after each case or thoroughly cleaned as necessary.

The following basic principles should be followed:

- written cleaning protocols should be prepared, including methods and frequency of cleaning. These should include policies for the supply of all cleaning and disinfectant products
- standard precautions (including wearing of personal protective equipment as applicable) should be implemented when cleaning surfaces and facilities (see appendix 3)
- cleaning methods should avoid generation of aerosols
- all cleaning items should be changed after each use and cleaned and dried before being used again. They should also be changed immediately following the cleaning of blood or body fluid/substance spills, cleaned and dried. Single use cleaning items are preferred where possible such as cleaning cloths which should be lint free
- sprays should not be used as they can become contaminated and are difficult to clean. Sprays are not effective as they do not touch all parts of the surface to be cleaned
- detergents should not be mixed with other chemicals
- all cleaning solutions should be prepared fresh before use.

Specific

Surface cleaning

- Floors in hospitals and day care facilities should be cleaned daily, or as necessary, with a vacuum cleaner fitted with a particulate-retaining filter, which should be changed in accordance with the manufacturer's instructions.
- The exhaust air should be directed away from the floor to avoid dust dispersal.
- A ducted vacuum cleaning system can also be used, as long as safe venting of the exhaust air is ensured.
- Damp dusting is essential using a lint free cloth. Brooms disperse dust and bacteria into the air and should not be used in patient/clinical areas. Dust-retaining mops, which are specially treated or manufactured to attract and retain dust particles, do not increase airborne counts as much as ordinary brooms and remove more dust from surfaces (Ayliffe et al 1999). However, brooms and dust-retaining mops should not be used in clinical areas where there is a high risk of infection associated with dust (e.g. burns units).

Procedure for routine surface cleaning

- All cleaning solutions should be prepared immediately prior to use.
- Work surfaces should be cleaned (wiped over) with a neutral detergent and warm water solution, rinsed and dried before, and after, each session or when visibly soiled. Spills should be cleaned up as soon as practical.
- When a disinfectant is required for surface cleaning, the manufacturer's recommendations for use and OH&S instructions should be followed.
- Buckets should be emptied after use, washed with detergent and warm water, rinsed in hot water and stored dry - turn upside down.
- Mops should be laundered or cleaned in detergent and warm water, rinsed in hot water then stored dry. Mop heads should be detachable or stored with mop head uppermost.

Specialised areas

- Isolation rooms and ensuite bathrooms should be cleaned at least twice daily dependant on the type of organism.
- Operating rooms and day procedure rooms including endoscopy rooms should be cleaned after each operating session and when visibly soiled. Thorough cleaning of the operating suite should be performed daily in addition to the cleaning performed after each operating session.
- Obstetric areas, particularly delivery suites should be cleaned after each delivery, when visibly soiled and at least daily.
- Oncology areas should be cleaned twice daily.
- Sterilising processing departments (SSDs) should be cleaned at least twice daily and when visibly soiled.

Wet areas
Toilets, sinks, washbasins, baths, shower cubicles, all fittings attached to showers, baths and hand basins and surrounding floor and wall areas should be cleaned at least daily and more frequently as required.

Walls and fittings
Walls and screens should be cleaned quarterly or if visibly soiled. Blinds and curtains should be cleaned quarterly or if visibly soiled. Carpets should be vacuumed daily and other floor surfaces washed daily and when soiled.

Bed and examination screens should be changed weekly and when visibly soiled.

Cleaning for Creutzfeldt-Jakob disease infectious agents
Spills of central nervous system tissue or cerebrospinal fluid should be absorbed onto paper towels and disposed of by incineration. The surface should then be soaked with 1 molar sodium hydroxide or 2.0–2.5% sodium hypochlorite, left for one hour and cleaned again with paper towels that are disposed of by incineration. Spills of blood or other body fluids and tissues should be cleaned using standard spills management procedures. Personal protective equipment used when cleaning contaminated surfaces should be incinerated after use. Reusable eye protection should be cleaned as above.

