DOI: 10.29326/2304-196X-2023-12-4-303-307

# Feline panleukopenia (review)

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**ABSTRACT**

Feline panleukopenia, a disease globally known since the beginning of the last century and originally attributed to canine distemper, has expanded its range of hosts since its discovery as a result of successful infections (both natural and experimental) in mustelids, raccoons and even primates. Evolutionarily, the disease pathogen gave rise to a new infectious agent – canine parvovirus, which, despite its DNA structure, demonstrates a relatively high mutation rate and the emergence of new variants. The disease is in most cases fatal to newborn kittens and causes severe manifestations in adult cats, severely affecting the vital systems of the body. The prognosis is often (up to 50%) unfavorable, while the animal's age plays a key role. Current preventive measures can ensure protection, however, vaccines are used in the absence of adequate testing on cats and dogs (for ethical reasons) and have a number of limitations in use. The persistence of the infectious agent in the environment and the growing number of stray animals allow the infectious agent to circulate unhindered in these populations, threatening the health of domestic cats and endangered felines in nature reserves and zoos. Easing of legislation for leading research centers, regulation of the number of stray animals, adequate prevention measures for target groups in animal shelters, nurseries and zoos can contribute to a significant reduction in the circulation in susceptible populations of pathogens not only of this disease, but also of the majority of other dangerous infections, such as rabies, feline rhinotracheitis, canine distemper and others.

**Keywords:** review, feline panleukopenia, parvoviruses, *Felidae*

**Acknowledgements:** The study was funded by the Federal Centre for Animal Health within the framework of "Veterinary Welfare" research work.

**For citation:** Kiselev A. M., Shcherbinin S. V., Galkina T. S. Feline panleukopenia (review). *Veterinary Science Today*. 2023; 12 (4): 303–307. DOI: 10.29326/2304-196X-2021-4-39-303-307.

**Conflict of interests:** The authors declare no conflict of interests.

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УДК 619:616.155.394:636.8:616.9-084(048)

# Панлейкопения кошек (обзор)

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**РЕЗЮМЕ**

Панлейкопения кошек – болезнь, известная в мире с начала прошлого века и первоначально отнесенная к чуме плотоядных, – с момента обнаружения расширила круг своих хозяев в результате успешных заражений (как в естественных условиях, так и искусственно) куньих, енотовых и даже приматов. Эволюционно возбудитель заболевания дал начало новому инфекционному агенту – парвовирусу собак, который, несмотря на ДНК-архитектуру, демонстрирует сравнительно высокую скорость мутаций и появление новых вариантов. Болезнь в большинстве случаев смертельна для новорожденных котят и вызывает сильные страдания взрослых кошек, тяжело поражая жизненно важные системы организма. Исход часто (до 50%) неблагоприятный, причем возраст кошки играет одну из ключевых ролей. Существующие меры профилактики способны защитить животных, однако вакцинные препараты используются при отсутствии адекватных испытаний на кошках и собаках (по этическим соображениям) и имеют ряд ограничений в применении. Устойчивость инфекционного агента в окружающей среде и растущее число безнадзорных животных позволяют беспрепятственно циркулировать возбудителю инфекции в данных популяциях, угрожая благополучию домашних кошек, а также вымирающих кошачьих в заповедниках и зоопарках. Послабления в законодательстве для ведущих исследовательских центров, регуляция численности безнадзорных животных, адекватная профилактика среди целевых групп в приютах, питомниках и зоопарках могут способствовать значительному снижению циркуляции в подверженных риску популяциях возбудителей не только данной болезни, но и большинства других опасных инфекций, например бешенства, ринотрахеита кошек, чумы плотоядных и других.

**Ключевые слова:** обзор, панлейкопения кошек, парвовирусы, кошачьи

**Благодарности:** Работа выполнена за счет средств ФГБУ «ВНИИЗЖ» в рамках тематики научно-исследовательских работ «Ветеринарное благополучие».

**Для цитирования:** Киселев А. М., Щербинин С. В., Галкина Т. С. Панлейкопения кошек (обзор). *Ветеринария сегодня*. 2023; 12 (4): 303–307. DOI: 10.29326/2304-196X-2021-4-39-303-307.

