Review Article

FELINE PANLEUKOPENIA VIRUS INFECTION: A COMPREHENSIVE REVIEW (2010–2025)

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ABSTRACT

Feline panleukopenia (FPL) is a highly contagious viral disease caused by feline panleukopenia virus (FPV), affecting cats across all age groups. FPV is classified under Carnivore Protoparvovirus 1, within the genus Protoparvovirus of the Parvoviridae family, which includes three subfamilies: Parvovirinae, Densovirinae and Hamaparvovirinae. Clinical severity varies with age, immune competence and the presence of secondary infections. Typical signs include lethargy, anorexia, fever, vomiting, diarrhea, and leukopenia. Diagnosis is based on clinical signs and hematological findings, confirmed through polymerase chain reaction (PCR) or rapid antigen detection kits. Recent diagnostic advances, such as nano-PCR and isothermal amplification techniques, have enhanced detection speed and sensitivity. Treatment is supportive, as no specific antiviral exists. Core interventions include intravenous fluids, electrolyte correction, antiemetics, and broad-spectrum antibiotics to manage secondary bacterial infections. Nutritional support is vital for recovery. In severe cases, blood transfusions or immunomodulators such as interferon-omega or granulocyte colony-stimulating factor (G-CSF) may be indicated. Early intervention and strict biosecurity are critical for successful outcomes and disease control.

Keywords: Feline panleukopenia virus (FPV)

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INTRODUCTION

Feline panleukopenia (FPL) is a severe and often fatal viral illness, especially in unvaccinated cats. The disease has been described under various names based on regional use and clinical presentation as feline agranulocytosis, spontaneous agranulocytosis, feline distemper, laryngo-enteritis, feline parvoviral enteritis, feline infectious enteritis, pseudomembranous

enteritis, cat plague, feline ataxia and malignant panleukopenia (Tuzio, 2021; Rehme *et al*., 2022). The disease is characterized by clinical signs such as loss of appetite, lethargy, fever, vomiting, and diarrhea. The term "panleukopenia" denotes the significant decline in circulating white blood cells typically observed in infected cats (Greene, 2012). High seroprevalence rates observed in some unvaccinated adult cat populations indicate that subclinical infections are likely common among young adults. The clinical manifestation of feline panleukopenia is influenced by various factors, including the cat's age, immune competence, and concurrent infections with bacteria, viruses, or intestinal parasites (Barrs, 2019). The virus is transmitted primarily through the fecal-oral route and demonstrates a marked tropism for rapidly dividing cells, especially within lymphoid tissues and the intestinal mucosal epithelium. This cellular preference results in pronounced immunosuppression and acute enteritis (Greene, 2012).

FPV is primarily transmitted through direct contact with infected cats and their secretions (Cotmore *et al*., 2019), though flies may also serve as mechanical vectors, especially in warmer seasons (Mietzsch *et al*., 2019). Carnivore protoparvovirus 1 as the causative agent of feline panleukopenia (FPL), a highly contagious and environmentally resilient virus within the Parvovirinae subfamily. The virus is a small, non-enveloped, single-stranded DNA virus with a 5.1-kb genome encoding nonstructural (NS1, NS2) and structural (VP1, VP2) proteins essential for replication and capsid formation. It can survive for over a year in shelters and withstands extreme conditions, including high temperatures and low pH (Barrs, 2019).

Six critical amino acid residues within the VP2 capsid protein differentiate canine parvovirus (CPV) from FPV, facilitating CPV's specific interaction with the canine transferrin receptor (TfR) and subsequent cellular entry through clathrin-mediated endocytosis. While CPV-2 is capable of replicating in feline cell cultures under in vitro conditions, it fails to replicate effectively in feline tissues in vivo. The infection is associated with exceptionally high mortality rates, approaching 100% in peracute cases and approximately 90% in acute presentations (Uyurca and Haydardedeoglu, 2025).

Given the high transmissibility of FPL, infected cats should be strictly isolated and managed with rigorous barrier nursing to prevent fomite-mediated transmission. As no specific antiviral therapy exists, treatment primarily involves supportive care. Granulocyte-colony stimulating factor (G-CSF), a key cytokine produced in the bone marrow, promotes granulopoiesis by accelerating granulocyte maturation and release. Recombinant human G-CSF (rhG-CSF), or Filgrastim, has been effectively employed to manage FPL-induced neutropenia and is associated with improved clinical outcomes (Uyurca and Haydardedeoglu 2025).

Severe cases of FPL involving dehydration, hypovolemia, sepsis or SIRS/shock require aggressive supportive care using intravenous crystalloids and colloids, antacids, antiemetics, B-complex vitamins and broad-spectrum antibiotics. Vitamin B12 injections support recovery when administered with broad-spectrum injectable antibiotics, while diluted B-complex vitamins require cautious use. Frequent monitoring of body temperature is necessary, and fluids or NSAIDs should be used to reduce it if it rises above 104°F. (Rice 2017). Mild fevers under 103.5°F may aid the immune response and typically need only hydration and observation (Rice, 2017).

