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Review Article

Epidemiology, Pathogenesis, and Control of Feline Calicivirus: Current Insights and Future Directions

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Feline calicivirus (FCV) is a globally distributed pathogen and a leading cause of upper respiratory tract disease in cats, responsible for recurrent outbreaks in shelters and multi-cat environments. Its non-enveloped, positive-sense RNA genome, replicated by an error-prone RNA-dependent RNA polymerase, results in high genetic variability and the emergence of immune escape variants. FCV infection presents with diverse clinical manifestations, from mild oral–respiratory disease and limping syndrome to the severe virulent systemic form (VS-FCV). Despite extensive research, no licensed antiviral agent with proven efficacy is available; treatment remains supportive and focused on symptom relief and prevention of secondary infections. Experimental compounds such as nitazoxanide, 2′-C-methylcytidine, and recombinant feline interferon-omega show in vitro antiviral potential but require clinical validation. Vaccination remains the main preventive measure, although protection is strain-dependent and incomplete. The virus's environmental resilience necessitates strict biosecurity measures and effective disinfection using chlorine-based or oxidizing agents. Integrated strategies combining improved vaccine design, antiviral development, and environmental control are essential to achieve sustainable management of FCV and reduce its impact on feline health and welfare.

Keywords: Feline calicivirus, RNA virus, genetic diversity, vaccination

INTRODUCTION

Feline calicivirus is a major etiological agent of upper respiratory tract disease in domestic cats, with a global distribution and substantial impact on feline health and welfare (Spiri et al., 2022). The effects of FCV extend beyond individual cases, playing a central role in recurrent outbreaks in high-density environments such as shelters, catteries, and veterinary clinics (Zhang et al., 2025). These settings, characterized by high population density, rapid animal turnover, and a high prevalence of carrier cats, facilitate rapid viral transmission and frequent re-introduction events (Stuntebeck et al., 2024). Longitudinal studies in shelters have demonstrated that transmission readily occurs through direct contact and respiratory secretions, while the virus's persistence on fomites prolongs its infectivity and increases the risk of biosecurity breaches (Hofmann-Lehmann et al., 2022).

Furthermore, the simultaneous circulation of multiple strains and frequent reinfection within the same host accelerate genetic diversification through mutation and recombination, increasing the likelihood of immune escape variants and recurrent outbreaks (Coyne et al., 2012). The combination of these factors makes FCV control in shelters considerably more complex than in stable household populations and necessitates specialized management protocols that include triage and isolation procedures, effective cleaning and disinfection, as well as vaccination and carrier monitoring strategies (Di Profio et al., 2025).

The unique biological characteristics of FCV, particularly the presence of an error-prone RNA-dependent RNA polymerase, contribute to its high mutation rate and broad antigenic variability (Zhang et al., 2025). This phenomenon facilitates the emergence of genetically diverse strains with varying pathogenic potential and presents major challenges to diagnostic accuracy, therapeutic consistency, and long-term vaccine efficacy (Di Profio et al., 2025).

Therapeutic and preventive options for FCV remain limited. To date, no licensed antiviral agents with proven efficacy are available, and existing treatments are largely supportive, focusing on symptomatic relief and management of secondary infections (Fumian et al., 2018). Host immune variability, coupled with the practical difficulties of implementing stringent biosecurity measures in multi-cat households, shelters, and breeding facilities, further complicates disease control (Wang and Lin, 2024). These limitations underscore an urgent need for integrated strategies encompassing the development of effective antiviral compounds, refinement of vaccination protocols to enhance cross-protection, deployment of rapid and sensitive diagnostic tools, and rigorous environmental management practices to reduce viral transmission.

This review aims to provide an updated synthesis of the molecular virology, immunopathogenesis, epidemiology, and clinical spectrum of FCV, alongside current and emerging preventive and therapeutic strategies. By presenting a comprehensive and mechanistically informed perspective, this article seeks to guide practitioners and researchers in designing more effective interventions to mitigate both the clinical burden and the epidemiological spread of FCV.

MOLECULAR BIOLOGY AND GENETIC DIVERSITY OF FELINE CALICIVIRUS

Feline calicivirus is recognized as one of the most important feline pathogens and a major

concern in veterinary medicine. Taxonomically, the virus belongs to the family *Caliciviridae*, order *Picornavirales*, and genus *Vesivirus* (Wei et al., 2024). Morphologically, FCV is a non-enveloped virus with icosahedral symmetry, measuring approximately 27–40 nm in diameter, and possesses a positive-sense, single-stranded RNA genome of about 7.7 kb (Cisternas, 2020; Zhang et al., 2025). The genome comprises three open reading frames (ORFs) that encode structural and non-structural proteins, including the RNA-dependent RNA polymerase (RdRp), the major capsid protein VP1, and the minor capsid protein VP2 (Li et al., 2024). At the 5′ end, the genome is covalently linked to a viral genome-linked protein (VpG) that serves as a replication primer, while the poly-A tail at the 3′ end enhances genome stability and translational efficiency (Smertina et al., 2021; Asif et al., 2025).

Among these proteins, VP1 is the principal structural and immunogenic component, playing a pivotal role in viral stability and infectivity (Asif et al., 2025). High-resolution structural analyses have shown that VP1 contains distinct functional domains that specifically interact with feline junctional adhesion molecule A (fJAM-A)—a receptor predominantly expressed on epithelial and endothelial cells—thereby mediating viral attachment, entry, and tissue tropism (Lu et al., 2018). Beyond its structural function, VP1 serves as a major determinant of FCV's antigenic variability (Cubillos-Zapata et al., 2020). Mutations or alterations within the hypervariable regions of this protein enable the virus to evade host immune recognition (immune escape), facilitating the emergence of novel strains with differing pathogenic potential (Wei et al., 2024). This feature complicates the development of broadly protective vaccines but simultaneously positions VP1 as a critical target for the design of cross-protective immunogens and antiviral inhibitors (Heng et al., 2025).

Studies have demonstrated that FCV evolves at an exceptionally high mutation rate, estimated at 10^{-2} to 10^{-3} substitutions per site per year—substantially higher than that of many other veterinary RNA viruses (Wei et al., 2024). Consequently, FCV may exist as a quasispecies within the host, facilitating rapid adaptation to immune and environmental pressures (Wei et al., 2024). This genetic plasticity is further enhanced by co-infection, recurrent reinfection, and recombination events occurring when multiple strains circulate simultaneously within a population (Hofmann-Lehmann, 2022). Under co-infection or sequential reinfection conditions, distinct genomes may interact within the same host cell, promoting error-prone template switching by RdRp and facilitating homologous recombination (Lee et al., 2021). This process results in mosaic genomes with novel genetic combinations, accelerating antigenic diversification and supporting the long-term persistence of FCV within feline populations (Coyne et al., 2012).

