

Feline Panleukopenia Virus

Feline panleukopenia, infectious enteritis, or parvoviral enteritis is usually caused feline panleukopenia virus (FPV; 90-95% of cases). Some cases are caused by currently circulating strains of the closely related canine parvovirus (CPV), the cause of parvoviral enteritis in dogs. This single-stranded DNA virus is highly infectious, has a tropism for rapidly dividing cells, and can cause severe illness and death in kittens and young cats lacking immunity. The virus can persist for many months in the environment. Disease severity depends on age, immune status and the presence of concurrent infections. Subclinical infection may occur in immune-competent adult cats.

How is FPV acquired and transmitted?

Transmission is by the faecal-oral route. Following oronasal exposure, the virus infects and replicates within the oropharyngeal lymphoid tissue. In most cats (i.e. those with protective immunity), an appropriate immune response is mounted, and they clear the infection at this stage without demonstrating any clinical signs. In a small number of cats (typically those that are naive or have had an inadequate response to vaccination) viraemia occurs 1 to 5 days following exposure.



Faecal shedding of virus is short-lived – typically for only 1-2 days following infection, but can be up to 6 weeks. Infectious viral particles may also be present in vomitus. However, these viruses are hardy, persisting for extended periods of time in the environment (for more than a year under favourable conditions) and therefore fomites, and people, represent the most important sources of infection.

What are the clinical signs of FPV infection?

Disease generally occurs after an incubation period of 2-10 days and can cause sudden death, clinical illness, or remain subclinical. Clinical illness can be mild consisting of anorexia and lethargy or can be more severe with fever, anorexia, weakness, dehydration, vomiting, diarrhoea (which can be watery to haemorrhagic), and rapid weight loss. Death can ensue if untreated. Infection acquired in early pregnancy can result in abortion, congenital abnormalities, or infertility. Kittens that become infected in utero or as neonates up to 1 week of age can develop cerebellar hypoplasia resulting in non-progressive ataxia. Infection in the earlier prenatal period can cause cerebral or spinal cord lesions as well as optic nerve and retinal lesions.

Bone marrow infection can cause bone marrow necrosis which leads to leukopenia (mainly neutropenia) and can also contribute to the anaemia and thrombocytopenia seen in some cases.

Reception Hours

Mon-Fri 9am - 5pm

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The ultimate cause of death in acute FPV infection is septicaemia (as a result of Gram-negative bacterial translocation from the gut), endotoxemia, and shock.

How do I diagnose FPV infection?

A presumptive diagnosis can often be tentatively made on history (e.g. unvaccinated kitten / young cat), physical examination, and clinicopathological results (e.g. profound neutropenia often alongside lymphopenia, hypoalbuminaemia, and/or hypocholesterolaemia along with various electrolyte and acid-base abnormalities). However, confirmation of a diagnosis should be attempted and absence of typical haematological changes does not rule FPV out.



All sick kittens / young cats (i.e. before their first booster vaccination) should have a faecal antigen test performed at point of admission.

CPV faecal antigen test: In clinically suspected cases, a faecal parvoviral antigen enzyme-linked immunosorbent assay (ELISA) or immunochromatographic (i.e. lateral flow) test can be performed. These tests detect virus shed in faeces and are highly specific for parvoviruses. They can be used to detect active parvoviral infection in both dogs and cats. False-positive results can occur following recent administration of modified live FPV vaccines (up to 14 days post-administration); however, a positive result in a kitten / cat showing appropriate clinical signs, even if recently vaccinated, should be assumed to have panleukopenia. False negative results can also occur (e.g., intermittent shedding)

and results should be interpreted alongside the full clinical picture. Where suspicion for FPV infection remains repeated antigen testing should be repeated after 1-2 days or FPV PCR performed.

Quantitative polymerase chain reaction (PCR): This is a highly sensitive and specific test for the presence of FPV. It is most frequently performed on faecal samples, and less frequently on dorsal tongue / oropharyngeal swabs. This test allows identification and amplification of small amounts of viral DNA. Due to the higher sensitivity, PCR assays may be useful when faecal antigen tests are negative but parvoviral infection is still suspected. However, as this is not a point-of-care test, it does not replace the faecal antigen test as the screening test of choice. PCR can infrequently detect modified live FPV vaccine strains in faeces after vaccination, but low viral load would be expected. The Molecular Diagnostics Unit offers a FPV PCR that can be used on faeces, faecal swab, whole blood, tissues, and saliva samples. It is quantitative and includes an internal control to ensure that components of faeces that could compromise the assay are

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removed during processing, but that control DNA is present (confirming adequate sampling technique and DNA extraction).

Serology (antibody testing): This test should not be used in the diagnosis of FPV infection. Interpretation is complicated by widespread exposure, vaccination or presence of maternally derived antibodies, and therefore detection of antibody is not used for diagnosis.

How is FPV prevented?

Vaccination against FPV when administered appropriately, can provide excellent immunity to FPV (and CPV) in cats. A variety of multivalent modified-live vaccines are available on the market that include attenuated FPV. However, maternally derived antibodies can interfere with the development of an appropriate adaptive immune response. The WSAVA recommend that the primary vaccination course includes administration of a dose on or after 16 weeks of age, with a booster at 6 months of age. Alternatively, pre-vaccination serology can be performed to determine whether an appropriate response has developed and whether further vaccinations are required.

How do I treat panleukopenia?

Treatment: entails correction of fluid deficits, management of hypoglycaemia (if present), broad-spectrum antimicrobials (especially if neutropenic) due to the risk of bacterial translocation, and nutritional support (including placement of a naso-oesophageal feeding tube if required). Early in the course of treatment, some cats may benefit from interferon-omega (as dogs do). Early and aggressive intervention can really improve outcome. Cats that survive the first 5 days usually recover. Cats with cerebellar hypoplasia typically do not progress and may improve slightly due to compensatory responses from other senses.



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