This review aims to provide updated knowledge on FPL, including its etiology, pathogenesis, clinical and pathological features, diagnostic imaging, hematological and clinicopathological findings, treatment approaches, preventive strategies and aspects of infection and immunity.

Etiology

Feline panleukopenia (FPL) is caused by infection with Carnivore protoparvovirus 1, a virus classified within the subfamily Parvovirinae and the genus Protoparvovirus. This pathogen is a small, non-enveloped, linear, single-stranded DNA virus possessing a genome of approximately 5.1 kilobases. The genome contains two major genes: the nonstructural (NS) gene and the structural gene. The NS gene encodes the NS1 and NS2 proteins, which play essential roles in intracellular transport, viral DNA replication, and capsid assembly. The structural gene encodes the VP1 and VP2 proteins that constitute the viral capsid. The capsid is composed of 60 protein subunits, approximately 10% of which are VP1 and 90% VP2 (Barrs, 2019).

Only six amino acid residues within the VP2 region distinguish the newly identified canine parvovirus (CPV) from the feline panleukopenia virus (FPV). These specific residues are essential for the virus's ability to bind to the canine transferrin receptor (TfR) and enable cellular entry via clathrin-mediated endocytosis. Consequently, although CPV-2 can replicate in feline cell cultures under in vitro conditions, it is unable to replicate effectively in feline tissues in vivo.

Incidence of feline panleukopenia in domestic cats

FPL continues to affect both household and shelter cats globally. PCR testing detected FPV in 77.8% of feline samples in Kerala, India (Raheena *et al*. 2017). In Mumbai, 70% of household cats tested positive for FPV (Kadam 2022). ELISA testing showed a 29.1% seroprevalence of FPV antibodies in unvaccinated healthy cats (Bakde 2019). Screening in Bangladesh revealed a 40.45% infection rate among household cats (Hossen *et al*., 2024). PCR identified FPV in 22.9% of suspected cases, with a 10.6% mortality rate and 45.9% case fatality rate (Kabir *et al.* 2023). Researchers in Germany found that 48.7% of shelter cats were shedding FPV, with young age, lack of vaccination and

group housing as major risk factors (Rehme *et al.* 2022). FPV accounted for 95% of FPL cases, while CPV variants including CPV 2a, CPV 2b and CPV 2c made up the remaining 5% (Barrs, 2019). Clinical outcomes varied depending on host immunity, co-infections and viral strain virulence (Uyurca and Haydardedeoglu 2025).

Age-wise incidence of feline panleukopenia in cats

The vulnerability to infection is further heightened in young animals due to the absence of a fully developed active immune response, combined with the presence of rapidly dividing cells, which creates a favourable environment for viral replication (Larry and Francis, 2011; Bakde, 2019). This contributes to a notably higher incidence of feline panleukopenia in kittens, particularly those under six months of age.

A critical period known as the "immunological gap" occurs between the decline of passive maternal immunity and the onset of active immunity induced by vaccination. During this window, kittens are especially susceptible to infection by feline panleukopenia virus (FPV) (Zenad and Radhy, 2020). The highest incidence of the disease has consistently been observed in kittens aged between 0 and 6 months (Kadam, 2022). In a prevalence study, kittens within this age group exhibited an infection rate of 22.9%, accompanied by a mortality rate of 16.4% (Kabir *et al.*, 2023). Similarly, findings from another study indicated that 56% of all feline panleukopenia cases occurred in cats aged 0–6 months, reinforcing the age-related susceptibility to the disease (Sawale *et al*., 2024).

Incidence of feline panleukopenia in cats with varying immunization status

The probability of an unvaccinated cat developing immunity to FPV through natural exposure increases with age, due to cumulative environmental contact with the field virus (DiGangi *et al*., 2012). FPL is most commonly observed in kittens that are either unvaccinated or have received incomplete vaccination schedules. This heightened susceptibility is primarily attributed to the decline of maternally derived antibodies (MDAs) and the presence of an "immunity gap" which is a critical window during which MDAs remain high enough to interfere with vaccine efficacy but too low to provide protection against actual infection (Day *et al*., 2016).

Unvaccinated or inadequately immunized cats of any age are significantly more prone to contracting feline panleukopenia. In a recent study, unvaccinated individuals were found to have an 8.83-fold increased risk of developing the disease, underscoring the importance of complete and timely vaccination (Kabir *et al*., 2023).

Breed-wise incidence of feline panleukopenia in cats

A higher incidence of FPL has been reported among non-descript breeds, stray cats, individuals from multi-cat households, and those lacking a history of deworming

(Bakde, 2019). These factors may contribute to increased environmental exposure and reduced baseline immunity in these populations.

Conversely, some studies have indicated that breed may not play a significant role in susceptibility. For instance, one investigation reported that all cat breeds appear equally vulnerable to FPL, with no evidence of breed-specific resistance (Pandey, 2022).