EPIDEMIOLOGY OF FELINE CALICIVIRUS

The prevalence and progression of FCV infection show considerable variation across regions and cat populations. In Beijing, the detection rate has reached 31% among owned cats (Wang et al., 2025), whereas in Hangzhou, prevalence is reported at approximately 43% (Zheng et al., 2021). Among stray cats in northeastern China, prevalence exceeds 45% (Wang et al., 2017), while in feral populations in Australia, FCV antibody seroprevalence approaches 70% (Amery-Gale et al., 2024). Globally, FCV infection rates among domestic

cats vary widely, ranging from approximately 9% to 48%, with an average prevalence typically between 22% and 30% (Afonso et al., 2017; Wei et al., 2024; Di Profio et al., 2025;). Several studies have reported higher FCV prevalence in shelter cats—ranging from 33% to 50%—compared to household cats, where prevalence is generally lower, around 8–20% (Zicola et al., 2009; Gourkow et al., 2013). This variability reflects the influence of environmental conditions, vaccination status, study design, diagnostic methodology, and population structure on infection rates (Afonso et al., 2017).

The risk of FCV infection increases significantly in kittens with immature immune systems, in inadequately vaccinated cats, and in individuals experiencing stress due to overcrowding, transportation, or relocation (Hofmann-Lehmann et al., 2022). These factors heighten susceptibility to infection and facilitate viral shedding and transmission, thereby increasing the likelihood of outbreaks in high-density environments such as shelters, breeding facilities, and multi-cat households (Coyne et al., 2007).

Some cats recovering from acute FCV infection may remain asymptomatic carriers, shedding the virus for months or even years (Hofmann-Lehmann et al., 2022). Studies indicate that most cats cease viral shedding within 30–75 days, during which neutralizing antibodies suppress viral replication and excretion by binding viral particles and preventing their entry into host cells, thereby reducing viremia and shedding duration (Abebe and Dejenie, 2023; Di Profio et al., 2025). However, this humoral response is not always sufficient to prevent carrier establishment, as the virus can persist in specific tissues such as the oropharynx and tonsils, where localized replication and quasispecies formation enable persistent infection without overt clinical signs (Di Profio et al., 2025).

In addition, FCV transmission is influenced by seasonal and management factors. Higher incidence rates have been reported during winter and early spring, coinciding with increased indoor crowding, poor ventilation, and seasonal kitten births (Gao et al., 2023; Umar et al., 2025). Nevertheless, FCV remains endemic year-round due to its strong environmental stability (Spiri et al., 2019). The virus can survive for up to 28 days on dry surfaces at room temperature, enabling transmission both through direct contact between cats and indirectly via contaminated fomites (Spiri et al., 2019; Al Hafid et al., 2022).

PATHOGENESIS OF FELINE CALICIVIRUS

Feline calicivirus enters the host primarily through the nasal, oral, or conjunctival routes, with the oropharynx serving as the principal site of initial viral replication (Hofmann-Lehmann et al., 2022). The viral capsid protein VP1 mediates entry into host cells by binding to the fJAM-A as the primary receptor, while α-2,6-linked sialic acid is thought to function as a co-receptor or attachment factor that enhances viral adsorption efficiency (Stuart and Brown, 2007). The involvement of these two receptors determines the virus's tissue tropism, infectivity, host range, and strain-dependent pathogenic potential (Bhella, 2015). Following receptor engagement, the virus is internalized via clathrin-mediated endocytosis, a process highly dependent on endosomal acidification to trigger uncoating of the viral capsid and release of the genome into the cytoplasm, enabling productive infection (Shivanna et al., 2014).

Infected cells exhibit characteristic cytopathic effects, including cell rounding, membrane

blebbing, and detachment from the substrate, accompanied by inhibition of host protein synthesis through cleavage of eukaryotic initiation factors (Di Profio et al., 2025). This translational shutdown disrupts essential cellular functions and induces caspase-dependent apoptosis, facilitating efficient release of progeny virions (Wu et al., 2021). A transient viremia occurs approximately 3–4 days post-infection, allowing systemic dissemination, while fJAM-A expression on epithelial, endothelial, leukocytic, and platelet surfaces facilitates hematogenous spread (Wei et al., 2024).

Viral replication predominantly occurs in the oral, respiratory, and conjunctival epithelia, producing vesicles that undergo epithelial necrosis and ulceration, often accompanied by neutrophilic infiltration (Karakaya-Bilen et al., 2025). Certain FCV strains can induce proliferative interstitial pneumonia or the so-called *limping syndrome*, an acute polyarthritis involving viral infiltration of synovial tissues, possibly through a type III hypersensitivity mechanism mediated by antigen–antibody immune complexes (Lanave et al., 2023; Wei et al., 2024). These immune complexes deposit within the synovium, activating complement and recruiting neutrophils that release proteolytic enzymes and reactive oxygen species, leading to localized inflammation (Wang, 2018; Kasiraja et al., 2025). In natural infections, this reaction occurs when viral antigens are still circulating as antibodies begin to develop, and in rare cases, similar responses have been observed following vaccination with replication-competent live-attenuated vaccines (Hofmann-Lehmann et al., 2022).

Severe cases may progress to VS-FCV, characterized by widespread vasculitis, subcutaneous edema, oral and cutaneous ulceration, and multiorgan necrosis affecting the liver, spleen, pancreas, and lungs, with high mortality rates—particularly in multi-cat populations (Duclos et al., 2024). Outbreak investigations have revealed that VS-FCV strains are genetically distinct and emerge independently through the accumulation of mutations, including the possible acquisition of novel glycosylation sites on the capsid protein, which may alter receptor interactions, antigenicity, and immune recognition (Lu et al., 2018; Bordicchia et al., 2021; Asif et al., 2025). In addition to virological factors, host-related conditions—such as age-dependent immune reactivity and dysregulated cytokine responses—also contribute to disease severity (Foley et al., 2006).

FCV persistence is further supported by complex immune evasion strategies, including antigenic drift, recombination, capsid epitope modification, host protein shut-off, and apoptosis modulation, enabling the virus to persist under adaptive immune pressure and giving rise to diverse clinical outcomes, including hypervirulent variants such as VS-FCV (Zhou et al., 2021; Wu et al., 2021).

IMMUNITY TO FELINE CALICIVIRUS

Understanding the immunological dynamics of FCV infection is crucial for both disease control and the development of effective vaccination strategies (Wang et al., 2023). In the early stages of life, kittens acquire passive protection through maternally derived antibodies (MDA), which are primarily transferred via colostrum. These maternal antibodies play a vital role in providing protection against FCV and other pathogens during the first few weeks of life (Wei et al., 2024). The half-life of maternal antibodies against FCV is estimated to be approximately 15 days, and they can generally be detected until 10–14 weeks of age (Radford et al., 2009; Di Profio et al., 2025). However,

field studies have shown substantial variation, with some kittens losing protective antibody levels as early as six weeks of age, while others retain them for a longer duration (DiGangi et al., 2012; Jakel et al., 2012).