**Конфликт интересов:** Авторы заявляют об отсутствии конфликта интересов.

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## INTRODUCTION

Feline panleukopenia (FPL), also known as feline distemper, is a highly contagious disease of cats (*Carnivora*, *Felidae*) characterized by high mortality in newborn kittens (> 90%) with an acute course and neurological disorders such as ataxia and blindness. Older kittens develop panleukopenia, neutropenia due to bone marrow and lymphatic tissue infection, and diarrhea due to enterocyte disruption. The clinical form of the disease is most often diagnosed in animals aged 2–5 months, and subclinical or mild disease forms prevail in older cats [1, 2]. Panleukopenia in cats is caused by such subspecies of *Protoparvovirus carnivoran 1* as FPV (90–95% of cases) and some strains of canine parvovirus (< 10% of cases) [3].

Feline panleukopenia virus (FPV) can also infect raccoons (*Procyonidae*) and mustelids (*Mustelidae*). Feline panleukopenia has been known as a separate nosological unit since the 1920s, but *Canine parvovirus* (CPV) occurred as an infectious agent only in the late 1970s [2, 4]. Interestingly, FPV mutates slowly as a result of random genetic drift, while CPV demonstrates a rate of genomic substitutions similar to that of RNA viruses, with about $10^{-4}$ substitutions per site per year [5].

The disease is widespread on all continents and in most countries. Vaccines against FPL are usually developed based on avirulent virus strains with replication capacity (attenuated live vaccines) [6].

This review deals with the disease that causes high mortality in domestic cats (*Felis catus*), thus posing a great interest with regard to improving quality of life for companion animals taking into account significant economic costs on pet keeping and treatment, as well as pedigree breeding and preservation of endangered feline populations. In addition, the spillover of the pathogen to wild susceptible animals and some fur animal species makes these populations vulnerable and poses a threat of the pathogen's entry to fur farms or establishment of virus reservoirs in wild animals.

The FPL agent is a non-enveloped virus containing a single-stranded DNA (ssDNA) and an icosahedral capsid [7, 8]. It belongs to the genus *Protoparvovirus*, one of the eleven genera of vertebrate viruses in the subfamily *Parvovirinae*, the family *Parvoviridae*. The FPV and CPV jointly with associated variants found in various carnivorous species such as minks and raccoons make up the species *Protoparvovirus carnivoran 1* [9].

The virus genome consists of 5.1 kbp and contains 2 open reading frames: non-structural (NS) and structural (VP) proteins. The NS gene encodes the NS1 and NS2 proteins involved in DNA replication, capsid assembly and intracellular transport, the VP gene encodes the capsid proteins VP1 and VP2. The virus capsid consists of 60 molecules of viral proteins, approximately 10% VP1 and 90% VP2, the latter of which allows the virus to bind to the transferrin receptor (TfR) of the host cell [10, 11]. Moreover, changes in the species-specific binding of capsid proteins to the host receptor determine susceptibility to FPV or CPV [12]. This phenomenon is reflected in the adaptation of the capsid protein to the receptors of other hosts, which ensured effective interspecies distribution, as can be seen from the example of infection of cats with new strains of canine parvovirus type 2 (CPV-2) [13]. The evidence suggests that the original type 2 (CPV-2) came from FPV infecting only cats in 1978, and gave rise to the antigenic variant CPV-2a through 5–6 non-synonymous mutations in the VP gene, which led to a change in the amino acid composition of VP2 in 1979–1981 [14].

The range of hosts includes domestic and wild cats, raccoons, minks, foxes [13, 15, 16]. Domestic dogs are not susceptible to the FPL agent, since the virus cannot bind with the host's TfR target cells. However, there are studies showing that during experimental infection of dogs, FPV replicated in lymphoid tissues (thymus, bone marrow), but this was not enough for successful infection *in vivo* [17, 18]. At the end of the XX century, the viral DNA was isolated from the faeces and formalin-fixed small intestines of captive cheetahs (*Acinonyx jubatus*) and free-living African wildcat (*Felis lybica*) and honey badger (*Mellivora capensis*) [15]. In 2008 FPV was recovered from 500 macaques demonstrating signs of hemorrhagic enteritis in the Chinese Experimental Animal Centre. The virus was identified morphologically, genetically, and the result of bioassay performed in felines was positive [19]. In 2022 a 9-month-old leopard (*Panthera pardus*) was diagnosed with panleukopenia in the Wildlife Rescue Center in India. This endangered feline species had been brought from a Transit Animal Treatment Center, which was, in fact, a shelter for providing care and treatment to wild animals. The genome sequence of the isolated virus was 99.14% similar to the sequence of nucleotides recovered from the raccoon virus [20]. An interesting case of FPV isolation from a banded linsang (*Prionodon linsang*) in Thailand was described by N. Inthong et al. in 2019. Amino acid analysis of VP2 protein revealed a high level of homology (over 98%) with FPV. The isolate was closely related to the FPV strains from Japan, South Korea and China [21].