However, findings from a recent study contradict this assertion, indicating that non-descript cats exhibited the highest prevalence, representing 85.72% of all documented FPL cases (Sawale *et al*., 2024).

Gender-wise incidence of feline panleukopenia in cats

The incidence of FPL has been reported to be higher in male cats, with several studies consistently noting that male individuals outnumber females among clinical admissions for the disease (Kruse *et al*., 2010). This trend may be influenced by behavioral factors, increased roaming or greater environmental exposure among males.

Contrary to these findings, a study recorded a greater frequency of FPL in female cats, indicating that sex-based susceptibility may vary across regions or populations (Bakde, 2019). In a more recent investigation involving 131 feline cases in Bangladesh, the highest prevalence was observed in young, unvaccinated male cats, highlighting age, sex, and immunization status as critical determinants of disease susceptibility (Hossen *et al*., 2024).

In contrast, another recent study reported that 65.71% of the affected cats were female, suggesting variability in gender-related prevalence and the potential influence of local epidemiological and management factors (Sawale *et al*., 2024).

Influence of type of housing on the incidence of feline panleukopenia

The FPV is highly resilient, capable of withstanding harsh environmental conditions and many commonly used disinfectants. This environmental stability allows the virus to persist for extended durations in shared or insufficiently sanitized spaces, thereby elevating the risk of transmission, particularly in densely populated settings (Greene, 2012).

Cats housed individually such as those in private residences with controlled environments—are at significantly lower risk of contracting FPV. This reduced susceptibility is largely attributed to limited contact with other animals and the implementation of higher standards of hygiene and preventive care (Scherk *et al.*, 2013).

Overcrowded shelter environments have been shown to negatively impact feline immune function due to chronic stress, which in turn heightens their vulnerability to infections such as FPV. This association between environmental stress and increased disease susceptibility explains the higher

incidence of panleukopenia in communal housing conditions (Litster and Benjanirut, 2014).

The implementation of strict quarantine measures for newly admitted or clinically ill cats has proven to be effective in minimizing the spread of FPV in shelters and catteries, underscoring the importance of robust biosecurity protocols (Stuetzer and Hartmann, 2014).

Outbreaks of feline panleukopenia are most frequently reported in group housing systems, including animal shelters and rescue facilities. Contributing factors include high population density, suboptimal vaccination coverage, and frequent movement or mixing of animals, all of which facilitate rapid viral transmission in these environments (Barrs, 2019).

Seasonal incidence of feline panleukopenia in domestic cgfats

The incidence of FPL has been observed to exhibit seasonal variation, with increased case numbers reported during specific times of the year. A notable rise in FPL cases is typically seen during the summer and early autumn months, a period that coincides with the influx of young, immunologically naive kittens into shelters. These kittens often experience a decline in MDA levels, rendering them highly susceptible to infection by the FPV (Barrs, 2019).

Similarly, a higher frequency of FPL cases has been reported during the warmer months, with environmental conditions such as elevated temperature and humidity potentially enhancing viral stability and facilitating transmission (Kadam, 2022).

Further supporting the seasonal nature of the disease, a study in Bangladesh documented an increase in FPL cases during the monsoon and post-monsoon periods. These climatic conditions may create favorable environments for virus survival and dissemination, particularly in overcrowded or poorly sanitized settings (Kabir *et al*., 2023).

Host Range

The principal hosts of FPV include domestic cats (*Felis catus*) and a variety of wild felids, such as tigers, leopards, and lions. These species are highly susceptible to infection and can develop severe clinical disease, particularly in the absence of prior immunization (Greene, 2012). Importantly, FPV is not considered zoonotic, as it does not infect humans (Decaro and Buonavoglia, 2012).

The host range of FPV extends beyond domestic felines to include certain wild members of the suborder Feliformia and some wild canids, such as raccoons and foxes. Despite occasional infections in these species, domestic dogs are not considered part of the virus's natural host spectrum (Allison *et al*., 2014). This host restriction is further reinforced by the virus's high specificity for feliform species, including both domestic and wild felids (Allison *et al.,* 2014).

Pathogenesis

FPV targets rapidly dividing cells, exploiting host-cell DNA polymerase to replicate within the nucleus during the S phase of the cell cycle (Berns, 1990; Deleu *et al*., 1999). Its tropism includes the bone marrow, lymphoid tissues, intestinal crypts, and developing neuroblasts, leading to immunosuppression, enteritis, and, in perinatal infections, cerebellar hypoplasia (Poncelet *et al.*, 2013; Uyurca and Haydardedeoglu, 2025).

Although FPV cannot efficiently bind the TfR, limiting systemic infection in dogs, it can replicate in certain canine lymphoid tissues (Parrish, 1999). In cats, replication begins in oropharyngeal lymphoid tissue within 18–24 hours post-infection, with viremia detectable by day 2–7. Infected animals shed the virus in secretions such as saliva, urine, vomit, and fecesoften even before clinical signs appear (Barrs, 2019).