Although protective, the presence of maternal antibodies poses a challenge because they can interfere with vaccine efficacy by neutralizing the vaccine virus before the kitten's immune system develops an adaptive response (Egberink et al., 2022; Veronesi and Fusi, 2023). To address this issue, international guidelines recommend a stepwise vaccination protocol. Primary FCV vaccination should begin at 6–8 weeks of age and be repeated every 3–4 weeks until approximately 16 weeks of age. Furthermore, an additional booster at 6 or 12 months is advised to ensure the establishment of active immunity after the decline of maternal antibodies, particularly in high-risk cats or in cases where maternal antibody interference is likely (Radford et al., 2009; Scherk et al., 2013; Egberink et al., 2022). This strategy ensures that at least one effective vaccine dose is administered after MDA levels fall below the interference threshold, resulting in optimal active immunity.

As maternal antibodies wane, long-term protection depends entirely on the development of active immunity through vaccination or natural infection (Egberink et al., 2022). Following FCV exposure or vaccination, neutralizing antibodies are typically detectable between days 7 and 14. The immune response to homologous strains develops more rapidly than that to heterologous ones (Cao et al., 2023). Moreover, $CD4^+$ helper T cells and $CD8^+$ cytotoxic T cells are involved in viral clearance and contribute to cross-strain immunity following both live-attenuated and inactivated vaccination (Spiri et al., 2021).

On the other hand, FCV exhibits a variety of immune evasion strategies that enable viral persistence within the host. The virus can suppress interferon responses, modulate apoptosis, and inhibit host protein synthesis (Wu et al., 2021). In addition, alterations in antigenic epitopes contribute to the virus's ability to evade immune recognition (Tian et al., 2020). Non-structural proteins such as P30, P32, and P39 are involved in modulating cellular pathways, including the induction of autophagy, which inhibits RIG-I signaling and suppresses type I interferon production (Mao et al., 2023). FCV has also been reported to downregulate IRF-1 and modulate IFNAR1 and IRF-3 expression, thereby reducing viral RNA detection and delaying the activation of innate antiviral responses (Yumiketa et al., 2016; Liu et al., 2018; Tian et al., 2020). These evasion mechanisms facilitate viral persistence in lymphoid and oropharyngeal tissues and contribute to sustained transmission within cat populations.

CLINICAL MANIFESTATIONS OF FELINE CALICIVIRUS

The most common and classical manifestation of FCV infection is acute disease affecting the oral cavity and upper respiratory tract (Hofmann-Lehmann et al., 2022). Characteristic lesions include ulcerations on the tongue and oral mucosa, often accompanied by sneezing, nasal discharge, fever, anorexia, and hypersalivation secondary to oral pain (Di Profio et al., 2025). In some cases, infection may extend to the lower respiratory tract, resulting in pneumonia characterized by dyspnea, coughing, lethargy, and persistent fever (Maboni et al., 2024). Although pneumonia solely attributable to FCV is relatively uncommon, it can be clinically significant when

exacerbated by co-infections (Maboni et al., 2024).

Beyond respiratory involvement, FCV infection can also induce musculoskeletal disease known as limping syndrome (Balboni et al., 2022). This condition is characterized by acute, shifting lameness affecting one or more limbs, accompanied by joint pain, swelling, reluctance to move, fever, and malaise (Lanave et al., 2023). Although typically self-limiting, these signs can cause considerable discomfort in affected cats.

Direct evidence of viral involvement in joint pathology has been demonstrated through the detection of FCV RNA and antigens in synovial tissues (Lanave et al., 2023). Moreover, immune-mediated mechanisms are thought to contribute, particularly type III hypersensitivity resulting from immune complex deposition (ABCD, 2025). This mechanism is suspected to underlie cases of post-infectious polyarthritis and, more rarely, those occurring after vaccination (Wei et al., 2024).

The most severe manifestation is VS-FCV, characterized by an acute onset of systemic illness including high fever, lethargy, anorexia, mucocutaneous ulceration, facial and limb edema, skin erythema or necrosis, jaundice, and multi-organ involvement such as hepatic and pulmonary damage (Duclos et al., 2024). The case fatality rate is markedly higher than that of classical FCV infections, particularly in naïve or stressed cat populations (Duclos et al., 2024).

THERAPEUTIC AND PREVENTIVE APPROACHES AGAINST FELINE CALICIVIRUS

Treatment options for FCV infection remain limited. To date, no small-molecule antivirals have been officially approved or consistently demonstrated clinical efficacy against FCV infection (Radford et al., 2009; Fumian et al., 2018). Most current management approaches rely on supportive care and the prevention or control of secondary bacterial infections to minimize complications.

In recent years, there has been growing interest in therapies targeting both the virus and host pathways (Beran et al., 2025; Gu et al., 2025). Recombinant feline interferon-omega (rFeIFN-ω) has been used as an immunomodulatory and antiviral adjunct in both clinical and experimental settings (Gil et al., 2013). However, published data remain inconsistent, and its clinical benefit has not been conclusively demonstrated, preventing its recommendation as a definitive antiviral therapy.

Several experimental compounds have shown in vitro activity against FCV, with some yielding promising results in controlled challenge models (Wu et al., 2015). Nitazoxanide, for example, inhibits FCV replication in cell culture and reduces clinical signs, viral load, and shedding in experimentally infected cats (Cui et al., 2020). The compound also exhibits synergistic effects with other agents in vitro, supporting drug repurposing strategies for short-term development, though further studies are needed to establish dosing and tolerability (Cui et al., 2020). Other potential strategies include broad-spectrum polymerase and protease inhibitors identified through in vitro screening, as well as small molecules or metal complexes such as copper chloride, which show dose-dependent antiviral activity and synergy with ribavirin (Li et al., 2020).

However, significant challenges remain in translating laboratory findings to clinical practice, including FCV's genetic and antigenic diversity, limited pharmacokinetic and toxicity data in cats, and a lack of statistically powered controlled clinical trials (Cao et al., 2022).

Rational strategies should prioritize: (i) repurposing compounds with established in vivo safety and efficacy profiles (McDonagh et al., 2015); (ii) combination therapy to reduce effective doses and minimize resistance (Li et al., 2020); (iii) the development of structure-based direct-acting protease or polymerase inhibitors (Wang et al., 2015); and (iv) coordinated clinical trials in shelters or outbreak settings to assess clinical benefit and effects on viral shedding (Cui et al., 2020).

Several nucleoside analogues and other small molecules, including 2′-C-methylcytidine (2CMC), NITD008, ribavirin, fexaramine, and rFeIFN-ω, have demonstrated anti-FCV activity (Ballin et al., 2014; Kim and Chang, 2018; Enosi et al., 2019; Cui et al., 2020). 2CMC and nitazoxanide exhibit low-micromolar EC_{50} values, while NITD008 shows sub-micromolar potency and a favorable toxicity profile, making it a strong candidate for further development (Fumian et al., 2018; Cui et al., 2020). Non-nucleoside approaches are also relevant: fexaramine inhibits FCV entry into cells, although resistance can arise from single mutations in the VP1 P2 domain (Kim and Chang, 2018). rFeIFN-ω continues to be used as an adjuvant therapy for FCV infection. Some studies report that rFeIFN-ω administration reduces clinical scores and viral loads, particularly in chronic cases such as stomatitis or in retrovirus-positive cats (Matsumoto et al., 2018; Garrett et al., 2023). Nevertheless, controlled clinical trials have yielded inconsistent results, with some studies showing no significant difference from placebo, likely due to variations in dosing protocols, immune status, and clinical condition of the animals (Ballin et al., 2014).

