

# Morbillivirus (canids and felids)(Infection with)

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

## AETIOLOGY

### **Classification of the causative agent**

Morbilliviruses belong to the family *Paramyxoviridae*, and are enveloped, negative-sense single-stranded RNA viruses. Members of the genus *Morbillivirus* are able to infect a wide range of hosts to cause varied types of disease, many of which are severe.

Canine morbillivirus infections are often referred to as “canine distemper” (CDV). Infections in domestic dogs are rare thanks to vaccination strategies, but free-roaming canines are still a source and can transmit the virus to many different species, including members of the family *Felidae*. Felines also have their own viral species that is often referred to as “feline panleukopenia virus” (FPV). CDV and FPV are very similar genetically. CDV is a threat to many endangered species worldwide, including many large cat species, black-footed ferrets, red pandas, and Caspian seals.

There are at least seven lineages of canine distemper worldwide:

- Asia-1 and -2
- American-1 and -2
- Arctic-like
- European wildlife
- Europe

### **Resistance to physical and chemical action**

Temperature:	Steam cleaning is effective.
pH:	Not determined
Chemicals/Disinfectants:	Susceptible to a 1:30 bleach dilution, potassium peroxydisulfate, accelerated hydrogen peroxide, and aldehydes with contact times over 10 minutes
Survival:	Environmental survival depends on ambient temperatures; colder temperatures prolong viability.

## EPIDEMIOLOGY

### **Hosts**

- Members of the following families are susceptible to infection:
  - *Canidae*
  - *Felidae*
  - *Procyonidae*
  - *Mustelidae*
  - *Hyaenidae*
  - *Viverridae*
- Collared peccary (*Tayassu tajacu*)
- Bears (*Ursus* spp.)

- Red pandas (*Ailurus fulgens*)
- Caspian and Baikal seals (*Pusa caspica*, *P. sibirica*)
- There have been rare reports of CDV in Sika deer (*Cervus nippon*), macaques (*Macaca* spp.), and wild boars (*Sus scrofa*) in Japan

### **Transmission**

- Direct contact with an infected animal
- Droplet and aerosol transmission
- Insects and humans may act as mechanical vectors

### **Sources**

- Body fluids (urine, saliva, blood) and faeces
- Contaminated fomites
- Contaminated environment

### **Occurrence**

Canine and feline morbilliviruses are enzootic in Africa, Europe, North and South America, and Asia. Reports of CDV had long been absent in Australia due to widespread vaccine use in domestic dogs, however, the virus is now sporadically recurring in semi-rural areas. It is believed that foxes, dingoes, and/or feral dogs are a reservoir and are reintroducing the virus to domestic dogs.

## **DIAGNOSIS**

The incubation period of CDV/FPV ranges from 3-6 days, and the duration of disease depends on host factors and any secondary infections. Juveniles are more susceptible to disease, particularly after maternal antibody wanes. Virus is shed a few days before the development of clinical signs and wanes significantly by 7 days post-infection.

### **Clinical diagnosis**

Clinical signs depend on viral strain, host factors such as immune status and age, and environmental pressures (stress). Approximately half of all CDV infections in canids are subclinical or produce only a mild, self-limiting respiratory disease.

Infected animals may become febrile, lethargic, and anorectic. Conjunctivitis and bilateral mucopurulent ocular discharge may accompany any respiratory disease. Severe infections cause a fever that continues to worsen; this “second wave” fever coincides with leukopenia and a systemic spread of the virus. Affected animals become severely anorectic, develop significant respiratory disease, and become depressed. Gastrointestinal signs are also possible and are characterised by vomiting and profuse watery diarrhoea.

Some animals may develop central nervous system (CNS) disease 1-3 weeks after acute signs of infection. Signs are typically progressive and include: seizures (“chewing gum fits”, epileptic seizures), cerebellar signs, vestibular signs, para- or tetraparesis, sensory ataxia, and/or myoclonus. There is currently no way to differentiate which animals are vulnerable to developing neurologic complications, and prognosis worsens with the development of CNS signs.