Transmission primarily occurs via the fecal–oral route, but indirect transmission through contaminated fomites and in utero infection is also significant. Infections during early gestation can result in fetal death or malformations, while late gestation exposure may cause cerebellar hypoplasia (Davis-Wurzler, 2014). Congenital infections may also produce ocular anomalies like retinal or optic nerve dysplasia (Stiles, 2021).

Despite only shedding virus for 1–2 days, infected cats contribute to environmental contamination, as FPV remains viable for extended periods on surfaces (Greene, 2012).

FPV shows high specificity for feline and feliform hosts, whereas CPV, a derivative of FPV, led to a global canine pandemic in the 1970s (Allison and Parrish, 2014). Experimental infections with CPV variants demonstrated cross-neutralization, although immune responses were variable, indicating antigenic similarities but differing immunogenicity (Nakamura *et al*., 2007). Neonatal or transplacental infections can manifest as fading kitten syndrome or central nervous system disorders (Evermann and Kennedy, 2011).

Clinical Signs

Vomiting is one of the most common clinical manifestations, frequently observed in both experimental and natural cases of FPV (Litster and Benjanirut, 2014; Raj and Haryanto, 2020; Pfankuche *et al*., 2017; Awad *et al*., 2018). Fever is also a predominant early symptom, reported in the majority of infected cats (Litster and Benjanirut, 2014; Awad *et al*., 2018; Raj and Haryanto, 2020). As the infection progresses, body temperature may decline, resulting in hypothermia, particularly during the terminal stages (Riya *et al*., 2020).

Diarrhea, often hemorrhagic, is a hallmark sign and a major cause of mortality in kittens (Stuetzer and Hartmann, 2014). FPV-induced enteritis disrupts intestinal crypt cell turnover, leading to villous atrophy, malabsorption, and increased permeability, which collectively contribute

to severe fluid loss and bloody diarrhea (Pfankuche *et al*., 2017; Miller *et al*., 2021). These gastrointestinal losses result in varying degrees of dehydration and poor skin condition (Mahendra *et al*., 2020).

Additional clinical features include stomatitis, oral ulcers, hypersalivation, halitosis, conjunctivitis, ocular lesions, corneal opacity, lacrimation, and in some cases, blindness (Sykes, 2020; Ramadhani *et al*., 2024). Neurological signs such as ataxia may occur in neonates due to cerebellar involvement, while pregnant queens may experience abortion, fetal resorption, or stillbirth (Sykes, 2020; Tuzio, 2021).

The disease is classified into four clinical forms—peracute, acute, subacute, and subclinical—depending on the progression and severity of symptoms (Tuzio, 2021). In overcrowded conditions, more severe clinical signs such as emaciation, mucous or bloody diarrhea, depression, oral erosions, congested mucous membranes, and weakness are frequently observed (Al-Autaish *et al*., 2024).

FPV pathogenesis also includes high fever (>39.5°C), abdominal pain, hypersalivation, epistaxis, otitis, and in severe systemic involvement, shock and death (Ramadhani *et al.*, 2024).

Hemato-biochemical variation in Feline panleukopenia-affected Cats

Hematological Parameters

Feline panleukopenia commonly causes leukopenia, thrombocytopenia, and anemia due to viral replication in rapidly dividing hematopoietic cells (Kruse *et al.,* 2010; Greene, 2012; Barrs, 2019). Microcytic hypochromic anemia has been associated with disrupted iron metabolism and inflammation. Anemia may also result from gastrointestinal bleeding, shortened erythrocyte lifespan, and poor iron absorption (Barrs, 2019). Infected cats often show significant reductions in total leukocytes, lymphocytes, monocytes, and platelets (Al-Autaish *et al.*, 2024).

Serum Biochemical Parameters

Biochemically, affected cats may present with hypoproteinemia and elevated AST, BUN, and ALP, indicating hepatic and renal involvement (Al-Autaish *et al*., 2024). Hyperbilirubinemia and icterus are linked to systemic inflammation or sepsis, with prognosis depending on the timeliness of the immune response.

Diagnosis

Immunochromatography using fecal samples

Although rapid tests show high specificity, their sensitivity can be below 50% (Kantere *et al.*, 2015). PCR offers better sensitivity and can detect prolonged fecal and urinary shedding (Barrs, 2019). In a field study, PCR detected FPV in 70% of cases, outperforming the SNAP test at 58% (Sawale *et al.*, 2024).

Due to antigenic similarity between FPV and CPV-2, ELISA and rapid tests developed for dogs are often used to detect

FPV in cats (Abd-Eldaim *et al*., 2009). Test sensitivity ranges from 50% to 80%, while specificity remains high (94%–100%) (Neuerer *et al.*, 2008).

However, false negatives may occur due to low viral shedding or maternal antibodies, and modified live vaccines can cause false positives for up to 14 days (Meason-Smith *et al.*, 2017).

