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An Integrated Review of Feline Calicivirus, Panleukopenia Virus, Herpesvirus, and Coronavirus: From Viral Entry to Disease Control

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Abstract. This article reviews four common viral pathogens in domestic cats: feline calicivirus (FCV), feline panleukopenia virus (FPV), feline herpesvirus type 1 (FHV-1), and feline coronavirus (FCoV). Although these four viruses are widespread, their biological characteristics and clinical manifestations differ significantly. FCV is an RNA virus with a high mutation rate, which explains the wide range of clinically observed disease severity—from relatively mild oral and respiratory symptoms to severe systemic illness associated with vasculitis. In contrast, FPV is a highly stable DNA parvovirus that primarily attacks rapidly dividing cells, particularly those in the intestinal crypts and bone marrow, leading to enteritis, severe leukopenia, and a high mortality rate, especially in kittens. FHV-1 primarily causes upper respiratory and ocular diseases, but because it can establish latent infection in sensory ganglia, the infection is usually lifelong and prone to relapse under stress. Feline coronavirus (FCoV) is typically confined to the gut and may cause only mild gastrointestinal symptoms; however, in some cats, viral mutations enable it to replicate within macrophages, ultimately leading to feline infectious peritonitis (FIP), a serious immune-mediated disease. In recent years, the advent of antiviral drugs (such as GS-441524) has significantly improved the prognosis of FIP. In summary, these infections remain a major clinical problem, and their control still relies on vaccination, timely diagnosis, appropriate supportive care, and targeted antiviral therapy when feasible.

Keywords: Feline calicivirus, eline panleukopenia virus, feline herpesvirus type 1, feline coronavirus, Therapy

1. Introduction

Cats belong to the domesticated member (felid) of the Felidae [1]. Domestic cats have strong bodies, keen senses, retractable claws, long tails, and teeth especially suitable for hunting [2]. The size and weight of cats vary greatly between sexes and breeds. In general, adult male cats have an average body length of 28 inch while adult female cats' average length is 20 inch [3]. For a healthy cat, the average weight is between 8 and 12 pounds. Cats are much more difficult to domesticate than dogs. In the more than 30,000 years of domestication, dogs have changed fundamentally compared to wild dogs, while domestic cats are almost identical to wild cats [4] As a kind of pet, the cat is of great

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help to the healthy development of people's psychology. Cats, like humans, develop a variety of diseases during their growth. However, some diseases of cats are not well studied.

Feline calicivirus (FCV), Feline panleukopenia virus (FPV), Feline herpesvirus (FHV) and Feline coronavirus (FCoV) have a great effect on cats. FCV is a single-stranded positive-sense RNA virus, which belongs to the genus Vesivirus of the Caliciviridae family. The virus particles have icosahedral structure, no capsule, and can infect almost all felids. FCV has a strong infectivity, when cats infected, they will show symptoms of respiratory and oral diseases [5]. This virus exists in multiple strains with varying levels of virulence. Some strains cause relatively mild clinical signs, such as nasal discharge, whereas others can lead to severe and potentially fatal disease. This diversity among strains partly explains why vaccination does not always provide complete protection, as available vaccines may not cover every circulating variant. The virus is primarily transmitted through direct contact between cats, including nose-to-nose interaction and grooming. Indirect transmission can also occur via contaminated fomites such as food bowls, litter boxes, or human hands after contact with infected animals. Notably, the virus demonstrates considerable environmental stability and may survive on dry surfaces for more than a week. As a result, once introduced into an environment, it can be difficult to eliminate [6]. After entering the body through the mouth, nose, or eyes, the virus begins replicating at the initial site of infection. From there, it can spread through the bloodstream to other tissues. The virus primarily infects epithelial cells lining the mouth and respiratory tract. In particularly virulent strains, infection may also extend to the cells that make up blood vessel walls. In many cases, the severity of illness in cats isn't determined by the virus alone. It also depends on the individual animal, especially how strong its immune response is. Less virulent strains often lead to relatively mild, localized symptoms. However, highly pathogenic strains can provoke an overwhelming inflammatory response that spreads throughout the body. When that level of systemic inflammation occurs, the outcome is often fatal [7].

