

## FELINE PANLEUKOPENIA/FELINE PARVOVIRUS (FPV) INFECTION

Feline panleukopenia is a highly infectious disease affecting members of the Felidae, and a number of other species including mink, ferrets, and raccoons. The disease is characterized by enteritis and a panleukopenia and has a high mortality. Vaccination is generally highly effective in controlling the disease.

Feline panleukopenia is caused by a parvovirus, a small, nonenveloped DNA virus similar to the canine parvovirus type 2 (CPV-2), which causes a severe hemorrhagic enteritis in dogs. Although feline panleukopenia is a long-established disease of cats, CPV-2 emerged suddenly in 1978 as a host range variant of FPV and spread rapidly within the dog population. The mechanism of its evolution is unknown, but it has been suggested that it originated from a wild-carnivore intermediate host. CPV-2 has largely been replaced by CPV-2a and CPV-2b, which now coexist in dog populations worldwide. Although CPV-2 isolates did not replicate in cats, both CPV-2a and CPV-2b do so and have been reported to constitute a small proportion of isolates found naturally in cats. There is also some evidence to suggest that the percentage of CPV isolates from cats in some parts of the world is increasing. As well as CPV-2a/2b, new antigenic variants called CPV type 2c have been described in Asian leopard cats and in dogs in some countries. Some have been shown to infect cats experimentally. Whether these 2c viruses are a common cause of natural infection in domestic cats is currently unknown.

Parvoviruses have an affinity and requirement for actively dividing cells. The main target tissues are the rapidly dividing cells of lymphoid tissue and the bone marrow, leading to panleukopenia, and the crypt epithelium of the intestinal mucosa, leading to enteritis. Infection early in pregnancy may lead to fetal death and resorption. From the middle third of gestation to immediately postnatally, infection may result in cerebellar hypoplasia in kittens.

The severity of the disease varies considerably, ranging from a subclinical infection to a peracute syndrome with sudden death. In general the disease appears to be more severe in young kittens. Other factors, such as a change of food or coinfection with other pathogens, which increase the mitotic rate in the intestinal villi, may increase the severity of disease. In a typical case the first signs of illness are lethargy, fever, and anorexia with apparent thirst but refusal to drink. Affected cats may vomit, particularly in the early stages. Profuse watery diarrhoea or dysentery then develops and cats may become severely dehydrated. Most fatalities occur within 3 to 5 days of the first signs of illness and are probably due to overwhelming bacterial infection, dehydration, and electrolyte imbalance. Experimental infections have suggested that infection with CPV in cats may lead to milder disease with reduced virus shedding compared to FPV, although the situation in the field is unclear.

Kittens with cerebellar hypoplasia show ataxia, incoordination, hypermetria, and often intention tremors. These signs persist for life. Nevertheless, affected kittens may learn to compensate and otherwise function normally. Forebrain lesions have been reported and may lead to seizures and behavioral abnormalities. Retinal lesions may also be present but are usually of no clinical significance. A possible role of FPV in myocarditis and idiopathic cardiomyopathy has also been suggested.

A presumptive diagnosis of feline panleukopenia may be made on history, vaccination status, and clinical signs. Disease is more commonly seen in rescue shelters and in groups of unvaccinated cats. Virus culture from fecal samples is difficult, and fecal shedding occurs only for a relatively short period after infection; electron microscopy is relatively insensitive. Enzyme-linked immunosorbent assays (ELISAs) and rapid immunomigration tests can be used for detection of viral antigen in fecal samples, with variable sensitivity and specificity. Care should be used, however, in interpreting positive test results from recently vaccinated cats, as these may be due to the presence of vaccine virus. PCR tests for the detection of viral DNA in fecal samples have also been used and these have good sensitivity. Antibody levels, which may reach very high titres following an active infection, may be useful as an aid to diagnosis, but are less useful for rapid diagnosis during an outbreak.

Diagnosis may also be confirmed at necropsy, where characteristic histopathologic changes, including the presence of intranuclear inclusion bodies, may be seen in the crypt epithelium of the small intestine. Specific diagnosis using in situ hybridization and immunohistochemistry has also been reported.

Treatment is largely supportive and is aimed at restoring the fluid and electrolyte imbalance and covering against secondary bacterial infection. Although recombinant feline interferon has been shown to be effective in canine parvovirus infection, its use in feline panleukopenia appears to be more limited.

Both modified-live and inactivated parenteral vaccines are available, and a modified-live intranasal vaccine in some countries. There is only one serotype of the virus, and the vaccines are generally highly effective in preventing disease. Protection against CPV 2b has also been shown experimentally and it is likely that FPV vaccines will protect against such isolates in the field. The AAFP recommends that modified-live injectable vaccines be used where rapid onset of protection is required, and a greater efficacy in overcoming MDA; killed vaccines are preferable in pregnant cats or disease-free colonies.

Primary vaccination usually takes place at 8 to 9 weeks of age with a second dose at 12 weeks. However, the duration of MDA in kittens can be quite variable, and those born to unvaccinated queens or those who have not suckled colostrum may have no or very low levels of MDA. In contrast, kittens born to recovered queens may have very high levels of MDA lasting up to 20 weeks. The AAFP and WSAVA vaccination guidelines therefore recommend beginning as early as 6 weeks, and repeating every 3 to 4 weeks until 16 weeks of age, with revaccination one year later. ABCD guidelines suggest vaccination at 8 to 9 weeks of age with a second vaccination at 12 to 13 weeks; earlier and later vaccination is recommended for situations with low and high MDA. Modified-live vaccines should not be used in kittens under 4 weeks of age or in pregnant queens because of the risk of the development of cerebellar hypoplasia.

Boosters have typically been administered every 1 to 2 years; however, there is good evidence that immunity may last longer, and a number of vaccines now have a 3-year claim for duration of immunity, as recommended by the AAFP, WSAVA, and ABCD.

Although the disease has largely been controlled by vaccination, cases still occur, particularly in rescue shelters and also sometimes in breeding colonies. FPV is a remarkably stable virus and may persist in infected premises for up to a year: subclinical infection of susceptible cats can also help maintain the virus within a population. Not all disinfectants are active against FPV but sodium hypochlorite and glutaraldehyde are effective. Because of the ability of the virus to survive in the environment, thorough disinfection of premises must be carried out following any disease outbreaks, and any new cats admitted to such infected premises should be fully vaccinated. Although persistent infections may occasionally occur after in utero or neonatal infection, carriers do not play a significant role in the epidemiology of the disease.