Polymerase Chain Reaction using fecal samples

Polymerase chain reaction (PCR) is considered a sensitive and reliable method for detecting FPV DNA in fecal samples, especially when antigen tests yield false negatives (Freisl *et al.*, 2017; Sykes, 2014).

Although PCR cannot always differentiate FPV from CPV due to shared genomic sequences, it remains more sensitive than antigen-based tests, particularly for early or low-level infections (Walter-Weingartner *et al*., 2021; Barrs, 2019). However, vaccine-derived viral DNA may occasionally yield false positives (Freisl *et al*., 2017).

PCR demonstrated superior diagnostic performance by detecting FPV in 21 out of 27 clinically suspected feline cases, compared to 10 positive results by immunochromatography and 8 by hemagglutination, both of which exhibited high specificity but limited sensitivity. The diagnostic accuracy and sensitivity of PCR have also been supported by subsequent studies, further validating its reliability over other methods (Raheena *et al*., 2017; Zenad and Radhy, 2020).

Diagnostic workflows often involve DNA electrophoresis using agarose gel and UV visualization to confirm viral DNA bands (Ramadhani *et al*., 2024). Though other techniques like ELISA, immunofluorescence, and virus isolation are still used, PCR remains the preferred confirmatory method (Nofira *et al*., 2022).

Therapeutic Approach

Aggressive fluid therapy is essential in severe cases with dehydration, hypovolemia, and shock. Crystalloids, colloids, plasma or blood transfusions are recommended, particularly in hypoalbuminemia or anemia (Lloret, 2016; Addie *et al*., 2009; Truyen *et al*., 2009; Greene and Decaro, 2012). Young kittens require glucose monitoring due to hypoglycemia risk (Barrs, 2019).

Parenteral antacids (e.g., esomeprazole, famotidine) are advised in cases of vomiting or hematemesis (Stuetzer and Hartmann, 2014). Supportive feeding with Hill's A/D and Karo syrup can prevent hepatic lipidosis without triggering vomiting (Rice, 2017).

Maropitant is the preferred antiemetic, with ondansetron as adjunct therapy. Mirtazapine should be used cautiously due to serotonin syndrome risk and uncertain safety in kittens (Porporato *et al*., 2018; Stuetzer and Hartmann, 2014).

Broad-spectrum antibiotics, especially cephalosporins, are used to prevent sepsis in immunosuppressed cats. Drug selection should consider renal function and resistance patterns (Greene and Decaro, 2012; Stuetzer and Hartmann, 2014; Barrs, 2019).

Fenbendazole reduces Giardia burden, potentially improving gut health and FPV outcomes (Keith *et al*., 2003). Deworming prior to vaccination is recommended to enhance immune response, though mild parasitism may not impact vaccine efficacy (Gruffydd-Jones *et al*., 2013; Natukunda *et al*., 2022; Weidinger *et al*., 2024).

Filgrastim (rhG-CSF) promotes leukocyte recovery in FPL but shows variable efficacy due to FPV-induced marrow suppression (Rice, 2017). It may interfere with platelet production and cause anemia or thrombocytopenia (Li *et al*., 2019). However, clinical trials report improved survival and reversal of leukopenia with early administration (Dascalu *et al*., 2024). Recommended dosage is 1–5 µg/kg SC once or twice daily (Plumb, 2025). Caution is advised based on immune status and disease progression (Uyurca and Haydardedeoglu, 2025).

Glycyrrhizinic acid containing products like Viusid have shown immunomodulatory and antioxidant effects by reducing pro-inflammatory cytokines and oxidative stress (Eroglu and Erdogan, 2023). In animal models, Viusid improved survival, immune response, and reduced morbidity (Ardana *et al.*, 2021). Elevated PTX-3 in FPV-infected cats indicates oxidative damage, supporting the role of antioxidants in supportive therapy (Eroglu and Erdogan, 2023).

Complications of Feline panleukopenia

FPV targets mitotically active tissues such as the intestinal crypts, lymphoid organs, and bone marrow, but infection during late gestation or perinatally can lead to cerebellar hypoplasia in kittens due to viral replication in the external germinal epithelium of the cerebellum. Productive infection of Purkinje cells in neonates may result from re-entry into the cell cycle (Poncelet *et al*., 2013). FPV replication remains restricted to actively dividing S-phase cells, especially in kittens under 10 days of age (Barrs, 2019).

Prognosis

Hypoalbuminemia, hypokalemia, and leukopenia are also linked to poor outcomes (Kruse, 2010). Survival rates are significantly influenced by access to intensive care; up to 80% of kittens receiving critical care survive, compared to similar mortality in untreated cases.

Control

Vaccination is highly effective in preventing FPV, inducing long-lasting sterilizing immunity when properly administered to naive cats. However, maternal antibodies can interfere with vaccine efficacy until 12–16 weeks of age, necessitating a final dose after this period

(Jakel *et al.*, 2012). Core vaccination protocols recommend administration at 8, 12, and 16–20 weeks, followed by a booster at 26–52 weeks and revaccination every three years (Day *et al*., 2016). In high-risk settings, inactivated vaccines may be initiated as early as 4 weeks and repeated every 3–4 weeks until 16 weeks (Day *et al*., 2016). The FVRCP trivalent vaccine is essential for all domestic cats and typically starts at 6–8 weeks (Truyen *et al*., 2009).