Because no effective and licensed antiviral therapy currently exists, supportive care remains the cornerstone of FCV management. This approach is palliative and aims to stabilize the patient and minimize secondary complications (Radford et al., 2009). Standard interventions include fluid therapy to correct dehydration, analgesics for oral ulcer pain, broad-spectrum antibiotics to control secondary bacterial infections, and nutritional support ranging from highly palatable food to feeding tube placement in cases of prolonged anorexia (Hofmann-Lehmann et al., 2022; Taylor et al., 2022). In severe respiratory cases, topical or nebulized saline and mucolytics such as bromhexine may help humidify airways, loosen secretions, and maintain mucosal function. However, potent mucolytics such as N-acetylcysteine should be used cautiously due to potential adverse effects, including increased airway resistance (Radford et al., 2009; Hofmann-Lehmann et al., 2022).

In VS-FCV cases, more aggressive immunomodulatory interventions such as systemic corticosteroids or exogenous interferons are occasionally used to mitigate life-threatening inflammation (Duclos et al., 2024). Corticosteroids may help suppress tissue damage caused by the host inflammatory response, particularly in stomatitis or severe inflammatory presentations associated with FCV infection (Soltero-Rivera et al., 2023).

For chronic stomatitis associated with FCV, management principles include reducing oral antigenic stimulation through intensive dental therapy, combined with immunomodulatory treatments such as short-term systemic corticosteroids, cyclosporine, or rFeIFN-ω (Soltero-Rivera et al., 2023). Evidence of benefit comes primarily from small or uncontrolled studies, so clinical decisions should be individualized and accompanied by long-term monitoring (Matsumoto et al., 2018).

Several experimental studies have also evaluated the antiviral activity of natural

compounds against FCV, including flavonoids such as quercetagetin, essential oils (e.g., lemon oil), and bacterial metabolites from *Lactococcus lactis* (Fumian et al., 2018; Pellegrini et al., 2023). These substances exhibit in vitro virucidal or replication-inhibitory effects, though in vivo and clinical evidence remains limited. The secretome produced by adipose-derived mesenchymal stem cells (AD-MSCs) has shown dose-dependent inhibition of FCV and feline herpesvirus type 1 (FHV-1) replication in cell cultures (Teshima et al., 2022). The proposed mechanism involves modulation of host gene expression related to antiviral and inflammatory pathways (Liu et al., 2018). Proteomic analyses of AD-MSC secretomes have revealed immunomodulatory cytokines and growth factors that may contribute to this activity. While in vitro results are promising, in vivo data and controlled clinical trials remain scarce, and the therapeutic efficacy of such secretomes in cats requires further validation.

Prevention remains the cornerstone of FCV control, with vaccination being the most effective tool. According to international guidelines such as WSAVA and AAHA/AAFP, FCV is classified as a core vaccine for cats, and vaccination protocols should be tailored to age, exposure risk, and environmental factors (Richards et al., 2020; WSAVA VGG, 2024). Modified-live FCV vaccines reduce clinical severity, viral load, and RNAemia duration following heterologous challenge but do not always prevent infection or viral shedding, functioning primarily to mitigate disease rather than confer sterilizing immunity (Spiri et al., 2021).

ENVIRONMENTAL CONTROL AND DISINFECTION

Feline calicivirus spreads efficiently through direct contact and contaminated fomites, particularly under conditions of high population density, stress, or the introduction of new cats with uncertain immune or vaccination status (Hofmann-Lehmann et al., 2022; Yang et al., 2024). Unlike enveloped viruses, FCV lacks a phospholipid membrane, leaving the VP1 capsid protein as the sole protective structure for its RNA genome (Asif et al., 2025). The absence of a lipid envelope directly contributes to the remarkable environmental stability of FCV and explains its resistance to many lipid-based detergents and disinfectants (Narula et al., 2024). Experimental studies have demonstrated that FCV can remain viable for several days to weeks on both moist and dry surfaces, retaining infectivity across a broad range of temperatures and humidity levels (Wißmann et al., 2021). Interestingly, the virus persists longer under low relative humidity (approximately 30%) compared to high humidity (70%) (Spiri et al., 2019).

These physicochemical properties have major implications for disease control and epidemiology. Environmental persistence enables transmission not only through direct cat-to-cat contact but also indirectly via contaminated objects such as food bowls, cages, bedding, clothing, and human hands (Hofmann-Lehmann et al., 2022). This persistence explains why outbreaks frequently continue in shelters, veterinary clinics, and multi-cat environments despite routine cleaning. Consequently, effective biosecurity requires the use of disinfectants proven to be active against non-enveloped caliciviruses—such as chlorine-based compounds, strong oxidizing agents, or validated specialty formulations—combined with thorough sanitation and strict population management (Whitehead and McCue, 2010).

For washable textiles and fabrics, experimental evidence and public health guidelines indicate that laundering at high temperatures (≥60 °C) with enzymatic detergent, supplemented with bleach when compatible with the material, significantly reduces viral load and disrupts transmission (Rutala and Weber, 2024). The mechanical action of washing and subsequent heat drying (tumble drying) further enhances decontamination efficacy (Tano and Melhus, 2014). Therefore, laundry protocols in shelters or veterinary facilities should account for water temperature, detergent composition, and segregation between contaminated and clean materials.

Non-washable surfaces require chemical disinfection using agents proven effective against non-enveloped viruses. Laboratory and field data (including norovirus models) support the use of sodium hypochlorite solutions at concentrations of 1,000–5,000 ppm available chlorine (corresponding to 1:50–1:10 dilutions of household bleach, depending on the product) with adequate contact time (Hall et al., 2011). Quantitative tests with FCV have shown that approximately 2,700 ppm hypochlorite can achieve >5-$\log_{10}$ viral reduction on stainless steel surfaces within one minute (Chiu et al., 2015). Alternative oxidizing systems such as peracetic acid and accelerated hydrogen peroxide (AHP) demonstrate comparable virucidal activity and are useful when chlorine is unsuitable due to corrosiveness or odor (Campagna et al., 2016). Conversely, quaternary ammonium compounds (QUATs) and 70% alcohol solutions exhibit limited or formulation-dependent efficacy against FCV, often requiring prolonged contact times or specific additives to achieve meaningful inactivation (Whitehead and McCue, 2010). The presence of organic matter (mucus, feces, blood) markedly decreases the effectiveness of any disinfectant; thus, preliminary cleaning is essential before chemical disinfection (Saha, 2022).

Alcohol-based disinfectants (ethanol, isopropanol, propanol) display variable performance because the non-enveloped structure of FCV makes it more resistant to lipid-dissolving mechanisms (Kampf, 2018). Their effectiveness depends on the type of alcohol, concentration, pH, formulation additives, and contact duration (Meyers et al., 2021). Under controlled experimental conditions, 1-propanol exhibits the highest virucidal activity, followed by ethanol, while 2-propanol is the least effective (Narula et al., 2024). Alkaline ethanol formulations (pH 10.8–12.0) significantly enhance inactivation (Ruhlandt et al., 2023), although their use must consider material compatibility and user safety.