FPV, also known as feline parvovirus or feline panleukopenia virus, is one of the most serious infectious pathogens in felines, especially kittens. The term "panleukopenia" describes the characteristic significant reduction in peripheral blood leukocytes [8]. FPV is a non-enveloped single-stranded DNA virus with a genome size of approximately 5.1 kb, classified as proparvovirus type 1. FPV infection usually occurs via the oral-nasal route. After initial replication in the oropharyngeal lymphoid tissue and regional lymph nodes, the virus enters the bloodstream and spreads throughout the body. FPV uses the transferrin receptor of host cells to enter cells, which is highly expressed on rapidly dividing cells. Therefore, the virus mainly attacks tissues with high mitotic activity, especially the bone marrow and intestinal crypt epithelium. Damage to these sites hinders hematopoiesis and intestinal epithelial renewal, leading to the severe leukopenia and enteritis seen clinically [9].

Feline herpesvirus type 1 (FHV-1) is an enveloped double-stranded DNA virus classified within the genus Varicellovirus of the subfamily Alphaherpesvirinae [10]. Cats infected with FHV-1 mainly cause feline viral rhinotracheitis and ocular disease, and may also cause severe symptoms such as pneumonia, gastritis and so on [11]. There are two types of FCoV: Feline Infectious Peritonitis Virus (FIPV) and Feline Enteric Coronavirus (FECV), of which, FIPV is more virulent. FCoV is a nonsegmented, single-stranded RNA virus that belongs to the order Nidovirales, family Coronaviridae. For FIPV biotype, cats develop symptoms of lassitude, inappetence, dehydration, diarrhea after infection. These diseases have a great impact on both the animal itself and the owner's economy and psychology [12]. Therefore, how to reduce the morbidity and improve the recovery rate or survival rate after the disease is particularly important.

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In this paper, the basic characteristics, infection mechanism, prevention and treatment methods of the above four viruses will be described, and the four viruses will be compared to find the similarities and differences, and the characteristics of each virus will be summarized, so as to have a deeper understanding of the four viruses, help to reduce the morbidity of animals infected with the virus and improve the recovery rate or survival rate after the disease.

2. FCV

2.1. Information of FCV

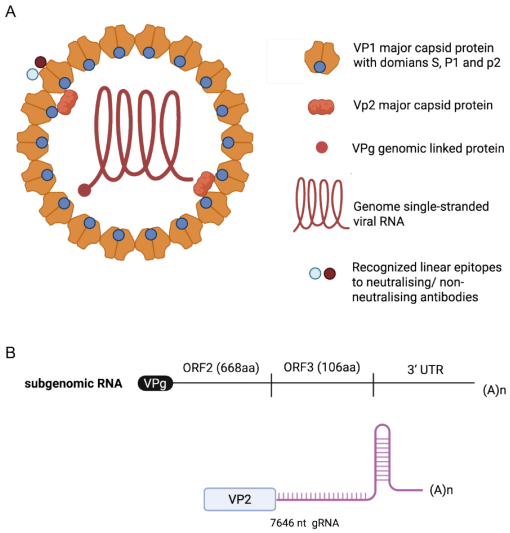


Figure 1. FCV. A. FCV viral structure is depicted schematically. B. Analysis of the genomic structure of FCV