CONCLUSION

Feline panleukopenia remains a highly contagious and life-threatening disease, especially in unvaccinated cats and kittens. Although molecular diagnostics have improved rapid detection, treatment still relies mainly on supportive care, with immunomodulators offering emerging benefits. Strict hygiene, isolation and timely vaccination remain the most effective tools for prevention and control. Continued research and strong vaccination practices are essential to reduce the disease burden and improve survival outcomes.

REFERENCES

Abd-Eldaim, M.M., Wilkes, R.P., Thomas, K.V. and Kennedy, M.A. (2009). Development and validation of a TaqMan real-time PCR assay for detection of feline panleukopenia virus. *Journal of Veterinary Diagnostic Investigation,* **21**(2) : 202–208.

Addie, D.D., Toth, S., Murray, G.D. and Jarrett, O. (2009). Risk of feline infectious peritonitis in cats naturally infected with feline coronavirus. *Journal of Feline Medicine and Surgery,* **11**(4) : 219–227.

Allison, A.B. and Parrish, C.R. (2014). Parvoviruses of carnivores: Their transmission and the emergence of new variants. *Journal of Infection, Genetics and Evolution*, **21**: 1–11.

Allison, A.B., Kohler, D.J., Fox, K.A., Brown, J.D., Gerhold, R.W., Shearn-Bochsler, V.I., Dubovi, E.J., Parrish, C.R. and Holmes, E.C. (2014). Frequent cross-species transmission of parvoviruses among diverse carnivore hosts. *Journal of Virology*, **88**(9) : 4584–4597.

Al-Autaish, H.H., Al-Fattli, H.H. and Al-Saffar, T.M. (2024). Clinical, hematological and biochemical alterations in cats naturally infected with feline panleukopenia virus. *Veterinary World,* **17**(1) : 88–95.

Ardana, I.B.K., Arimbawa, I.M., and Sumiarto, B. (2021). Immunomodulatory effect of glycyrrhizinic acid in viral infections: an experimental study. *Veterinary Medicine International,* **2021 :** 1–8.

Awad, R.A., Khalil, W.K.B. and Attallah, A.G. (2018). Feline panleukopenia viral infection in cats: application of some molecular methods used for its diagnosis. *Journal of Genetic Engineering and Biotechnology*, **16**(2): 491–497.

Bakde, R.A. (2019). Clinico-epidemiological studies and molecular diagnosis of feline panleukopenia in cats. MVSc thesis, Kerala Veterinary and Animal Sciences University, Pookode, Kerala.

Barrs, V.R. (2019). Feline panleukopenia: a re-emergent disease. *Veterinary Clinics of North America: Small Animal Practice,* **49**(4): 651–670.

Berns, K.I. (1990). Parvovirus replication. *Microbiology Reviews*, **54**(3) : 316–329.

Cotmore, S.F., Agbandje-McKenna, M., Chiorini, J.A., Mukha, D.V., Pintel, D.J., Qiu, J., Soderlund-Venermo, M., Tattersall, P., Tijssen, P., Gatherer, D. and Davison, A.J. (2019). The family *Parvoviridae. Archives of Virology,* **164 :** 1239–1247.

Dascalu, L., Popescu, R., Militaru, M. and Niculae, M. (2024). Effect of recombinant human G-CSF in feline panleukopenia-induced leukopenia. *Animals*, **14**(2) : 221.

Davis-Wurzler, G.M. (2014). Feline panleukopenia virus. *Veterinary Clinics of North America: Small Animal Practice*, **44**(1): 53-63.

Day, M.J., Horzinek, M.C., Schultz, R.D. and Squires, R.A. (2016). WSAVA guidelines for the vaccination of dogs and cats. *Journal of Small Animal Practice,.,* **57**(1) : E1–E45.

Decaro, N. and Buonavoglia, C. (2012). Canine parvovirus - a review of epidemiological and diagnostic aspects, with emphasis on type 2c. *Veterinary Microbiology*, **155**(1) : 1–12.

Deleu, L., Pujol, A., Nuesch, J.P.F. and Rommelaere, J. (1999). Inhibition of cell cycle progression by the NS1 protein of parvovirus. *Journal of Virology*, **73**(6) : 4995–500).

DiGangi, B.A., Levy, J.K., Griffin, B., Reese, M.J. and Dingman, P.A. (2012). Prevalence of protective antibody titers for feline panleukopenia virus. *Journal of the American Veterinary Medical Association,* **241**(1) : 61–65.

Evermann, J.F. and Kennedy, M.A. (2011). Viral diseases of the feline nervous system. *Veterinary Clinics of North America: Small Animal Practice*, **41**(1) : 123–144.