CONCLUSION

Feline calicivirus continues to pose major challenges due to its efficient transmission, high genetic variability, and remarkable environmental stability. Although current control relies on vaccination, supportive therapy, and strict hygiene, outbreaks remain frequent in high-density populations, reflecting the limited breadth of vaccine-induced protection and the absence of licensed antivirals. Future advances should focus on next-generation vaccines targeting conserved epitopes, host-directed or combination antiviral therapies, and innovative yet practical disinfection strategies. Integrating these biomedical advances with population management and environmental control will be critical to achieving more effective and sustainable FCV control while improving feline health and welfare.

REFRENCES

1. ABCD. Guideline for adverse reactions to vaccination. ABCD cats & vets website. Accessed 2025 Oct 6. Available from: https://www.abcdcatsvets.org/guideline-for-adverse-reactions-to-vaccination/
2. Abebe EC, Dejenie TA. Protective roles and protective mechanisms of neutralizing antibodies against SARS-CoV-2 infection and their potential clinical implications. *Front Immunol*. 2023;14:1055457. doi:10.3389/fimmu.2023.1055457
3. Afonso MM, Pinchbeck GL, Smith SL, Daly JM, Gaskell RM, Dawson S, Radford AD. A multi-national European cross-sectional study of feline calicivirus epidemiology, diversity and vaccine cross-reactivity. *Vaccine*. 2017;35(20):2753-60. doi:10.1016/j.vaccine.2017.03.030
4. Al Hafid MK, Susetya H, Nugroho WS. Cat viral diseases pattern in Prof. Soeparwi Animal Hospital in 2017–2019. In: 2nd International Conference on Tropical Wetland Biodiversity and Conservation; 2022. *IOP Conf Ser Earth Environ Sci*. 2022;976:012012. doi:10.1088/1755-1315/976/1/012012
5. Amery-Gale J, Woinarski J, Hartley CA, Devlin JM. High prevalence of antibodies against feline calicivirus in Australian feral and stray cat (Felis catus) populations. *Aust Vet J*. 2024;102(11):550-63. doi:10.1111/avj.13369
6. Asif S, Yingkun D, Meng C. Unlocking the secrets of feline calicivirus: advances in structural and nonstructural proteins and its role as a key model for other caliciviruses. *Virol J*. 2025;22:152. doi:10.1186/s12985-025-02750-6
7. Balboni A, Verin R, Buldrini I, Zamagni S, Morini M, Terrusi A, Gallina L, Urbani L, Dondi F, Battilani M. Natural cases of polyarthritis associated with feline calicivirus infection in cats. *Vet Res Commun*. 2022;46(2):613-19. doi:10.1007/s11259-022-09933-4
8. Ballin AC, Schulz B, Helps C, Sauter-Louis C, Mueller RS, Hartmann K. Limited efficacy of topical recombinant feline interferon-omega for treatment of cats with acute upper respiratory viral disease. *Vet J*. 2014;202(3):466-70. doi:10.1016/j.tvjl.2014.09.030
9. Beran RK, Vijjapurapu A, Nair V, Du Pont V. Host-targeted antivirals as broad-spectrum inhibitors of respiratory viruses. *Curr Opin Virol*. 2025;73:101492. doi:10.1016/j.coviro.2025.101492
10. Bhella D. The role of cellular adhesion molecules in virus attachment and entry. *Philos Trans R Soc Lond B Biol Sci*. 2015;370(1661):20140035. doi:10.1098/rstb.2014.0035
11. Bordicchia M, Fumian TM, Van Brussel K, Russo AG, Carrai M, Le S-J, Pesavento PA, Holmes EC, Martella V, White P, Beatty JA, Shi M, Barrs VR. Feline calicivirus virulent systemic disease: clinical epidemiology, analysis of viral isolates and in vitro efficacy of novel antivirals in Australian outbreaks. *Viruses*. 2021;13(10):2040. doi:10.3390/v13102040
12. Campagna MV, Faure-Kumar E, Treger JA, Cushman JD, Grogan TR, Kasahara N, Lawson GW. Factors in the selection of surface disinfectants for use in a laboratory animal setting. *J Am Assoc Lab Anim Sci*. 2016;55(2):175-88.
13. Cao L, Liu J, Li Y, Xie D, Yan Q, Li Q, Cao Y, Du W, Li J, Ye Z, Zhou D, Kang C, Cao S. Biological characteristics of feline calicivirus epidemic strains in china and screening of broad-spectrum protective vaccine strains. *Vaccines*. 2023;11(12):1858. doi:10.3390/vaccines11121858
14. Cao L, Li Q, Shi K, et al. Isolation and phylogenetic analysis of feline calicivirus strains from various region of China. *Anim Dis*. 2022;2:16. doi:10.1186/s44149-022-00047-7
15. Chiu S, Skura B, Petric M, McIntyre L, Gamage B, Isaac-Renton J. Efficacy of common disinfectant/cleaning agents in inactivating murine norovirus and feline calicivirus as surrogate viruses for human norovirus. *Am J Infect Control*. 2015;43(11):1208-12. doi:10.1016/j.ajic.2015.06.021

