

PARVOVIRUS SPP.

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

AETIOLOGY

Classification of the causative agent

There are several parvoviruses that infect different animal species worldwide, including domestic and wild mammals, crustaceans, and arthropods. Parvoviruses (family *Parvoviridae*) are non-enveloped, negative-sense single-stranded DNA viruses. *Parvoviridae* is divided into 2 subfamilies: *Densovirinae*, which infect arthropods, and *Parvovirinae*, which infect vertebrates. The greatest concern to wildlife are viruses in the *Parvovirinae* subfamily, whose genera include *Erythroparvovirus*, *Dependoparvovirus*, *Amdoparvovirus*, *Bocaparvovirus*, and *Parvovirus*. Parvoviruses are mostly of concern to domestic animals; they can be transmitted to or from wildlife and may be a threat to captive wildlife species.

Resistance to physical and chemical action

Temperature: Resistant to dry heat up to 90°C; may be inactivated with steam cleaning

pH: Inactivated at low pH

Chemicals/Disinfectants: Inactivated by >2500 ppm sodium hypochlorite, beta-propiolactone, oxidising agents, hydroxylamine, 2% sodium hydroxide, and formalin

Survival: Can survive up to one year in organic material; inactivated by ultraviolet radiation

EPIDEMIOLOGY

There are several genera and species of parvoviruses; the ones most relevant to wildlife are listed below.

Hosts

- *Amdoparvovirus*
 - Aleutian mink disease virus (AMDV)
 - Ferrets (*Mustela putorius furo*)
 - European mink (*Mustela lutreola*)
 - American mink (*Mustela vison*)
 - Skunks (genus *Mephitidae*)
 - Raccoons (*Procyon lotor*)
- *Bocaparvovirus*
 - Canine parvovirus 1 (CPV1)
 - Domestic dogs (*Canis lupus familiaris*)
 - Goose parvovirus (GPV)
 - Geese
 - Domestic geese (*Anser anser domesticus*)
 - Snow goose (*Chen caerulescens*)
 - Ducks
 - Muscovy ducks (*Cairina moschata*)
- *Parvovirus*
 - Canine parvovirus 2 (CPV2)
 - Asian palm civets (*Paradoxurus hermaphroditus*)
 - Asian small-clawed otters (*Aonyx cinerea*)

- Common genets (*Genetta genetta*)
- Coyotes (*Canis latrans*)
- Domestic canines (*Canis lupus familiaris*)
- Egyptian mongooses (*Herpestes ichneumon*)
- European red foxes (*Vulpes vulpes*)
- Maned wolves (*Chrysocyon brachyurus*)
- Marsican brown bears (*Ursus arctos marsicanus*)
- Pine martens (*Marten marten*)
- Raccoons (*Procyon lotor*)
- Raccoon dogs (*Nyctereutes procyonoides*)
- Wolves (*Canis spp.*)
- Feline panleukopenia virus (FPLV)
 - Arctic foxes (*Alopex lagopus*)
 - Asian palm civets (*Paradoxurus hermaphroditus*)
 - Cheetahs (*Acinonyx jubatus*)
 - Domestic felines (*Felis catus domesticus*)
 - Ferrets (*Mustela putorius furo*)
 - Florida panthers (*Felis concolor coryi*)
 - Iberian lynx (*Lynx pardinus*)
 - Mink (*Mustela* and *Neovision* genera)
 - Mountain lions (*Puma concolor*)
 - European wildcats (*Felis silvestris*)
- Mink enteritis virus (MEV)
 - Mink (*Mustela* and *Neovision* genera)
- Porcine parvovirus 1 (PPV1)
 - Domestic swine (*Sus scrofa domesticus*)
 - Wild boars (*Sus scrofa*)

Transmission

- AMDV
 - The virus is shed in raccoon faeces and transmitted via the faecal-oral route
 - Flies are thought to be mechanical vectors
 - Vertical transmission
 - Direct contact with infected mink
- CPV2
 - Oral or nasal contact with excreta, fomites, or faeces containing the virus
- FPLV
 - Ingestion of excreta
 - Contact with mechanical vectors or fomites
- PPV1
 - Transplacental (vertical) transmission

Sources

- AMDV
 - Raccoon excreta
 - Flies
 - Infected animals
- CPV2
 - Excreta from infected animals, including faeces
- FPLV
 - Excreta from infected animals
 - Mechanical vectors
 - Fomites
 - Infected animals
- PPV1
 - Boar semen