The pathogen is transmitted through direct contact with the secretions of infected animals, including faeces, blood, urine. In addition, there is a vertical transmission pathway, while infection can result in abortion, mummification and stillbirth [22].

Parvoviruses are extremely stable in the environment, and indirect transmission probably plays an important role in spreading and maintaining the agent circulation in a population, especially in wild carnivores. Transmission between domestic and wild carnivores is assumed to be easy, and fomites may be factors in long-distance transmission. Apparently, this causes high mortality in intact populations [23].

In populations where parvoviruses are constantly circulating, new cases occur mainly among young animals that get infected due to decrease in colostral antibody titers, and the infection dynamics in seasonal breeders may strongly depend on stock replacement, often resulting in cyclical incidence [23].

After FPV entry into the cell via clathrin-mediated endocytosis, endosomes with a virion fuse with the nuclear membrane. After the virus is released from the capsid in the nucleus, it uses the DNA polymerase of the host cell for replication [24, 25]. Since the virus can replicate only in actively dividing cells (occurring in the S-phase of mitosis), it possesses tropism to lymphoid tissue, bone marrow, epithelium of intestinal crypts and other actively dividing cells of newborn kittens. FPV can replicate in Purkinje cells of the cerebellum of kittens under 10 days of age [26]. The tissue tropism determines the clinical signs and pathological-anatomical picture.

The incubation period lasts 4–5 days, and the clinical course of the disease can rapidly develop causing death. The gate of infection are the palatopharyngeal tonsils, after which viremia develops rapidly. The primary pathological site of virus replication is located in the intestinal crypts due to the high mitotic activity of the latter, causing severe enteritis and diarrhea. The virus also targets lymphoid tissue, which leads to pancytopenia (less than 4,000 cells/μL). In the later stages of the disease it can be observed that a white blood cells count returned to normal. In some cases, icterus may also be noted [22].

The disease clinical signs demonstrate the nervous system pathology (depression, ataxia, anorexia), fever, the digestive tract pathology (vomiting, diarrhea), hypersalivation [19, 27, 28, 29]. The development of the clinical picture directly depends on the age of the animal. Thus, the virus replicates in multiple tissues and often causes cerebellar hypoplasia, and, consequently, nervous disorders in newborn animals. The virus replication is limited to lymphoid cells and small intestine cells, causing temporary panleukopenia and diarrhea in older animals [30]. Interestingly, newborn animals show no signs of diarrhea, probably due to a lower rate of reproduction of intestinal epithelial cells at the beginning of life, but infection of fetuses and newborns usually leads to death or irreversible disabling damage to organ systems [31].

Necropsy performed in kittens and adult cats showed the following common pathological and anatomical picture: local small- and large-focal enteritis with spot and/or petechial hemorrhages in the serous membrane. Lesions are most pronounced in the jejunum and ileum. Thickening of the intestinal walls accompanied by edema and hemorrhagic lymphadenitis of the mesenteric lymph nodes are often found [32, 33]. Histological lesions in the small intestine include multi-focal necrosis and loss of crypt architecture. The effects of secondary bacterial infection are also observed. FPV is known to be teratogenic in case of intrauterine infections. In the last stages of pregnancy, the virus targets mitotically active brain and eye tissues. This leads to cerebellar hypoplasia, hydrocephalus, and retinal dysplasia [34, 35].

The DNA molecule of *Protoparvovirus carnivoran 1* persists for a long time in the tissues of convalescent animals, leaving behind a molecular trace [16]. The virus can remain latent in peripheral blood monocytes, as evidenced by the successful FPV cultivation from monocytes of healthy cats with high titers of virus neutralizing antibodies [36, 37, 38]. The antibody-mediated immune response viewed as a production of virus neutralizing antibodies is prevalent in FPV-induced infection. Colostral immunity plays an important role in the protection of newborns. The infection mainly occurs in young animals aged 2–4 months. Cell-mediated immunity also plays an important role in the recovery [31].