16. Cisternas F, Navarro C, Jara MA. Feline calicivirus. *Mol Detect Primers Des*. 2020;5(7):54-72.
17. Coyne KP, Christley RM, Pybus OG, Dawson S, Gaskell RM, Radford AD. Large-scale spatial and temporal genetic diversity of feline calicivirus. *J Virol*. 2012;86(2). doi:10.1128/JVI.00701-12
18. Coyne KP, Edwards D, Radford AD, Cripps P, Jones D, Wood JL, Gaskell RM, Dawson S. Longitudinal molecular epidemiological analysis of feline calicivirus infection in an animal shelter: a model for investigating calicivirus transmission within high-density, high-turnover populations. *J Clin Microbiol*. 2007 Oct;45(10):3239-44. doi:10.1128/JCM.01226-07
19. Cubillos-Zapata C, Angulo I, Almanza H, Borrego B, Zamora-Ceballos M, Castón JR, Mena I, Blanco E, Bárcena J. Precise location of linear epitopes on the capsid surface of feline calicivirus recognized by neutralizing and non-neutralizing monoclonal antibodies. *Vet Res*. 2020;51(1):59. doi:10.1186/s13567-020-00785-x
20. Cui Z, Li D, Xie Y, Wang K, Zhang Y, Li G, Zhang Q, Chen X, Teng Y, Zhao S, Shao J, Xingmeng F, Zhao Y, Du D, Guo Y, Huang H, Dong H, Hu G, Zhang S, Zhao Y. Nitazoxanide protects cats from feline calicivirus infection and acts synergistically with mizoribine in vitro. *Antiviral Res*. 2020;182:104827. doi:10.1016/j.antiviral.2020.104827
21. Digangi BA, Levy JK, Griffin B, Reese MJ, Dingman PA, Tucker SJ, Dubovi EJ. Effects of maternally-derived antibodies on serologic responses to vaccination in kittens. *J Feline Med Surg*. 2012;14(2):118-23. doi:10.1177/1098612X11432239
22. Di Profio F, Carnevale M, Marsilio F, Pellegrini F, Martella V, Di Martino B, Sarchese V. Feline calicivirus infection: current understanding and implications for control strategies. *Animals*. 2025;15(14):2009. doi:10.3390/ani15142009
23. Duclos AA, Guzmán Ramos PJ, Mooney CT. Virulent systemic feline calicivirus infection: a case report and first description in Ireland. *Ir Vet J*. 2024;77:1. doi:10.1186/s13620-024-00262-3
24. Egberink H, Frymus T, Hartmann K, Möstl K, Addie DD, Belák S, Boucraut-Baralon C, Hofmann-Lehmann R, Lloret A, Marsilio F, Pennisi MG, Tasker S, Thiry E, Truyen U, Hosie MJ. Vaccination and antibody testing in cats. *Viruses*. 2022;14(8):1602. doi:10.3390/v14081602
25. Enosi Tuipulotu D, Fumian TM, Netzler NE, Mackenzie JM, White PA. The adenosine analogue NITD008 has potent antiviral activity against human and animal caliciviruses. *Viruses*. 2019;11:496. doi:10.3390/v11060496
26. Foley J, Hurley K, Pesavento PA, Poland A, Pedersen NC. Virulent systemic feline calicivirus infection: Local cytokine modulation and contribution of viral mutants. *J Feline Med Surg*. 2006;8(1):55–61. doi:10.1016/j.jfms.2005.08.002
27. Fumian TM, Tuipulotu DE, Netzler NE, Lun JH, Russo AG, Yan GJH, White PA. Potential therapeutic agents for feline calicivirus infection. *Viruses*. 2018;10(8):433. doi:10.3390/v10080433
28. Gao J, Li Y, Xie Q, Al-zaban MI, Al-Saeed FA, Shati AA, Al-Doaiss AA, Ahmed AE, Nawaz S, Ebrahem H, Irshad I, Kulyar MF, Li J. Epidemiological investigation of feline upper respiratory tract infection encourages a geographically specific FCV vaccine. *Vet Sci*. 2023;10(1):46. doi:10.3390/vetsci10010046
29. Garrett R. The efficacy of recombinant feline interferon-omega in treating symptomatic cats infected with feline immunodeficiency virus. *Vet Evid*. 2023;8(3):1–12. doi:10.18849/ve.v8i3.666
30. Gil S, Leal RO, Duarte A, McGahie D, Sepúlveda N, Siborro I, Cravo J, Cartaxeiro C, Tavares LM. Relevance of feline interferon omega for clinical improvement and reduction of concurrent viral excretion in retrovirus infected cats from a rescue shelter. *Res Vet Sci*. 2013;94(3):753-63. doi:10.1016/j.rvsc.2012.09.025
31. Gourkow N, Lawson JH, Hamon SC, Phillips CJ. Descriptive epidemiology of upper respiratory disease and associated risk factors

in cats in an animal shelter in coastal western Canada. *Can Vet J*. 2013;54(2):132–8.

32. Gu X, Zheng M, Gao Y, Lin S, Zhang X, Chen C, Zhu H, Sun W, Zhang Y. Overview of host-directed antiviral targets for future research and drug development. *Acta Pharm Sin B*. 2025;15(4):1723-51. doi:10.1016/j.apsb.2025.03.011
33. Hall AJ, Vinjé J, Lopman B, Park GW, Yen C, Gregoricus N, Parashar UD; Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, CDC. Updated norovirus outbreak management and disease prevention guidelines. MMWR Recomm Rep. 2011 Mar 4;60(RR-3):1-15. Available from: https://www.cdc.gov/mmwr/preview/mmwrhtml/rr6003a1.htm
34. Heng W, Zang D, Li R, Jiang Q, Liu J, Jia H, Kang H. A novel replication-deficient FCV vaccine provides strong immune protection in cats. *J Virol*. 2025;99(8):e0009325. doi:10.1128/jvi.00093-25
35. Hofmann-Lehmann R, Hosie MJ, Hartmann K, Egberink H, Truyen U, Tasker S, Belák S, Boucraut-Baralon C, Frymus T, Lioret A, Marsilio F, Pennisi MG, Addie DD, Lutz H, Thiry E, Radford AD, Möstl K. Calicivirus infection in cats. *Viruses*. 2022;14(5):937. doi:10.3390/v14050937
36. Jakel V, Cussler K, Hanschmann KM, Truyen U, König M, Kamphuis E, Duchow K. Vaccination against feline panleukopenia: implications from a field study in kittens. *BMC Vet Res*. 2012;8:62. doi:10.1186/1746-6148-8-62
37. Kampf G. Efficacy of ethanol against viruses in hand disinfection. *J Hosp Infect*. 2018;98(4):331-8. doi:10.1016/j.jhin.2017.10.014
38. Karakaya-Bilen E, Akgül G, Yılmaz-Koc O. Suspected feline calicivirus infection triggering ulcerative oral and skin lesions in cats following routine ovariohysterectomy: a postoperative risk assessment. *Vet Med Sci*. 2025;11(5):e70540. doi:10.1002/vms3.70540
39. Kasiraja V, Bakar NAA, Suliman NA. Rheumatoid arthritis unmasked: the immune complex as a key driver of disease progression. *Explor Immunol*. 2025;5:1003208. doi:10.37349/ei.2025.1003208
40. Kim Y, Chang KO. Fexaramine as an entry blocker for feline caliciviruses. *Antiviral Res*. 2018;152:76-83. doi:10.1016/j.antiviral.2018.02.009
41. Lanave G, Buonavoglia A, Pellegrini F, Di Martino B, Di Profio F, Diakoudi G, Catella C, Omar AH, Vasinioti VI, Cardone R, Santo G, Martella V, Camero M. An outbreak of limping syndrome associated with feline calicivirus. *Animals*. 2023;13(11):1778. doi:10.3390/ani13111778
42. Lee SY, Kim YK, Kim YS, Na EJ, Kim YJ, Oem JK. Intergenic recombination in feline calicivirus associated with a hemorrhagic-like disease in the Republic of Korea. *Acta Virol*. 2021;65(2):232-6. doi:10.4149/av_2021_206
43. Li D, Cui Z, Li G, Zhang L, Zhang Y, Zhao H, Zhang S, Guo Y, Zhao Y, Men F, Zhao S, Shao J, Du D, Huang H, Wang K, Hu G, Li T, Zhao Y. Antiviral effect of copper chloride on feline calicivirus and synergy with ribavirin in vitro. *BMC Vet Res*. 2020;16(1):231. doi:10.1186/s12917-020-02441-0
44. Li L, Liu Z, Shi J, Yang M, Yan Y, Fu Y, Shen Z, Peng G. The CDE region of feline Calicivirus VP1 protein is a potential candidate subunit vaccine. *BMC Vet Res*. 2024;20:379. doi:10.1186/s12917-024-03914-2
45. Liu Y, Liu X, Kang H, Hu X, Liu J, Tian J, Qu L. Identification of feline interferon regulatory factor 1 as an efficient antiviral factor against the replication of feline calicivirus and other feline viruses. *Biomed Res Int*. 2018 Jun 12;2018:2739830. doi:10.1155/2018/2739830
46. Lu Z, Ledgerwood ED, Hinchman MM, Dick R, Parker JSL. Conserved surface residues on the feline calicivirus capsid are essential for interaction with its receptor feline junctional adhesion molecule A (fJAM-A). *J Virol*. 2018;92(5):e00035-18. doi:10.1128/JVI.00035-18
47. Maboni G, Che S, Tallmadge R, De Luca E, Goodman LB, Weese JS, Sanchez S. Feline respiratory disease complex: insights into the role of viral and bacterial co-infections. *Front*