In 1957, FCV was first isolated from the gastrointestinal tract of cats in New Zealand [13] FCV is a non-enveloped, single-stranded, plus-strand RNA virus with a genome of about 7.5kb, a 20-hedral symmetry, and a diameter of about 27 to 40nm (Figure 1a). Although FCV is classified as a single species taxonomically, it exhibits marked heterogeneity in both epidemiology and clinical manifestations. Numerous genotypes and strains of FCV circulate in the cat population, and the virus evolves rapidly, with particularly pronounced variability in the capsid protein VP1 region. As a result, pathogenicity varies substantially among different strains. Based on clinically relevant strain phenotypes, FCV can be broadly categorized into the classical form—characterized mainly by upper respiratory signs and oral ulceration—and the virulent systemic form, which may induce vasculitis, a systemic inflammatory response, and even high mortality [14]. There are three overlapping open reading frames (ORFs) in FCV genome, ORF 1-3, encoding non-structural proteins, the VP1 (viral protein 1) major capsid protein and the VP2 (viral protein 2) minor capsid protein, respectively. Non-structural proteins have six subtypes including NSP1–NSP6/7, which is hydrolyzed state of a large polymeric protein precursor, encoded by ORF 1. ORF 2 encodes the precursors of VP1 major capsid proteins that are cleaved after translation, and then the mature capsid proteins and the leader of the capsid protein (LC) are released. ORF3 encodes the minor capsid protein VP2. Electron microscopy reveals 32 cup-shaped depressions on the virion surface, arranged with icosahedral symmetry. This morphology gives rise to the family name "Caliciviridae." The depressions reflect the geometric organization of capsid protein subunits [15]

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2.2. Infectious mechanism

FCV can be transmitted both directly, through close contact between cats, and indirectly via contaminated fomites or aerosolized secretions. Variability in viral–receptor interactions contributes to differences in tissue tropism, disease severity, and host range, making receptor usage an important determinant of pathogenesis.

FCV primarily infects epithelial cells of the oropharynx and upper respiratory tract. Adhesion molecule-A, a transmembrane protein expressed on epithelial cells, is its main functional receptor. Glycosaminoglycans, including heparan sulfate, may promote initial attachment by increasing the local concentration of the virus on the cell surface [16]. After binding to the receptor, the virus enters the cell via clathrin-mediated endocytosis. As a non-enveloped virus, FCV does not fuse directly with the plasma membrane; instead, the internalized viral particles are transported in endosomes, and the capsid protein undergoes conformational changes as the endosomes gradually acidify. These structural rearrangements allow release of the positive-sense RNA genome into the cytoplasm. The genomic RNA functions directly as mRNA and is translated into a large polyprotein, which is subsequently cleaved into non-structural proteins, including RNA-dependent RNA polymerase (RdRp). Structural proteins, particularly VP1, are produced and assemble into icosahedral capsids in the cytoplasm. Newly synthesized genomic RNA is packaged into assembling particles to generate mature virions [17]. Progeny viruses are mainly released through cell lysis, which contributes to epithelial damage and the development of oral and respiratory lesions. Viremia enables dissemination to other tissues, including synovial membranes and, in virulent systemic strains, vascular endothelium, where infection is associated with vasculitis and systemic complications. Receptor specificity, particularly interaction with JAM-A, plays a central role in determining viral tropism and disease expression.

2.3. Prevention and treatment

There is no specific antiviral therapy approved for FCV. Management focuses on supportive and symptomatic care to help the cat's immune system clear the infection. Foundational Care: Fluid therapy to correct dehydration and nutritional support (e.g., appetite stimulants, assisted feeding) are critical. Symptom Control: Broad-spectrum antibiotics are used for secondary bacterial infections. Pain relief, anti-inflammatories, and mucolytics address oral ulcers, fever, and respiratory congestion. Cui et al. 's team found that equine immunoglobulin F(ab')2 fragments, nitazoxanide, mizoribine, icariin (ICA), formononetin (FMN), caffeic acid phenethyl ester (CPAE) are potential agents for the treatment of FCV infection [18].