Maintenance of cleaning equipment
- Cleaning items (including solutions, water, buckets, cleaning cloths and mop heads) should be changed after each use. They should also be changed immediately following the cleaning of blood or body substance spills.
- These items should be washed in detergent and warm water, rinsed and stored dry between uses. Mops with detachable heads should be laundered between uses.

Spills of laboratory cultures of human pathogens
Spills of laboratory cultures should be absorbed on to paper towels and disposed of as clinical waste. The contaminated surfaces should be treated with 2.0–2.5% sodium hypochlorite, left for one hour and cleaned again with paper towels that are disposed of as clinical waste.

Laboratories should also refer to AS/NZS 2243.3:2002: Safety in laboratories - Microbiological aspects and containment facilities.

Waste disposal

General
All health care facilities should have policies and procedures in place for the correct management of all waste generated. The Environmental Protection Authority (EPA) has clear guidelines on how waste should be managed. The National Health and Medical Research Council (NHMRC) also has guidelines on the management of waste generated in health care facilities.

Waste is classified into three main groups of waste:

- general
- clinical
- pharmaceutical

All waste should be stored in secure areas until collected. Waste disposal companies licensed with the EPA will collect all clinical and pharmaceutical waste for disposal in specialised waste disposal facilities which are also licensed by the EPA.

Waste should be removed from clinical areas at least three times each day and more frequently as needed such as from specialised areas. Waste bags should be tied before removing from the area.

General waste
Place in general waste bin for removal.

Clinical waste
Place in biohazard bags as soon as possible. Biohazard bags have a biohazard symbol and are currently coloured yellow.

Single use sharps should be placed (by the user) into a sharps container that meets the Australian and New Zealand Standards AS 4031:1992 and AS/NZS 4261:1994.

Pharmaceutical waste
When uncertain about how to dispose of leftover pharmaceuticals they should be returned to pharmacy for correct disposal.

Most disinfectants can be disposed of through the sewer system by running cold water into the sink prior to pouring the disinfectant into the sink. Leaving the cold water running for a few moments after the disinfectant has been disposed of as this dilutes the disinfectant.

Further information

- National Health & Medical Research Council 1999, *National guidelines for waste management in the health industry*, http://www.nhmrc.gov.au
- Environmental Protection Authority Victoria 1993, *Manual for the management and disposal of biomedical wastes in Victoria* (under review)
- Australian/New Zealand Standards AS/NZS 3816:1998, *Management of clinical and related wastes*
- Australian/New Zealand Standards AS 4031:1992, *Non-reusable containers for the collection of sharp medical items used in health care areas*
- Australian/New Zealand Standards AS/NZS 4261:1994, *Reusable containers for the collection of sharp items used in human and animal medical applications*

Appendix 7: Infections in children's services centres

Children in day care centres and other children's services centres and kindergartens are particularly at risk of developing communicable diseases because of:

- Close contact with other children and staff
- Lack of previous exposure to common infections
- Lack of toilet training
- Lack of control of other body secretions
- Mouthing behaviour

These risk factors may be increased when staff are not appropriately trained, group sizes are large, and mixing of age groups occurs.

Infections with the following organisms have been shown to be more common in these settings, or have been reported as epidemic:

- Respiratory Syncytial virus (RSV)
- Influenza virus
- *Haemophilus influenzae* type b
- *Neisseria meningitidis*
- *Shigella* spp
- Rotavirus
- *Giardia lamblia*
- *Cryptosporidium*
- Hepatitis A
- *E. coli*
- *Campylobacter* spp
- Parvovirus B19 (erythema infectiosum)
- Coxsackievirus group A (hand, food and mouth disease)
- *Streptococcus*, *pyogenes*, *Staphylococcus aureus* (impetigo)
- Cytomegalovirus
- Scabies
- Head lice

Appendix 8: School exclusion table

The following table indicates the minimum period of exclusion from schools and children's service centres required for infectious diseases cases and contacts as prescribed under Regulations 13 and 14 of the Health (Infectious Diseases) Regulations 2001 – Schedule 6. In this Schedule 'medical certificate' means a certificate of a registered medical practitioner.