Microbiol. 2024;15:1455453. doi:10.3389/fmicb.2024.1455453

48. Mao J, Ye S, Deng J, Song J, Wang Z, Chen A, Zhou P, Li S. Feline calicivirus p39 inhibits innate immune responses by autophagic degradation of retinoic acid inducible gene I. *Int J Mol Sci*. 2023;24(6):5254. doi:10.3390/ijms24065254
49. Matsumoto H, Teshima T, Iizuka Y, Sakusabe A, Takahashi D, Amimoto A, Koyama H. Evaluation of the efficacy of the subcutaneous low recombinant feline interferon-omega administration protocol for feline chronic gingivitis-stomatitis in feline calicivirus-positive cats. *Res Vet Sci*. 2018;121:53-8. doi:10.1016/j.rvsc.2018.10.003
50. McDonagh P, Sheehy PA, Fawcett A, Norris JM. Antiviral effect of mefloquine on feline calicivirus in vitro. *Vet Microbiol*. 2015;176(3-4):370-7. doi:10.1016/j.vetmic.2015.02.007
51. Meyers C, Kass R, Goldenberg D, Milici J, Alam S, Robison R. Ethanol and isopropanol inactivation of human coronavirus on hard surfaces. *J Hosp Infect*. 2021;107:45-9. doi:10.1016/j.jhin.2020.09.026
52. Narula P, Lokshman MK, Pathak SB, Mukherjee S, Banerjee M. Chemical inactivation of two non-enveloped viruses results in distinct thermal unfolding patterns and morphological alterations. *BMC Microbiol*. 2024;24(1):413. doi:10.1186/s12866-024-03565-1
53. Pellegrini F, Camero M, Catella C, Fracchiolla G, Sblano S, Patruno G, Trombetta CM, Galgano M, Pratelli A, Tempesta M, Martella V, Lanave G. Virucidal activity of lemon essential oil against feline calicivirus used as surrogate for norovirus. *Antibiotics*. 2023;12(2):322. doi:10.3390/antibiotics12020322
54. Radford AD, Addie D, Belák S, Boucraut-Baralon C, Egberink H, Frymus T, Gruffydd-Jones T, Hartmann K, Hosie MJ, Lloret A, Lutz H, Marsilio F, Pennisi MG, Thiry E, Truyen U, Horzinek MC. Feline calicivirus infection. ABCD guidelines on prevention and management. *J Feline Med Surg*. 2009;11(7):556-64. doi:10.1016/j.jfms.2009.05.004
55. Richards JR, Elston TH, Ford RB, Gaskell RM, Hartmann K, Hurley KF, Lappin MR, Levy JK, Rodan I, Scherk M, Schultz RD, Sparkes AH. 2020 AAHA/AAFP feline vaccination guidelines. *J Am Anim Hosp Assoc*. 2020;56(4):249–65. doi:10.5326/JAAHA-MS-7123
56. Ruhlandt M, Becker B, Paulmann D, Dotzauer A, Arndt A, Todt D, Steinmann E, Steinmann J, Brill FH. Impact of concentration, temperature and pH on the virucidal activity of alcohols against human adenovirus. *Am J Infect Control*. 2023;51(9):1011-6. doi:10.1016/j.ajic.2023.01.014
57. Rutala WA, Weber DJ; Healthcare Infection Control Practices Advisory Committee (HICPAC). Guideline for disinfection and sterilization in healthcare facilities, 2008. Update June 2024. Chapel Hill (NC): University of North Carolina Health Care System; 2024. [cited 2025 Oct 7]. Available from: https://www.cdc.gov/infection-control/hcp/disinfection-and-sterilization/index.html
58. Saha R. Cleaning and disinfection: an important pillar of contamination control. Am Pharm Rev. 2022 Jun 1. Available from: https://www.americanpharmaceuticalreview.com/Featured-Articles/587075-Cleaning-and-Disinfection-An-Important-Pillar-of-Contamination-Control/
59. Scherk MA, Ford RB, Gaskell RM, Hartmann K, Hurley KF, Lappin MR, Levy JK, Little SE, Nordone SK, Sparkes AH. 2013 AAFP feline vaccination advisory panel report. *J Feline Med Surg*. 2013;15(9):785-808. doi:10.1177/1098612X13500429. Erratum in: *J Feline Med Surg*. 2013;15(11):NP2. Erratum in: *J Feline Med Surg*. 2014;16(1):66.
60. Shivanna V, Kim Y, Chang KO. Endosomal acidification and cathepsin L activity is required for calicivirus replication. *Virology*. 2014;464-465:287-95. doi:10.1016/j.virol.2014.07.025
61. Smertina E, Hall RN, Urakova N, Strive T, Frese M. Calicivirus non-structural proteins: potential functions in replication and host cell