## **Lesions**

- Hyperkeratosis of the nose and footpads
- Enteritis
  - Dilated intestinal crypts and damaged villi
- Lymphoid necrosis, depletion of Peyer's patches
- Hepatic and pancreatic acinar atrophy and/or congestion
  - May cause icterus
- Conjunctivitis
- Rhinitis and tracheitis
- Ulcerative glossitis
- Bronchointerstitial pneumonia with epithelial necrosis and alveolar wall thickening
- Neuronal demyelination and/or necrosis
- Gliosis
- Nonsuppurative meningoencephalitis
- Polioencephalitis
- Inclusions and/or syncytia in astrocytes and epithelial cells of the lung, stomach, renal pelvis, and urinary bladder
- If infected at a young age:
  - Odontodystrophy
  - Metaphyseal osteosclerosis of long bones
  - Cerebellar hypoplasia if infected *in utero*

## **Differential diagnoses**

- Kennel cough (canine infectious tracheobronchitis)
- Canine parvovirus
- Feline leukaemia virus
- Rabies
- Enteric parasitic infections
- Salmonellosis
- Toxin exposure

## **Laboratory diagnosis**

### **Samples**

*For isolation of agent*

- Conjunctival swabs
- Any tissue sample that contains epithelium
- Thymus and other lymphoid tissues
- Urine and/or faeces (may substitute with rectal swab)

*Serological tests*

- Blood mononuclear cells (lymphocytes, macrophages, dendritic cells)
  - Virions are only detected in these cells later in the course of disease.

### **Procedures**

### *Identification of the agent*

- Reverse-transcriptase polymerase chain reaction (RT-PCR)
- Virus isolation
- Immunohistochemistry
- Fluorescent antibody staining
- Faecal enzyme-linked immunosorbent assay (ELISA)
- Electron microscopy
- Histopathology

### *Serological tests*

- Indirect fluorescent antibody tests
- Haemagglutination inhibition
- ELISA

## **PREVENTION AND CONTROL**

### ***Sanitary prophylaxis***

- In captive facilities, environmental decontamination, isolation of non-immunised animals, and vector control are useful to prevent the spread of the virus.
  - If possible, bathe recovered animals to remove any virus that may be present on the haircoat.

### ***Medical prophylaxis***

- Modified-live vaccines are available for domestic canids, but their use often complicates clinical diagnosis (i.e., if a young animal received a vaccine but was infected before the vaccine induced protective immunity).
  - Proper vaccine administration induces life-long immunity, but may be inhibited by the presence of maternal antibody.
  - Vaccines usually encompass viral strains from the American-1 lineage, and antigen cross-reactivity is sufficient enough to provide protection against other strains.
  - Standard modified-live vaccines should not be used in species outside of the canid family due to adverse reactions.
- Inactivated vaccines have been used in zoo animals with variable efficacy.
- Vectored canarypox vaccines have a higher efficacy and are now preferred for zoo animals.
- Medical treatment includes the administration of hyperimmune serum or immune globulin immediately after exposure.

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

### ***Risks to public health***

- Canine and feline parvoviruses are not infectious to humans.

## **Risks to agriculture**

- Mink-farming operations are at risk for CDV infection; appropriate sanitary and medical prophylactic measures should be considered to reduce the likelihood of disease. Mortality rates may approach 100% in unvaccinated populations.

## **REFERENCES AND OTHER INFORMATION**

- Beineke, A., Baumgärtner, W., Wohlsein, P. (2015). Cross-species transmission of canine distemper virus - an update. *One Health*, 1, 49-59.
- Fenner, F. J. (2011). Canine Distemper Virus. In N. J. MacLachlan and E. J. Dubovi (Eds.), *Fenner's Veterinary Virology* (4th ed., pp. 317-320). Elsevier.
- Kapil, S., Allison, R. W., Johnston, L., Murray, B. L., Holland, S., Meinkoth, J., Johnson, B. (2008). Canine distemper virus strains circulating among North American dogs. *Clinical and Vaccine Immunology*, 15(4), 707-712.
- Loots, A. K., Mitchel, E., Dalton, D. L., Kotzé, A., Venter, E. H. (2017). Advances in canine distemper virus pathogenesis research: a wildlife perspective. *Journal of General Virology*, 98(31), 311-321.
- Norris, J. M., Krockenberger, M. B., Baird, A. A., Knudsen, G. (2006). Canine distemper: re-emergence of an old enemy. *Australian Veterinary Journal*, 84, 362-363.
- Sykes, J. E. (2014). Feline Panleukopenia Virus Infection and Other Viral Enteritidies. *Canine and Feline Infectious Diseases*. (pp. 187-194). Saunders.

\*

\* \*

The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department ([scientific.dept@oie.int](mailto:scientific.dept@oie.int)). Last updated 2019. Written by Marie Bucko and Samantha Gieger with assistance from the USGS National Wildlife Health Center.