Listeria monocytogenes (Infection with)

Aetiology  Epidemiology  Diagnosis  Prevention and Control
Potential Impacts of Disease Agent Beyond Clinical Illness  References

AETIOLOGY

Classification of the causative agent

Listeria monocytogenes, the causative agent of listeriosis, is a Gram-positive, facultatively-intracellular rod capable of causing septicemia, neurologic disease, and reproductive disease in a wide range of hosts. It is regarded as a highly consequential human foodborne pathogen. Because it is ubiquitous in the environment, it can also cause devastating disease in veterinary species, especially ruminants.

Resistance to physical and chemical action

Temperature: Capable of tolerating a wide range of temperatures; may replicate when refrigerated (5°C); can survive some pasteurization techniques but autoclaving is effective (moist heat 121°C/15min or dry heat 160-170°C/1hr)

pH: Tolerates a broad pH range
Chemicals/Disinfectants: Enzymatic detergents greatly facilitate biofilm degradation but should be followed with a disinfectant; quaternary ammonium compounds, povidone iodine, chlorhexidine, 7.0% ethanol, glutaraldehyde, and sodium hypochlorite are typically effective; efficacy is increased if formulated with ozonated water rather than tap water
Survival: Can persist in environments with a low oxygen tension due to its facultative anaerobic nature; tolerates dry and salty conditions; very environmentally stable and is known to utilise adaptive responses to increasingly tolerate undesirable conditions

EPIDEMIOLOGY

Hosts

- L. monocytogenes is capable of infecting an incredibly wide range of animal species; this is not an exhaustive list.
- Wild and domestic ruminants
  - Sheep (*Ovis* spp.)
  - Goats (*Capra* spp.)
  - Cows (*Bos* spp.)
  - Water buffalo (*Bubalus bubalis*)
  - Deer (particularly *Capreolus capreolus, Dama dama, Odocoileus virginianus*)
  - Moose (*Alces alces*)
  - Llamas (*Lama glama*)
  - Giraffes (*Giraffa camelopardalis*)
- Wild and domestic swine (*Sus* spp.)
- Wild and domestic horses (*Equus ferus* ssp.)
- Wild and domestic lagomorphs (particularly *Lepus, and Oryctolagus* ssp.)
- Wild and domestic rodents (families *Myomorpha* and *Hystricomorpha*)
- Wild and domestic marsupials
  - Antechinus (*Antechinus* spp.)
  - Sugar gliders (*Petaurus breviceps*)
  - Possums (*Trichosurus vulpecula, Pseudocheirus peregrinus*)
Red-necked wallabies (*Macropus rufogriseus*)
- Skunks (family *Mephitidae*)
- Mink (*Neovison vison*)
- Order Carnivora
  - Servals (*Felis serval*)
  - Leopards (*Panthera pardus*)
  - Coyotes (*Canis latrans*)
  - Raccoons (*Procyon lotor*)
  - Foxes (*Vulpes spp.*)
- Wild and domestic birds
  - Poultry (order *Galliformes*)
  - Ducks (order *Anseriformes*)
  - Pigeons (*Columba spp.*)
  - Canaries (*Serinus canaria*)
  - Cockatiels (*Nymphicus hollandicus*)
- Humans (*Homo sapiens*) and non-human primates
  - Macaques (*Macaca nigra, M. mulatta*)
  - Chimpanzees (*Pan troglodytes*)
  - Marmosets (*Callithrix jacchus*)
  - Mona monkeys (*Cercopithecus mona*)
- Reported in reptiles rarely
  - Bearded dragons (*Pogona vitticeps*)
  - Alligators (*Alligator spp.*)

**Transmission**

- Ingestion
- Contact with broken mucous membranes, conjunctiva, or skin

**Sources**

- *L. monocytogenes* is ubiquitous in the environment
  - Common sources are contaminated feed and water
- Faeces
- Reproductive fluids and membranes, e.g., placenta, aborted foetus

**Occurrence**

Animals are usually asymptomatic carriers of *L. monocytogenes*. Outbreaks of listeriosis in wildlife species are typically sporadic and associated with exposure to high burdens of the bacterium over a short period of time. Otherwise, clinical signs may arise secondary to immunocompromise, predisposing disease, or excessive environmental stress.