Occurrence

American mink are thought to be the original host of AMDV. The virus is more likely to cause clinical signs in mink that are genetically homozygous recessive for pale coat color. This virus is of particular concern for mink farms worldwide, especially in the Netherlands, Finland, Sweden, Norway, and Russia (Karelia and Kola Peninsula). Antibodies to AMDV have been found in feral European mustelids and common genet in France and England, and in wild American mink from France, Spain, and southern England. MEV was first discovered in 1947 in Ontario, Canada on a mink farm. Mink farm outbreaks of this virus have occurred in the United States, Canada, Poland, Finland, Denmark, Sweden, France, and the Netherlands. Pathology of this virus in wild mink is unknown.

CPV2 was recognised as a new virus in 1978 and is thought to have originated from FPLV. There have been several outbreaks of this virus in raccoons in the south eastern United States. CPV2 is believed to be responsible for morbidity and mortality in wolf populations surrounding Yellowstone National Park, United States. It is also thought that CPV2 has caused a gradual decrease in wolf populations in Minnesota, United States since the 1970s. There have been outbreaks of CPV2 in wild raccoons in the south-eastern United States. The virus may also affect captive canids. There are three known strains of CPV2: CPV2a, CPV2b and CPV2c. CPV2c has been found to infect captive Asian small-clawed otters. CPV1, another parvovirus that infects dogs, is of lesser concern to wildlife.

FPLV has a worldwide distribution. The virus is an appreciated threat to the endangered Florida panther and is known to infect other threatened and endangered wildlife species, such as the Iberian lynx and European wildcat.

GPV outbreaks in domestic geese are associated with the breeding season (spring) and migration in free-ranging geese; feral geese generally do not display clinical signs. A 2004 GPV outbreak on Swedish goose farms was associated with exposure to wild geese. GPV is a concern in countries that farm geese, such as Russia, Japan, and China, as well as some European countries (e.g., France, Germany, Denmark, and Great Britain).

PPV1 was first recognized in 1967 and has a worldwide distribution. Though it has not been documented, it is theoretically possible that wild boar could transmit the virus to domestic swine. Serosurveys indicate that approximately one third of wild boars in South Korea have antibodies to PPV1.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information System - Wild (WAHIS-Wild) Interface [http://www.oie.int/wahis_2/public/wahidwild.php/Index].

DIAGNOSIS

The incubation period of AMDV varies from months to years. The three known strains of CPV2 have similar clinical presentations and pathophysiologies. Viral shedding occurs 4-5 days after infection, and can continue for about 10 days after the end of clinical signs. FPLV is very contagious and most often infects kittens. The incubation period is about 2-7 days. Incubation of MEV is 4-8 days and the most serious infections occur in kits.

Clinical diagnosis

Clinical signs for parvoviruses are similar for both domestic and wild animals.

In domestic mink, clinical signs of AMDV infection include reproductive inefficiency, renal failure, uraemia, oral and gastrointestinal bleeding, and wasting. Kits may be weak and succumb to acute interstitial pneumonia. Ferrets may experience wasting and have paresis or ataxia in their hindlimbs. MEV infection presents as watery, mucoid, bloody diarrhoea, anorexia, depression, and dehydration.

Most CPV2 infections present with little or mild clinical signs. Severe cases include anorexia, vomiting, bloody faeces, and foul-smelling diarrhoea. Disease can be more severe if there are co-infections with pathogens

such as *Salmonella* spp. or *Giardia* spp. Rarely, young animals develop myocardial injury between one and two months of age. Raccoon dogs develop gastroenteritis. While serology has shown that red pandas can be infected with parvovirus (either CPV2 or FPLV), there is no association with clinical disease.

Clinical signs of FPLV infection include a fever $>40^{\circ}\text{C}$. Domestic cats generally die during the acute phase of the disease. If cats survive peracute infection, they may develop a rough haircoat and/or vomit. Fatalities may occur due to dehydration from diarrhoea. This virus has caused mortality in Asian palm civets.

GPV, clinically known as Derzy's disease, can cause death in 8-30 day old goslings. Clinical signs include weight and feather loss.

