



# Feline Diseases

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## Feline Panleukopenia

Feline panleukopenia is a highly contagious, often fatal, viral disease of cats that is seen worldwide. Kittens are affected most severely. The causative parvovirus is very resistant; it can persist for 1 yr at room temperature in the environment, if protected in organic material.

### Etiology and Pathogenesis

Feline panleukopenia virus (FPV) is closely related to mink enteritis virus and the type 2 canine parvoviruses (CPV) that cause canine parvoviral enteritis. FPV can cause disease in all felids and in some members of related families (eg, raccoon, mink), but it does not harm canids.

Virus particles are abundant in all secretions and excretions during the acute phase of illness and can be shed in the feces of survivors for as long as 6 wk after recovery. Being highly resistant to inactivation, parvoviruses can be transported long distances via fomites (eg, shoes, clothing). However, FPV can be destroyed by exposure to a 1:32 dilution of household bleach (6% aqueous sodium hypochlorite), 4% formaldehyde, and 1% glutaraldehyde for 10 min at room temperature. Peroxygen disinfectants are also highly effective.

Cats are infected oronasally by exposure to infected animals, their feces, secretions, or contaminated fomites. Most free-roaming cats are thought to be exposed to the virus during their first year of life.

FPV infects and destroys actively dividing cells in bone marrow, lymphoid tissues, intestinal epithelium, and—in very young animals—cerebellum and retina. In pregnant queens, the virus may spread transplacentally to cause embryonic resorption, fetal mummification, abortion, or stillbirth. Alternatively, infection of kittens in the perinatal period may destroy the germinal epithelium of the cerebellum, leading to cerebellar hypoplasia, incoordination, and tremor. FPV-induced cerebellar ataxia has become a relatively rare diagnosis, because most queens passively transfer sufficient antibodies to their kittens to protect them during the period of susceptibility.

### Clinical and Pathological Findings

Most infections are subclinical, as evidenced by the high seroprevalence of anti-FPV antibodies among unvaccinated, healthy cats. Those cats that become ill are usually <1 yr old. Peracute cases may die suddenly with little or no warning. Acute cases show fever (104°–107°F [40°–41.7°C]), depression, and anorexia after an incubation period of 2–7 days. Vomiting usually develops 1–2 days after the onset of fever; it is typically bilious and unrelated to eating. Diarrhea may begin a little later but is not always present. Extreme dehydration develops rapidly. Affected cats may sit for hours at their water bowl,

although they may not drink much. Terminal cases are hypothermic and may develop septic shock and disseminated intravascular coagulation.

Physical examination typically reveals profound depression, dehydration, and sometimes abdominal pain. Abdominal palpation—which can induce immediate vomiting—may reveal thickened intestinal loops and enlarged mesenteric lymph nodes. In cases of cerebellar hypoplasia, ataxia and tremors with normal mentation are seen. Retinal lesions, if present, appear as discrete gray foci.

The duration of this self-limiting illness is seldom >5–7 days. Mortality is highest in young kittens <5 months old.

There are typically few gross lesions, although dehydration is usually marked. Bowel loops are usually dilated and may have thickened, hyperemic walls. There may be petechiae or ecchymoses on the intestinal serosal surfaces. Perinatally infected kittens may have a noticeably small cerebellum. Histologically, the intestinal crypts are usually dilated and contain debris consisting of sloughed necrotic epithelial cells. Blunting and fusion of villi may be present.

### Diagnosis

A presumptive diagnosis is usually based on compatible clinical signs in an inadequately vaccinated cat and the presence of leukopenia (nadir 50–3,000 WBC/ $\mu$ L). Neutropenia is a more consistent finding than lymphopenia. Total WBC counts <2,000 cells/ $\mu$ L are associated with a poorer prognosis. During recovery from infection, there is typically a rebound neutrophilia with a marked left shift. Diagnosis can sometimes be confirmed using an in-office immunochromatographic test kit intended for detection of fecal CPV antigen. However, fecal antigen is detectable only for a short time after infection and false-negative results are common.