manipulation. *Front Microbiol*. 2021;12:712710. doi:10.3389/fmicb.2021.712710

62. Soltero-Rivera M, Goldschmidt S, Arzi B. Feline chronic gingivostomatitis: current concepts in clinical management. *J Feline Med Surg*. 2023;25(8):1-15. doi:10.1177/1098612X231186834
63. Spiri AM. An update on feline calicivirus. *Schweiz Arch Tierheilkd*. 2022;164(3):225–41. doi:10.17236/sat00346
64. Spiri AM, Meli ML, Riond B, Herbert I, Hosie MJ, Hofmann-Lehmann R. Environmental contamination and hygienic measures after feline calicivirus field strain infections of cats in a research facility. *Viruses*. 2019;11(10):958. doi:10.3390/v11100958
65. Spiri AM, Novacco M, Meli ML, Stirn M, Riond B, Fogle JE, Boretti FS, Herbert I, Hosie MJ, Hofmann-Lehmann R. Modified-live feline calicivirus vaccination elicits cellular immunity against a current feline calicivirus field strain in an experimental feline challenge study. *Viruses*. 2021;13(9):1736. doi:10.3390/v13091736
66. Stuart AD, Brown TDK. Alpha2,6-linked sialic acid acts as a receptor for Feline calicivirus. *J Gen Virol*. 2007;88(Pt 1):177-86. doi:10.1099/vir.0.82158-0
67. Stuntebeck E. Feline calicivirus (FCV). Shelter Medicine. University of Wisconsin–Madison School of Veterinary Medicine; 2024 Dec 11. Available from: https://sheltermedicine.wisc.edu/library/resources/feline-calicivirus-virulent-systemic-feline-calicivirus-vs-fcv
68. Tano E, Melhus A. Level of decontamination after washing textiles at 60°C or 70°C followed by tumble drying. *Infect Ecol Epidemiol*. 2014;4:24314. doi:10.3402/iee.v4.24314
69. Taylor S, Chan DL, Villaverde C, Ryan L, Peron F, Quimby J, O'Brien C, Chalhoub S. 2022 ISFM consensus guidelines on management of the inappetent hospitalised cat. *J Feline Med Surg*. 2022 Jul;24(7):614-40. doi:10.1177/1098612X221106353. Erratum in: *J Feline Med Surg*. 2023 Feb;25(2):1098612X221149867. doi:10.1177/1098612X221149867
70. Teshima T, Yasumura Y, Suzuki R, Matsumoto H. Antiviral effects of adipose tissue-derived mesenchymal stem cells secretome against feline calicivirus and feline herpesvirus type 1. *Viruses*. 2022;14(8):1687. doi:10.3390/v14081687
71. Tian J, Kang H, Huang J, Li Z, Pan Y, Li Y, Chen S, Zhang J, Yin H, Qu L. Feline calicivirus strain 2280 p30 antagonizes type I interferon-mediated antiviral innate immunity through directly degrading IFNAR1 mRNA. *PLoS Pathog*. 2020;16(10):e1008944. doi:10.1371/journal.ppat.1008944
72. Umar S, Muhammad S, Mouzahim M, Pleva SM, Zhongqi Q, Weidong Y, Gao D. Burden of common respiratory pathogens among cats in China. *Vet Med Sci*. 2025;11(1):e70082. doi:10.1002/vms3.70082
73. Veronesi MC, Fusi J. Feline neonatology: From birth to commencement of weaning - what to know for successful management. *J Feline Med Surg*. 2022;24(3):232-42. doi:10.1177/1098612X221079709
74. Wang D, Zhu J, Yang H, Lyu Y. Epidemiology and molecular characterization of feline calicivirus in Beijing, China. *Animals*. 2025;15(4):494. doi:10.3390/ani15040494
75. Wang F, Chen C, Liu X, Yang K, Xu X, Yang H. Crystal structure of feline infectious peritonitis virus main protease in complex with synergetic dual inhibitors. *J Virol*. 2015;90(4):1910-7. doi:10.1128/JVI.02685-15
76. Wang J. Neutrophils in tissue injury and repair. *Cell Tissue Res*. 2018;371(3):531-9. doi:10.1007/s00441-017-2785-7
77. Wang K, Pei Z, Hu G. First report of feline calicivirus (FCV) infection in stray cats in northeast China. *Pol J Vet Sci*. 2017;20(3):595-8. doi:10.1515/pjvs-2017-0072
78. Wang ZL. Feline calicivirus (FCV): from molecular characteristics to vaccine development prospects. *Int J Mol Vet Res*. 2023;13(2):1-10. doi:10.5376/ijmvr.2023.13.0002
79. Wang ZL, Lin XF. Overcoming feline calicivirus: modern treatment methods and

comprehensive management strategies. *Int J Mol Vet Res*. 2024;14(1):1–8. doi:10.5376/ijmvr.2024.14.0001

80. Wißmann JE, Kirchhoff L, Brüggemann Y, Todt D, Steinmann J, Steinmann E. Persistence of pathogens on inanimate surfaces: a narrative review. *Microorganisms*. 2021;9(2):343. doi:10.3390/microorganisms9020343
81. Wei Y, Zeng Q, Gou H, Bao S. Update on feline calicivirus: viral evolution, pathogenesis, epidemiology, prevention and control. *Front Microbiol*. 2024;15:1388420. doi:10.3389/fmicb.2024.1388420
82. Whitehead K, McCue KA. Virucidal efficacy of disinfectant actives against feline calicivirus, a surrogate for norovirus, in a short contact time. *Am J Infect Control*. 2010;38(1):26–30. doi:10.1016/j.ajic.2009.03.015
83. WSAVA Vaccination Guidelines Group (VGG). 2024 Guidelines for the vaccination of dogs and cats. World Small Animal Veterinary Association (WSAVA). 2024. Available from: https://wsava.org/wp-content/uploads/2024/05/2024-Guidelines-for-the-Vaccination-of-Dogs-and-Cats.pdf
84. Wu H, Huang J, Liu Y, Pan Y, Li Y, Miao Q, Qu L, Tian J. Feline calicivirus proteinase-polymerase protein degrades mRNAs to inhibit host gene expression. *J Virol*. 2021;95(13):e0033621. doi:10.1128/JVI.00336-21
85. Wu H, Zhang X, Liu C, Liu D, Liu J, Tian J, Qu L. Antiviral effect of lithium chloride on feline calicivirus in vitro. *Arch Virol*. 2015;160(12):2935-43. doi:10.1007/s00705-015-2534-8
86. Yang D, Ju H, Li X, Shen H, Ge F, Yang X, Zhao H, Wu X, Zhu X, Wang X, et al. Epidemiological surveillance of respiratory diseases in urban stray cats in Shanghai. *Animals*. 2024;14(11):1562. doi:10.3390/ani14111562
87. Yumiketa Y, Narita T, Inoue Y, Sato G, Kamitani W, Oka T, Katayama K, Sakaguchi T, Tohya Y. Nonstructural protein p39 of feline calicivirus suppresses host innate immune response by preventing IRF-3 activation. *Vet Microbiol*. 2016;185:62-7. doi:10.1016/j.vetmic.2016.01.015
88. Zhou L, Fu N, Ding L, Li Y, Huang J, Sha X, Zhou Q, Song X, Zhang B. Molecular characterization and cross-reactivity of feline calicivirus circulating in southwestern China. *Viruses*. 2021;13(9):1812. doi:10.3390/v13091812
89. Zheng M, Li Z, Fu X, Lv Q, Yang Y, Shi F. Prevalence of feline calicivirus and the distribution of serum neutralizing antibody against isolate strains in cats of Hangzhou, China. *J Vet Sci*. 2021;22(5):e73. doi:10.4142/jvs.2021.22.e73
90. Zhang Y, Cheng Z, Guo C, Yang T, Zhao W, Qian J, Xia X, Bi J, Zhou D, Xu S, Li Z, Zhu Y, Zhang H, Tan Y, Bi Z. Discovery of a novel genogroup feline calicivirus through molecular evolution in group-housed cats in China. *Sci Rep*. 2025;15(1):19059. doi:10.1038/s41598-025-03585-5
91. Zicola A, Saegerman C, Quatpers D, Viandier J, Thiry E. Feline herpesvirus 1 and feline calicivirus infections in a heterogeneous cat population of a rescue shelter. *J Feline Med Surg*. 2009;11(12):1023-7. doi:10.1016/j.jfms.2009.05.023