Disease or condition	Exclusion of cases	Exclusion of contacts
Amoebiasis *(Entamoeba histolytica)*	Exclude until diarrhoea has ceased	Not excluded
Campylobacter	Exclude until diarrhoea has ceased	Not excluded
Chickenpox	Exclude until fully recovered or for at least 5 days after the eruption first appears. Note that some remaining scabs are not a reason for continued exclusion	Any child with an immune deficiency (for example, leukaemia) or receiving chemotherapy should be excluded for their own protection. Otherwise not excluded
Conjunctivitis	Exclude until discharge from eyes has ceased	Not excluded
Diarrhoea	Exclude until diarrhoea has ceased or until medical certificate of recovery is produced	Not excluded
Diphtheria	Exclude until medical certificate of recovery is received following at least two negative throat swabs, the first not less than 24 hours after finishing a course of antibiotics and the other 48 hours later	Exclude family/household contacts until cleared to return by the Secretary
Haemophilus type b (Hib)	Exclude until medical certificate of recovery is received	Not excluded
Hand, foot and mouth disease	Until all blisters have dried	Not excluded
Hepatitis A	Exclude until a medical certificate of recovery is received, but not before 7 days after the onset of jaundice or illness	Not excluded
Herpes ('cold sores')	Young children unable to comply with good hygiene practices should be excluded while the lesion is weeping. Lesions to be covered by dressing, where possible	Not excluded
Human immuno-deficiency virus infection (HIV/AIDS)	Exclusion is not necessary unless the child has a secondary infection	Not excluded
Impetigo	Exclude until appropriate treatment has commenced. Sores on exposed surfaces must be covered with a watertight dressing	Not excluded
Influenza and influenza like illnesses	Exclude until well	Not excluded
Leprosy	Exclude until approval to return has been given by the Secretary	Not excluded
Measles	Exclude until at least 4 days after the onset of rash	Immunised contacts not excluded. Unimmunised contacts should be excluded until 14 days after the first day of appearance of rash in the last case. If unimmunised contacts are vaccinated within 72 hours of their first contact with the first case they may return to school
Meningitis (bacteria)	Exclude until well	Not excluded
Meningococcal infection	Exclude until adequate carrier eradication therapy has been completed	Not excluded if receiving carrier eradication therapy

Disease or condition	Exclusion of cases	Exclusion of contacts
Mumps	Exclude for 9 days or until swelling goes down (whichever is sooner)	Not excluded
Poliomyelitis	Exclude for at least 14 days from onset. Re-admit after receiving medical certificate of recovery	Not excluded
Ringworm, scabies, pediculosis (head lice)	Re-admit the day after appropriate treatment has commenced	Not excluded
Rubella (german measles)	Exclude until fully recovered or for at least four days after the onset of rash	Not excluded
Salmonella, Shigella	Exclude until diarrhoea ceases	Not excluded
Severe Acute Respiratory Syndrome (SARS)	Exclude until medical certificate of recovery is produced	Not excluded unless considered necessary by the Secretary
Streptococcal infection (including scarlet fever)	Exclude until the child has received antibiotic treatment for at least 24 hours and the child feels well	Not excluded
Trachoma	Re-admit the day after appropriate treatment has commenced	Not excluded
Tuberculosis	Exclude until receipt of a medical certificate from the treating physician stating that the child is not considered to be infectious	Not excluded
Typhoid fever (including paratyphoid fever)	Exclude until approval to return has been given by the Secretary	Not excluded unless considered necessary by the Secretary
Verotoxin producing *Escherichia coli* (VTEC)	Exclude if required by the Secretary and only for the period specified by the Secretary	Not excluded
Whooping cough	Exclude the child for 5 days after starting antibiotic treatment	Exclude unimmunised household contacts aged less than 7 years and close child care contacts for 14 days after the last exposure to infection or until they have taken 5 days of a 10 day course of antibiotics
Worms (Intestinal)	Exclude if diarrhoea present	Not excluded

Exclusion cases and contacts is not required for Cytomegalovirus Infection, Glandualr fever (mononucleosis), Hepatitis B or C, Hookworm, Cytofalovirus Infection, Molluscum contafiosum, or , Parvovirus (erythema infectiosum, fifth disease).