Listeriosis has been documented as a significant cause of mortality events in breeding *Antechinus* males.

**DIAGNOSIS**

There are three main presentations of disease caused by *L. monocytogenes*: septicaemia, neurologic disease, and reproductive disease. The mechanisms by which a particular presentation is determined are not fully understood. It has been hypothesized that latent infections can recur when cell-mediated immune mechanisms are impaired, potentially releasing the intracellular pathogen from macrophages. There is debate regarding whether the organism gains entry to the body through the gastrointestinal tract or through oral
mucous membranes and the conjunctiva; it is likely these routes of entry hold varying levels of significance between species.

*L. monocytogenes* utilises retrograde axonal transport to infect the central nervous system. Encephalitis in domestic ruminants often presents 3-4 weeks after ingesting soiled feed, but the incubation time may be as prolonged as 7 weeks. Septicaemia develops rapidly, typically in 1-2 days, and abortions occur approximately 1 week post-exposure.

**Clinical diagnosis**

Animals are often found acutely dead because they typically do not develop clinical signs secondary to septicaemic listeriosis. Uterine infections typically cause abortions in pregnant dams with few other systemic signs. Exceptions include rabbits and chinchillas, which may develop gastrointestinal signs alongside metritis.

Encephalitis/rhombencephalitis and meningoencephalitis are perhaps the most common and well-recognized presentations of listeriosis. Affected animals become depressed, ataxic, and often paralytic. Ruminants and South American camels are particularly susceptible to severe disease and commonly develop unilateral neurologic signs, such as circling, torticollis and opisthotonos, nystagmus, and cranial nerve V/VII deficits. Paresis may develop in the fore- or hindlimbs; rarely are animals tetraparetic. Ocular disease can also be present and is sometimes regarded as a fourth, separate presentation of listeriosis.

Clinical disease in birds is rare. Young birds are more susceptible to both infection and development of clinical disease. Poultry typically present with septicaemia or encephalitis with paresis/paralysis of the limbs, torticollis, tremors, incoordination, and stupor/depression.

Few cases of reptilian listeriosis have been documented. Affected individuals have displayed nonspecific signs of disease, primarily obtundation and anorexia, but gassy intestinal bloat appears to be a common feature. All documented cases have been fatal, despite treatment.

**Lesions**

- **Encephalitis/meningoencephalitis**
  - Cloudy, oedematous or congested meninges
  - Suppurative meningoencephalitis with microabscesses and microgliosis, primarily in the brainstem and cranial nerve tracts/nucleoli
  - There may be few to no gross lesions apparent

- **Septicaemia**
  - Multifocal areas of hepatic inflammation and necrosis
  - Spleen, lung, kidney, and enteric lymphoid tissue may also be inflamed and necrotic
  - Peritonitis
  - Neonates may have haemorrhagic gastrointestinal lesions

- **Abortion**
  - Multifocal areas of placental cotyledonary necrosis with intercotyledonary suppurative placentitis
  - Foetus may have lesions that reflect septicaemia
    - Serous cavities may contain serosanguinous fluid
    - Generalised oedema
    - The right half of the liver may reflect more necrotic and inflammatory change than the left
    - 1-3 cm mucosal erosions in the abomasum (variable presence)
    - Spleen and lung may also be necrotic
    - Autolysis may mask lesions

- **Birds**
  - If septicaemic: yellow to yellow-tan areas of necrotising myocarditis, necrotising hepatitis, splenomegaly, ascites, and petechial haemorrhages on viscera
    - Infrequently, granulomatous and caseous hepatitis, catarrhal enteritis
If encephalitic: malacia, mixed- and lymphocytic inflammation, perivascular cuffing, and gliosis
- The medulla, cerebellum, and optic lobes are most severely affected
- Gross lesions due to encephalitis are often lacking, but lesions due to septicaemia may be concurrent in these animals