3. FPV

3.1. Information of FPV

The earliest recognition of FPV dates back to 1928. Its genome consists of single-stranded DNA and spans about 5 kb in length [19]. FPV virions are small (approximately 25 nm in diameter), non-enveloped particles with icosahedral symmetry and a single recognized serotype. The genome contains two major open reading frames located at opposite ends: the 5′ region encodes the non-structural proteins NS1 and NS2, whereas the 3′ region encodes the capsid proteins VP1 and VP2. Transmission occurs mainly through the fecal–oral route. Owing to its marked environmental stability, the virus can remain infectious for prolonged periods outside the host. FPV preferentially

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infects rapidly proliferating cells, especially hematopoietic progenitor cells in the bone marrow and epithelial cells in the intestinal crypts. Damage to these tissues is the underlying cause of severe leukopenia, immunosuppression, and severe enteritis during infection. Clinically, infected cats (especially kittens or unvaccinated cats) typically present with fever, vomiting, diarrhea, dehydration, and may have a high mortality rate.

3.2. Infectious mechanism

FPV primarily targets rapidly dividing cells, which explains its marked effects on bone marrow, lymphoid tissues, and intestinal crypt epithelium. Damage to these tissues results in the characteristic clinical manifestations of the disease, including severe leukopenia and enteritis. These pathological changes arise largely from the depletion of hematopoietic progenitor cells and the disruption of normal epithelial renewal. FPV enters susceptible cells through interaction with the type 1 transferrin receptor (TfR1), a receptor highly expressed on proliferating cells. The capsid protein VP2 mediates this binding process, thus restricting viral replication primarily to tissues with high mitotic activity. After attachment, the viral particle is taken up by clathrin-mediated endocytosis. Acidification of the endosome compartment induces capsid rearrangement, including exposure of functional domains within VP1, allowing the virus to escape into the cytoplasm. Unlike many RNA viruses, FPV replication occurs within the cell nucleus. The intact viral particle is transported into the nucleus via the nuclear pore complex using capsid-associated nuclear localization signals and host import proteins. Once inside the nucleus, the single-stranded viral DNA genome is converted into a double-stranded form by host cellular enzymes. Transcription is then carried out by host RNA polymerase II, producing mRNAs that encode several non-structural proteins, most notably NS1. This protein plays a critical role in viral DNA replication through a rolling-circle hairpin mechanism. Because this process depends on the host cell's S-phase mechanism, viral replication is closely related to cell proliferation. The structural proteins synthesized in the cytoplasm then re-enter the nucleus, where capsid assembly and genome packaging occur. The gradual accumulation of viral components disrupts normal cellular processes, ultimately leading to cell lysis and the release of progeny viral particles to infect neighboring cells [20].

3.3. Prevention and treatment

To date, no specific antiviral drugs for FPV have been approved. Existing treatments are primarily supportive and symptomatic, aiming to help cats through the critical viremia phase until their immune system can clear the virus. The main measures are as follows: Aggressive Fluid Therapy: Intravenous crystalloids (e.g., lactated Ringer's solution) to correct severe dehydration, electrolyte imbalances (hypokalemia), and hypoglycemia. Secondly, antiemetics are employed to control the relevant symptoms. Maropitant citrate (a neurokinin-1 receptor antagonist) is the antiemetic of first choice for controlling persistent, life-threatening vomiting, and its effect can be enhanced when combined with ondansetron (a 5-HT3 receptor antagonist). A few reports have suggested that recombinant feline omega interferon might improve clinical outcomes in cats with FPV infection, but the evidence so far is limited and has not been consistently reproduced. By comparison, vaccination remains the most reliable preventive strategy. The relative antigenic stability of FPV likely contributes to the sustained effectiveness of available vaccines [21].

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4. FHV-1

4.1. Information of FHV-1

FHV-1 is an enveloped double-stranded DNA virus belonging to the subfamily Aherpesviruses. The FHV-1 viral particle consists of a DNA genome packaged within an icosahedral capsid, surrounded by a cortex and a lipid envelope carrying viral glycoproteins. These glycoproteins mediate viral attachment to epithelial cells (especially in the upper respiratory tract and conjunctiva, where acute infections frequently occur). After initial replication, FHV-1 remains latent in sensory neurons. Reactivation may occur following a stress response, leading to recurrence of clinical symptoms. Compared to non-enveloped viruses, the presence of the lipid envelope reduces FHV-1's stability in the environment but facilitates its entry into host cells. Due to its long latency period and ability to periodically reactivate, FHV-1 infection is persistent and recurrent [22].