Reproductive failure (e.g., abortions, stillborn piglets) due to PPV1 has been reported in a domesticated herd of wild boars. In domestic swine, the virus causes reproductive disease in swine characterised by stillbirths, mummification, embryonic death, and infertility (SMEDI). Both live and mummified foetuses may be born into litters. These reproductive failures are more likely to happen in gilts than sows. Adult swine generally do not develop clinical signs. Rarely, they present with skin lesions and diarrhoea. PPV1 replicates in the tonsils and oronasal cavities, after which it enters the lymphatic system. After approximately 15 days, the virus crosses the placenta to infect the foetus. However, PPV1 has not been found to infect the placenta itself. Unvaccinated swine herds infected with PPV1 may experience abortion storms. Immunity is conferred to piglets from maternal antibodies in colostrum. Adult swine must be seronegative in order to pass along the virus. There are other porcine parvoviruses (e.g., PPV2-7) that have not been associated with clinical signs.

Lesions

- AMDV
 - Domestic mink
 - Splenomegaly
 - Arteritis, immune complex-mediated vasculitis
 - Hypergammaglobulinaemia
 - Plasmacytosis
 - Hepatitis
 - Anaemia
 - Nonsuppurative meningoencephalitis
 - Membranous glomerulonephritis
- CPV2
 - Enterocytes in intestinal crypts with mucosal collapse
 - Mucosal and serosal haemorrhage
 - Lymphopenia, neutropenia
 - Young canids
 - Myocardial necrosis and inflammation
 - Myocardial fibrosis
 - Dilated cardiomyopathy
 - Pulmonary oedema
 - Hepatic congestion
 - Lymphocytic myocarditis
- FPLV
 - Panleukopenia
 - Total white blood cell count may be between 1,000-2,000/mL blood
 - Neutrophil counts may be $<200/\text{mL}$
- GPV
 - Diffuse or focal vacuolar degeneration
 - Necrotising hepatitis and enteritis
 - Necrosis of cardiac muscle
 - Inclusion bodies in thyroid, intestines, spleen, myocardium
 - Oral and pharyngeal ulceration
- MEV
 - Erosion of intestinal surface mucosa

- Blunting and attenuation of intestinal villi
- Dilation of intestinal crypts
- Ballooned intestinal epithelial cells
- Lymphoid depletion and necrosis in spleen and lymph nodes
- PPV1
 - Domestic swine
 - Foetuses
 - Oedema
 - Stunted growth
 - Haemorrhage and serosanguinous fluids in body cavities
 - Haemorrhage of muscle and subcutaneous tissue
 - Necrosis of kidneys, liver, lungs, and skeletal muscle
 - Piglets
 - Vesicle-like lesions and slit-like erosions in oral cavity and snout
 - Necrotic and exudative dermatitis
 - Sows/Gilts
 - Focal accumulation of mononuclear cells in endometrium and lamina propria
 - Focal accumulation of lymphocytes in uterus
 - Grey to brown dehydrated placenta
 - Boars
 - Experimental infection of the testicles has resulted in seminiferous tubule degeneration and sloughing of multinucleated cells

Differential diagnoses

- AMDV
 - Wasting
 - Eosinophilic gastroenteritis
 - *Helicobacter mustelae*-associated gastritis
 - Proliferative bowel disease
 - Lymphoma
 - Hindlimb paralysis/paresis
 - Canine distemper
 - Rabies
 - Neoplasia
 - Thromboembolism
 - *Mycobacterium* spp. infection
 - Central nervous system disease
- CPV2
 - Canids
 - Clostridial enteritis
 - Canine coronavirus
 - Canine herpesvirus
 - Salmonellosis
 - Anticoagulant rodenticide toxicity
- FPLV
 - Wobbly cat syndrome
 - Feline leukaemia virus (FeLV)
 - Feline immunodeficiency virus (FIV)
 - Salmonellosis
- GPV
 - Duck virus enteritis (DVE)
 - Duck virus hepatitis (ducklings)
 - Aspergillosis
 - Bacterial septicaemia (due to *Pasteurella multocida* or *Riemerella anatipestifer*)
 - Haemorrhagic nephritis
 - Virus K disease

- MEV
 - Mink
 - Epizootic catarrhal enteritis
- PPV1
 - Brucellosis
 - Pseudorabies
 - Leptospirosis
 - Porcine reproductive and respiratory syndrome (PRRS)
 - Pyometra
 - Toxoplasmosis