The treatment of animals with panleukopenia primarily includes transfusion therapy with electrolyte replenishment. Since the disrupted intestinal crypt architecture contributes to bacteremia, and intensification of neutropenia aggravates this process and often leads to sepsis, antibacterial therapy with broad-spectrum drugs (especially against gram-negative and anaerobic bacteria) is required. An easily digestible diet is preferred, feeding should not be suspended. It is important to understand that many cats with panleukopenia, especially those that come from shelters, also have a parasitic infection, and therefore coprolarvoscopy, coproovoscopy and appropriate treatment with anthelmintics is an important factor, since intestinal parasitosis is a common concomitant disease [10, 22, 39].

As previously reported, FPV is highly resistant to environmental factors, as well as to many detergents [39, 40, 41]. In shelters, staff may act as mechanical virus carriers and, therefore, pose a hazard to unvaccinated cats [42]. Diseased carnivores shed the pathogen in high titers (up to $10^9$ $TCID_{50}$ per gram of faeces), and the virus quickly accumulates in nurseries and shelters, since the latter are characterized by initial populations of animals with an unknown vaccination history and frequent personnel turnover. Due to the high contagiousness of FPV, susceptible animals can get infected even after thorough disinfection of premises [40]. There is an acute problem of high feline mortality due to FPL in shelters in Europe, the USA and Australia [10, 43, 44]. Therefore, it is recommended that only kittens and cats subjected to successful vaccination are placed in such an environment [40].

Live and inactivated vaccines against FPL inducing stable immunity levels in animals have been developed. Live vaccines generally induce rapid development of protective antibodies in immunocompetent cats [45]. However, even a single dose of an inactivated vaccine against FPV can confer a sufficient antibody-mediated response in previously uninfected cats within a short period of time [46]. Despite this, there are some restrictions on the use of live attenuated vaccines: 1) the vaccine shall not be used in pregnant females due to the risk of virus penetration to the fetus and subsequent dysfunction of the developing cerebellum; 2) the vaccine shall never be administered to kittens under 4 weeks of age for the same reason: to avoid damage to the cerebellum, which is still developing in newborn kittens. At the moment, there are no studies available aimed at finding out what vaccine type or manufacturer are more effective. Due to the high resistance of the virus in the environment and the wide spread

of the disease in the world, all cats are at risk of infection. Animals that are mostly kept in households can become infected via fomites. Therefore, vaccination is recommended for all cats lacking adequate immunity [47].

As a rule, the colostral antibody titers in kittens decrease to a threshold level by 12 weeks of age, so the first vaccination is carried out at the age of 8–9 weeks, and followed by revaccination in 3–4 weeks. The FPL vaccination strategy should be based on the preliminary determination of maternal antibody titers, since their high concentration in animal blood can lead to the neutralization of the virus vaccine strains included in live vaccines [6, 47, 48]. In this regard, the advantage of inactivated vaccines should be emphasized, since the immune response induced post administration is not affected by the level of colostral antibodies.

Recent amendments in the Russian legislation [49], having an ethical background, may significantly limit tests of preventive and therapeutic medicinal products in target animals. This state of affairs forces us to receive incomplete and not always reliable information about the effectiveness of such products, which means that it puts animal populations that are in need of protection from infection, in a vulnerable position.

*Forecast*. FPV can cause a serious and potentially fatal disease in cats; 30–50% of diseased animals die despite intensive treatment [50, 51].

According to F. Porporato et al. [52] and F. Ferri et al. [53], either FLV immunocompetent cats or cats without signs of depression and with a higher body weight during admission to the clinic demonstrated a high survival rate. There is a high probability of an unfavorable outcome on day 3 of hospitalization or later even with adequate leukopenia treatment provided.

## CONCLUSION

Feline panleukopenia, known for more than a hundred years, still remains a serious feline concern that is not receiving enough attention in the world. Strict ethical standards for companion animals may limit the adequate implementation of preventive measures. The high degree of the pathogen's resistance in the environment poses a threat of its entry into animal husbandry and zoos, as well as the establishment of wildlife reservoirs. Veterinary services in most world countries need to pay closer attention to this feline infection in the era of significant costs to ensure safe coexistence of companion animals and humans.

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*Received 26.07.2023*
*Revised 23.08.2023*
*Accepted 30.10.2023*

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