4.2. Infectious mechanism

The infection mechanism of FHV-1 is a biphasic process, consisting of an initial acute lysis phase in the mucosal epithelium and a subsequent establishment of lifelong latent infection in the neural tissue [23].

FHV-1 first replicates in the epithelial cells of the conjunctiva and upper respiratory tract. FHV-1 glycoproteins (such as gD) promote its binding to cell receptors, mediating the fusion of the FHV-1 envelope with the cytoplasmic membrane. Next, the FHV-1 nucleocapsid is released into the cytoplasm and transported to the nucleus via microtubules, while its DNA enters the nucleus through nuclear pores. FHV-1 gene expression follows strict temporal regulation, allowing for genome replication and capsid assembly within the nucleus. Mature FHV-1 particles are released via exocytosis or, in some cases, with cell lysis, leading to epithelial damage induced by acute infection. Some FHV-1 particles invade the sensory nerve endings at the site of primary infection and reach the neuronal cell body (usually located in the trigeminal ganglion) via retrograde transport. Within the neuron, the FHV-1 genome is circularized and persists in a nonproductive state. Reports have confirmed that FHV-1 reactivation can be induced by bodily stress, immunosuppression, or exposure to corticosteroids. The virus can restart replication, and newly assembled viral particles are transported antegradely along the axon back to the mucosal surface, thus causing viral shedding. This process is sometimes accompanied by recurrent clinical symptoms, and sometimes there are no obvious symptoms [24].

4.3. Prevention and treatment

FHV-1 has the ability to reactivate under certain conditions, which is why it can infect cats for life. Since the virus cannot be completely cleared after the initial infection, the management of FHV-1 infection focuses on controlling clinical symptoms rather than eliminating the virus. Antiviral treatment, such as oral famciclovir, is commonly used during acute exacerbations of FHV-1 infection; for cases of ocular infection in cats, cidofovir can be applied topically. Treatment for FHV-1 usually requires fluid and nutritional support, and antibiotics may be used as appropriate when secondary bacterial infection is suspected. Prevention of FHV-1 primarily relies on routine vaccination. While the widespread use of modified live vaccines and inactivated FVRCP vaccines cannot completely prevent infection or stop latent infection, they can effectively reduce the severity

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of clinical disease. In multi-cat environments, it is generally recommended to isolate infected animals and reduce environmental stressors to limit viral transmission and disease recurrence [25].

In summary, primary prevention of FHV-1 is mainly through vaccination. During acute exacerbations of FHV-1 infection, antiviral drugs such as famciclovir/cidofovir are used to alleviate clinical symptoms. At the same time, stress should be reduced to avoid recurrent FHV-1 infection.

5. FCoV

5.1. Information of FCoV

Feline coronavirus (FCoV) infects domestic cats and is commonly discussed in relation to two clinical outcomes: enteric infection and feline infectious peritonitis (FIP), the latter arising from mutations that occur within the virus in certain individuals. FCoV is an enveloped RNA virus with characteristic spike proteins, but in most cases infection remains confined to the intestine and may cause only mild or transient gastrointestinal signs, if any. A proportion of infected cats never develop noticeable clinical disease. In some animals, however, viral mutations allow replication within macrophages, which is associated with the development of FIP. This condition may present with persistent fever, weight loss, effusions, or organ involvement, depending on the form of disease. Diagnosis relies on the overall clinical picture together with laboratory and imaging findings, as detection of FCoV alone is insufficient to confirm FIP.

5.2. Infectious mechanism

FCoV has an envelope carrying the spike protein and replicates primarily in mature epithelial cells of the distal small intestine and colonic villi. Typically, FCoV infection causes only mild or transient diarrhea, and subclinical infection is also common. Infected cats shed large amounts of the virus in their feces, causing environmental contamination and spreading in multi-cat environments. Other cats require prolonged and continuous shedding of the virus, and only a few are able to completely clear FCoV [26].