**Differential diagnoses**

- Rabies
- Brain abscess
- Coenurosis
- Thromboembolic meningoencephalitis
- Polioencephalomalacia
- Toxoplasmosis
- Other causes of septicaemia, such as:
  - Yersiniosis
  - Salmonellosis
- Conjunctivitis
- Tyzzer’s disease
- Q-Fever (*Coxiella burnetii*)
- Pregnancy toxaemia/ketosis
- Bovine Spongiform Encephalopathy
- Lead toxicity
- Vestibular disease
- Birds
  - Marek’s disease
  - Virulent Newcastle disease
  - West Nile virus

**Laboratory diagnosis**

**Samples**

*For isolation of agent*

- Liver and/or spleen
- Brain
- Heart (poultry)
- Foetus, foetal stomach content
- Placenta
- Milk from mastitic dams
- Blood (if septicaemic)
- Cerebrospinal fluid (inconsistent)
- Ocular swabs
- Feed samples

**Serological tests**

- Not recommended for veterinary cases of listeriosis

**Procedures**

**Identification of the agent**

- Culture on blood agar/broth or nutrient agar
  - Cold enrichment and low oxygen tension may facilitate isolation, especially if the sample is contaminated
If using brain tissue, weekly re-cultures of refrigerated tissue may be required until the organism is isolated. This process may take months and is relatively work-intensive. As such, this method is becoming less utilised.

- Polymerase chain-reaction (PCR)
- Loop-mediated isothermal amplification (LAMP)
- Immunohistochemistry (IHC) and immunofluorescent assays (IFA)
  - Sensitive and specific; particularly useful if culture is not available or fails to yield a sample
  - Preferred method for poultry
- Pulsed-field gel electrophoresis (PFGE)
  - Primarily used in foodborne illness outbreak investigations
- Whole-genome sequencing
  - Primarily used in foodborne illness outbreak investigations

**Serological tests**

- Serology is infrequently used in veterinary species due to prominent cross-reactivity with enterococci and *Staphylococcus aureus*, low assay sensitivity and specificity, and the presence of high titres that confound results in healthy animals.
  - Anti-listeriolysin IgG antibody capture enzyme-linked immunosorbent assays (ELISAs) are available but results are often inconsistent

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 3.9.6 *Listeria monocytogenes* in the latest edition of the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.

**PREVENTION AND CONTROL**

**Sanitary prophylaxis**

- *L. monocytogenes* is very difficult to control because it is ubiquitous in the environment
- Best practices indicate maintaining proper hygiene at feeding areas, especially where free-ranging ruminants are present, and preventing contamination with faeces
- Do not discard spoiled silage or haylage where it remains easily accessible to wildlife

**Medical prophylaxis**

- There is no commercially available vaccine available for *L. monocytogenes* in wildlife
- Vaccines utilised for livestock are not particularly efficacious; livestock owners commonly decline their use due to a lack of perceived benefit

**POTENTIAL IMPACTS OF DISEASE AGENT BEYOND CLINICAL ILLNESS**

**Risks to public health**

- While listeriosis is an appreciable foodborne illness in humans, cases are typically associated with dairy products and other foods that have been processed improperly; wild free-ranging animals themselves are not considered a significant source of infection for humans.
- Personal protective equipment should be used if handling contaminated tissues such as aborted foetuses and placentas in zoological parks, rehabilitation facilities, or research settings. Localised cutaneous lesions may occur with direct skin contact.

**Risks to agriculture**

- Listeriosis can significantly impact livestock if feed is improperly stored or is otherwise contaminated. Reproductive losses and deaths due to neurologic disease or septicaemia can have grave financial impact on industries that rely on cattle, sheep, and goats.
It is not likely, however, that free-ranging wildlife interacting with livestock directly is culpable for outbreaks of listeriosis. Ensure free-ranging animals do not have access to haylage or silage to prevent faecal contamination and storage compromise.

REFERENCES AND OTHER INFORMATION


The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Science Department (scientific.dept@oie.int). Last updated 2020. Written by Samantha Gieger and Erin Furmaga with assistance from the USGS National Wildlife Health Center.