Laboratory diagnosis

Samples

For isolation of agent

- CPV2
 - Faeces
- FPLV
 - Intestines
 - Lymph nodes
 - Faeces
- GPV
 - Liver
 - Heart
 - Spleen
 - Intestines
 - Kidneys
- PPV1
 - Foetal tissues
 - Thoracic fluid (stillborn pigs)
 - Semen

Serological tests

- Serum
- Whole blood

Procedures

Identification of the agent

- CPV2
 - Antigen enzyme-linked immunosorbent assay (ELISA) of faecal samples
 - Electron microscopy of faecal samples
 - Immunohistochemistry (IHC) for CPV2 antigens
 - Minor groove binder (MGB) probe assay
 - Distinguishes between CPV2 field and vaccine strains
 - PCR
 - Real-time PCR (RT-PCR)
- FPLV
 - Antigen or indirect immunofluorescence assay (IFA)
 - PCR
 - Viral isolation
- GPV
 - Viral isolation using embryonated eggs and cells from Muscovy ducks and geese

- Pool tissues of the same type in a sterile tube
- PPV1
 - Immunofluorescence assay (IFA)
 - Polymerase chain reaction (PCR)

Serological tests

- AMDV
 - Counterimmunoelectrophoresis (CIEP)
 - Lateral flow ELISA
- CPV2
 - IgM titers
- GPV
 - Agar gel precipitation (AGP) test to find GPV antibodies
- FPLV
 - Antigen-capture enzyme immunoassay (EIA)
 - Antigen-capture ELISA
 - Haemagglutination-inhibition assay (HI)
- PPV1
 - Paired acute and convalescent sera for titre evaluation
 - Antibody-capture ELISA

PREVENTION AND CONTROL

Sanitary prophylaxis

- Quarantine animals for at least 30 days when they are introduced to a captive facility to prevent the introduction of parvoviruses
- Rodent control to prevent the spread of parvoviruses; this includes destruction of rodent nests and rodenticide use
- It is advised to disinfect and clean equipment after handling mink suspected to have AMDV. Steam cleaning and spraying with 2% sodium hydroxide should be used to properly clean pens.

Medical prophylaxis

Vaccination against parvovirus is recommended for some captive wildlife. Below are examples of vaccination strategies in domestic and some wildlife species.

- AMDV
 - No vaccines are available for use in mink
- CPV2
 - Captive procyonids and palm civets should be vaccinated with a killed vaccine
 - Vaccination is not recommended for red pandas
 - Vaccination is generally not practiced in captive ursids
 - In wild canids, live-attenuated vaccines are given every 2-3 weeks starting at 6-8 weeks of age until 16-20 weeks old
 - Modified live vaccine (MLV)
 - This vaccine has been used in gray wolves (*Canis lupus*), red wolves (*Canis rufus*), and adult maned wolves (*Chrysocyon brachyurus*)
 - There is concern that this type of vaccine can cause death in juvenile animals
 - MLV is not recommended for use in maned wolf puppies until titres have been established after using a killed vaccine
- FPLV
 - Vaccination is not recommended for red pandas
 - Vaccinating cheetahs is recommended; protocols will vary by facility
 - Serology can be used to monitor antibody titres
- GPV

- Vaccinate laying geese with attenuated virus vaccine
- MEV
 - Mink can be vaccinated starting at 6-8 weeks of age with a 3-way combination vaccine against MEV, *Pseudomonas*, and botulism. The vaccine should be given annually.
- PPV1
 - Chemical-inactivated vaccine
 - Modified live vaccine (MLV)
 - Leptospirosis/Parvovirus/Erysipelas combination vaccine
 - Boars are vaccinated 6 months
 - Sows should be vaccinated at weaning
 - Gilts should be vaccinated at 5 weeks and then at 2 weeks before breeding

POTENTIAL IMPACTS OF DISEASE AGENT BEYOND CLINICAL ILLNESS

Risks to public health

- While zoonotic transmission of AMDV is uncommon, AMDV antibodies were found in two Danish mink farmers with vascular disease whose mink had a history of AMDV.

Risks to agriculture

- Parvoviruses are a threat to several farmed species as they cause ill-thrift and death, and therefore production losses for farmers.
 - AMDV and MEV are a threat to mink farmers
 - CPV2 has been reported on a raccoon dog farm in Finland
 - GPV outbreaks have been reported on goose farms in Europe and Asia
 - PPV1 is a threat to swine herds throughout the world

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<p>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.</p>