Feline infectious peritonitis occurs in some cats infected with FCoV and is caused by mutations in the FCoV gene during intestinal replication, such as mutations in the spike protein gene. These mutations are associated with changes in viral cell tropism. After such mutations occur, FCoV acquires the ability to replicate in monocytes and macrophages, and once these cells are infected, FCoV can spread throughout the body via the bloodstream. Infected monocytes accumulate in the walls of blood vessels and perivascular tissues, especially on the surface of serous membranes and in various organs, triggering a strong inflammatory response. Whether the clinical manifestation is exudative or non-exudative seems to depend on the strength of the host cell-mediated immune response and the extent of vascular involvement [27].

In summary, whether FCoV infection is a localized intestinal infection or a fatal systemic disease depends on whether FCoV mutates during replication. Mutated FCoV can hijack the host's immune cells, using them as vectors for spread, and ultimately triggering a severe, immune-mediated pathological process.

5.3. Prevention and treatment

Strategies for preventing FCoV infection primarily focus on limiting fecal-oral transmission, especially in multi-cat environments. Due to the prevalence of feline enteric coronaviruses and the lack of a stable and effective vaccine, practical control measures rely heavily on hygiene

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management, including frequent litter box cleaning and reducing environmental contamination. Treatment for FCoV primarily targets cases that develop into feline infectious peritonitis (FIP). In recent years, antiviral drugs such as the nucleoside analog GS-441524 have been widely used with good clinical results. Treatment for FCoV infection typically lasts approximately 12 weeks, combined with supportive care to control inflammation and other complications. While relapses of FCoV infection are possible, with appropriate antiviral treatment, FIP is no longer considered an absolute terminal illness [28].

6. Conclusion and future perspectives

This review examines four viral pathogens commonly encountered in feline populations: feline calicivirus (FCV), feline panleukopenia virus (FPV), feline herpesvirus type 1 (FHV-1), and feline coronavirus (FCoV). Although these viruses differ in genome organization, replication strategies, and clinical presentation, all are readily transmitted and display marked tissue tropism. FCV and FHV-1 are typically spread through respiratory and ocular secretions and mainly affect the upper respiratory tract and oral mucosa. FPV and FCoV are transmitted primarily by the fecal–oral route. FPV targets rapidly dividing intestinal and hematopoietic cells, whereas FCoV initially replicates in intestinal epithelium and, in some cases, acquires the ability to replicate in macrophages.

Viruses enter cells through the interaction between proteins on their surface and receptors on the host cell. Specifically, FCV uses adhesion molecule A, FPV binds to type 1 transferrin receptors, and FHV-1 targets cellular receptors including gangliosides. FCoV, however, has some unique characteristics; if it mutates during replication in the gut, it can alter its cell tropism, allowing it to infect monocytes and macrophages. It is these diverse receptor interactions that ultimately determine which cells and tissues the virus prefers to attack, and what kind of pathological changes it causes.

Prevention and treatment depend on the specific characteristics of the virus and the availability of effective treatments. Supportive care is often necessary, especially against FCV and FPV infections. Vaccines are commonly used for FPV and FHV-1, with FPV vaccines generally offering more reliable protection because their antigenicity is relatively stable and doesn't change much. FCV is different; the strains are diverse, and the pathogenicity of FCoV depends on viral mutation, making the effectiveness of vaccines uncertain. Therefore, in multi-cat environments, maintaining good hygiene and environmental management becomes paramount. A major change in recent years has been the emergence of antiviral drugs, such as GS-441524. Its advent has directly changed the treatment landscape for feline infectious peritonitis—previously, only supportive care was available; now, there are finally real treatment options. Of course, to find better prevention and treatment methods, further in-depth research is needed on the pathogenic mechanisms of viruses and how hosts respond to them.

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