Eroglu, A. and Erdogan, H.M. (2023). Pentraxin-3 and oxidative stress markers in cats infected with feline panleukopenia virus. *Veterinary Medicine and Sciences,* **9**(3) : 1130–1138.

Freisl, M., Truyen, U., Müller, A. and Speck, S. (2017). Evaluation of a real-time PCR assay for detection of feline panleukopenia virus. J*ournal of Virological Methods*, **244** : 66–72.

Greene, C.E. and Decaro, N. (2012). Feline panleukopenia. In: Greene, C.E. (ed.) *Infectious diseases of the dog and cat*. 4th ed. Missouri: Elsevier Saunders, 67–80.

Gruffydd-Jones, T.J., Addie, D., Belák, S., Boucraut-Baralon, C., Egberink, H., Frymus, T. et al. (2013). ABCD guidelines on feline panleukopenia. *Journal of Feline Medicine and Surgery*, **15**(7) : 546–558.

Hossen, M.L., Kabir, A., Rahman, M.S. and Islam, M.A. (2024). Molecular detection and epidemiology of feline panleukopenia virus in Bangladesh. *Veterinary World*, **17**(2) : 310–318.

Jakel, V., Cussler, K., Hanschmann, K.M., Truyen, U. and König, M. (2012). Influence of maternally derived antibodies on vaccination against feline panleukopenia virus. *Veterinary Microbiology*, **154**(1–2) : 61–68.

Kantere, M.C., Athanasiou, L.V., Spyrou, V., Billinis, C. and Kyriakis, C.S. (2015). Diagnostic performance of rapid tests for feline panleukopenia virus. *Journal of the Hellenic Veterinary Medical Society,* **66**(1) : 1–8.

Kadam, S.R. (2022). Molecular epidemiology of feline panleukopenia virus in domestic cats. MVSc thesis, Maharashtra Animal and Fishery Sciences University, Nagpur, India.

Kabir, A., Rahman, M.S., Hossen, M.L. and Islam, M.A. (2023). Clinicopathological findings and risk factors of feline panleukopenia in Bangladesh. *Veterinary Medicine and Science,* **9**(4) : 1780–1790.

Keith, C.L., Radecki, S.V. and Lappin, M.R. (2003). Evaluation of fenbendazole for treatment of Giardia infection in cats. *Journal of the American Veterinary Medical Association* **222**(11) : 1548–1551.

Kruse, B.D., Unterer, S., Horlacher, K., Sauter-Louis, C. and Hartmann, K. (2010). Prognostic factors in cats with feline panleukopenia. *Journal of Veterinary Internal Medicine*, **24**(6) : 1271–1276.

Larry, J.T. and Francis, J.D. (2011). Immune system development in kittens. *Veterinary Clinics of North America: Small Animal Practice.,* **41**(2) : 341–354.

Li, R., Zhang, Y., Zhou, J. and Wu, J. (2019). Adverse hematological effects of recombinant G-CSF therapy. *Veterinary Immunology and Immunopathology,* **214** : 109889.

Litster, A. and Benjanirut, C. (2014). Risk factors for feline panleukopenia virus infection in shelters. *Journal of Feline Medicine and Surgery*, **16**(12) : 1034–1041.

Mahendra, P., Aarthi, K.S. and Kumanan, K. (2020). Clinical and pathological studies on feline panleukopenia. *Indian Journal of Veterinary Pathology,* **44**(1) : 45–49.

Meason-Smith, C., Levy, J.K., Crawford, P.C. and Slater, M.R. (2017). False-positive results on feline panleukopenia virus antigen tests. *Journal of Veterinary Diagnostic Investigation,* **29**(5) : 678–681.

Mietzsch, M., Penzes, J.J., Agbandje-McKenna, M. and Parrish, C.R. (2019). Structural biology of parvoviruses. *Viruses*, **11**(6) : 533.

Miller, S.A., Pesavento, P.A. and Murphy, B.G. (2021). Pathology of feline panleukopenia virus infection. *Veterinary Pathology,* **58**(2) : 319–329.

Nakamura, K., Sakamoto, M., Ikeda, Y., Sato, E., Kawakami, K., Miyazawa, T., Tohya, Y., Takahashi, E., Mikami, T. and Mochizuki, M. (2007). Pathogenic potential of canine parvovirus types 2a and 2c in cats. *Clinical and Diagnostic Laboratory Immunology,* **14**(4) : 422–430.

Natukunda, A., Mwiine, F.N. and Atuhaire, D.K. (2022). Effect of parasitism on vaccine response in cats. *Veterinary Parasitology*, **303** : 109671.

Neuerer, F.F., Horlacher, K., Truyen, U. and Hartmann, K. (2008). Comparison of different in-clinic tests for the detection of feline panleukopenia virus. *Journal of Feline Medicine and Surgery,* **10**(5) : 448–455.

Nofira, A., Ramadhani, R., Putra, A.A.G. and Setiawan, B. (2022). Molecular detection of feline panleukopenia virus in cats using PCR. *Veterinary World,* **15**(9) : 2213–2218.

Pandey, R. (2022). Epidemiological studies on feline panleukopenia in domestic cats. *Indian Veterinary Journal*, **99**(3) : 25–29.

Parrish, C.R. (1999). Host range relationships and the evolution of canine parvovirus. *Veterinary Microbiology,* **69**(1–2) : 29–40.

Pfankuche, V.M., Kühn, J., Deutsch, M.J., Gerhauser, I., Baumgärtner, W. and Puff, C. (2017). Intestinal pathology in feline panleukopenia virus infection. *Veterinary Pathology,* **54**(4) : 543–554.

Plumb, D.C. (2025). Plumb's veterinary drug handbook. 10th ed. Stockholm, WI: PharmaVet Inc.

Poncelet, L., Coppens, A. and Peeters, D. (2013). Cerebellar hypoplasia associated with feline panleukopenia virus infection. *Journal of Comparative Pathology,* **148**(2–3) : 182–186.

Porporato, F., Riondato, F., Cappello, S. and Valle, E. (2018). Safety of mirtazapine in cats. *Journal of Feline Medicine and Surgery*, **20**(6) : 517–524.

Raj, K. and Haryanto, A. (2020). Clinical signs and molecular diagnosis of feline panleukopenia virus. *Journal of Advanced Veterinary and Animal Research,* **7**(3) : 512–518.

Ramadhani, R., Nofira, A., Putra, A.A.G. and Setiawan, B. (2024). Molecular detection and clinicopathological changes of feline panleukopenia virus. *Veterinary World*, **17**(1) : 145–152.

Rehme, C., Truyen, U. and Hartmann, K. (2022). Feline panleukopenia virus infections in shelters: Risk factors and shedding patterns. *Viruses*, **14**(4) : 755.

Rice, D. (2017). Supportive therapy in feline panleukopenia. *Veterinary Nurse*, **8**(9) : 496–502.

Riya, R., Mini, M. and Pillai, U.N. (2020). Clinical and hematological changes in cats affected with feline panleukopenia. *International Journal of Current Microbiology and Applied Sciences*, **9**(8) : 2417–2424.

Sawale, G.K., Kadam, S.R., Bakde, R.A. and Kulkarni, M.B. (2024). Epidemiological and diagnostic evaluation of feline panleukopenia virus infection in cats. *Indian Journal of Veterinary Medicine, **44**(1) :15–22.*

Scherk, M.A., Ford, R.B., Gaskell, R.M., Hartmann, K., Hurley, K.F., Lappin, M.R. et al. (2013). 2013 AAFP feline vaccination advisory panel report. *Journal of Feline Medicine and Surgery*, **15**(9) : 785–808.

Stiles, J. (2021). Feline ocular manifestations of systemic viral diseases. *Veterinary Clinics of North America: Small Animal Practice,* **51**(2) : 373–389.

Stuetzer, B. and Hartmann, K. (2014). Feline parvovirus infection and associated diseases. *Veterinary Journal*, **201**(2): 150–155.

Sykes, J.E. (2014). Canine and feline parvovirus infections. In: Sykes, J.E. (ed.) Canine and feline infectious diseases. Missouri: Elsevier Saunders, 141–151.

Sykes, J.E. (2020). Feline viral diseases. *Veterinary Clinics of North America: Small Animal Practice,* **50**(5) : 1115–1136.

Truyen, U., Addie, D., Belák, S., Boucraut-Baralon, C., Egberink, H., Frymus, T. Gruffydd-Jones, T., Hartmann, K., Hosie, M.J., Lloret, A., Lutz, H., Marsilio, F., Grazia Pennisi, M., Radford, A.D., Thiry, E. and Horzinek, M.C. (2009). Feline panleukopenia. ABCD guidelines on prevention and management. *Journal of Feline Medicine and Surgery*, **11**(7) : 538–546.

Tuzio, H. (2021). Feline panleukopenia virus infection: Clinical update. *Veterinary Clinics of North America: Small Animal Practice*, **51**(2) : 403–415.

Uyurca, N. and Haydardedeoglu, A.E. (2025). Advances in treatment and immunomodulation in feline panleukopenia virus infection. *Veterinary Medicine and Science*, **11**(1) : 95–108.

Walter-Weingartner, M., Meili, T., Truyen, U. and Hartmann, K. (2021). Diagnostic performance of PCR assays for feline panleukopenia virus. *Journal of Veterinary Diagnostic Investigation*, **33**(3) : 567–573.

Weidinger, P., Speck, S. and Truyen, U. (2024). Parasite burden and vaccine responsiveness in cats. *Veterinary Parasitology,* **317 :** 109856.

Zenad, M.M. and Radhy, A.M. (2020). Molecular diagnosis and epidemiology of feline panleukopenia virus. *Plant Archives*, **20**(1) : 1565–1570.