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Feline Calicivirus: Emerging Perspectives on Pathogenesis and Disease Management

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Abstract

The Feline Calicivirus (FCV) is an extremely contagious virus that can produce numerous clinical signs in cats. In most cases, FCV presents as mild upper respiratory disease along with oral ulcers. But some of the more virulent strains can cause severe systemic disease, resulting in a high level of morbidity and a high incidence of mortality after contracting the disease. Its high mutation rate and genetic diversity complicate diagnosis, treatment, and vaccine efficacy. This study synthesizes current findings on the epidemiology, molecular characteristics, and pathogenic mechanisms of FCV, with emphasis on regional strain variation and its implications for vaccine development. Phylogenetic analyses reveal the emergence of multiple genogroups and novel variants, some displaying poor cross-neutralization, underscoring the limitations of existing vaccines. Recent investigations highlight the role of both structural and nonstructural viral proteins in immune evasion, viral replication, and host interactions. Advances in understanding FCV evolution and receptor usage, such as the identification of feline junctional adhesion molecule-1 (JAM-1) as a key receptor, provide new directions for antiviral strategies. Collectively, these insights emphasize the urgent need for broad-spectrum vaccines and integrated control measures to mitigate FCV prevalence and disease burden in feline populations worldwide.

Key Words: Respiratory disease, structural and nonstructural protein, Feline junctional adhesion molecule-1, vaccine efficacy.

Virology and Molecular characterization of FCV

This small, virus does not have a lipid envelope and is composed of 7.7 kb of RNA that is non-circular, single stranded, positive-sense, and codes for three different proteins using three open reading frames (ORFs). Additionally, both the 3' and 5' ends of the genome have a poly-A sequence. While ORF3 encodes for a protein that has structural components, ORF2 is responsible for producing VP1 (the main component of the capsid), whereas ORF1 produces proteins that are not part of the structure, including the virus's own protease and RNA-dependent RNA polymerase. Each of the six regions (A through F) within the capsid protein are located in variable and highly conserved areas, with B, D, and F being quite similar between viruses, and C and E being very different. However, B-cell epitopes found in the major component of the virus (Region E) can be useful in distinguishing between stains and for molecular typing [44].

The FCV capsid is mainly made up of VP1 proteins that have icosahedral symmetry. High-resolution cryo-electron microscopy revealed that upon binding of a receptor to VP1, a conformational change in VP1 is associated, which leads to the exposure of regions of the protein that are crucial for cell entry or infection. Consequently, the flexibility of the VP1 conformation enables FCV to exhibit antigenic variation because of the conformational change of VP1 during the life cycle of the virus [14]. VP2 is an important auxiliary component of the capsid because of the formation of a channel-like structure, also known as a portal, which enables the entry of the viral RNA genome into the cytoplasm of the host cell, where it initiates replication of the virus [12,13].

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The attachment of FCV to receptors on the surface of feline cells results in infection (fJAM-A). The amino acids of fJAM-A are conserved and provide a point of attachment for the virus's VP1, allowing it to enter the cell. Once in the cell, the virus is unable to infect it right away. In order for the virus to be able to infect the cellular cytoplasm, it must first cross the membranes of endosomal compartments. The viral protein VP2 is responsible for providing a means for the viral RNA genome to escape from the endosomal compartments into the cytoplasm of the infected cell and begin the process of replicating in the cytoplasm of the infected cell [12].

After the virus is inside the cytoplasm of the infected cell, FCV replicates using its non-structural proteins that have assembled into replication complexes, which ultimately produce two forms of RNA: full-length (genomic) and shorter (subgenomic). Recent studies have shown that correct processing of the virus's ORF1 protein (which is a polyprotein) is essential for the efficient production of infective progeny virus and efficient viral replication. Disruption of the viral replication-related proteins will have a very negative effect on the synthesis of viral RNA and serves to illustrate the important function that these proteins serve in the FCV life cycle [20].

Molecular analysis of FCV reveals extensive genetic variation across strains. While the virus mutates throughout its genome, changes most often pile up in the capsid gene - the one responsible for building the VP1 protein. Because of this instability, new versions of the virus emerge frequently, differing enough to be recognized as separate antigenic types. These shifts matter - they've been tied to reduced protection from existing vaccines. Not just immunity takes a hit; some of these altered forms link closely to severe, body-wide infections. As the surface proteins change shape, they don't only dodge immune responses - such modifications can shift which animals get infected and how sick they become [2,14].

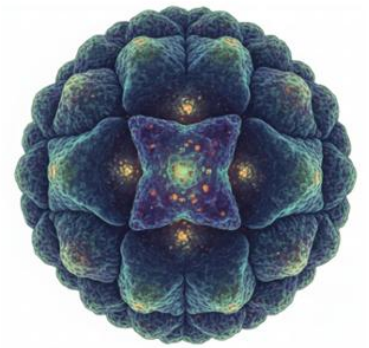

Fig.1 Electron cryo-micrograph of an isolated calicivirus from a cat, displaying its icosahedral symmetry top down and the dimeric arrangement of the capsomeres that extends out to form the cup-like shape

Viral Structure and Genome

Recent improvements in structural (emphasizing structural detailing) virology, mainly due to advances in cryo-electron microscopy have greatly improved our knowledge with respect to feline calicivirus (FCV) molecular organization. Recent studies have determined that there are 180 equal copies of the VP1 capsid protein in each FCV particle, and that the VP1 protein has been shown to exhibit 3 quasi-equivalent conformations (A, B and C) that assist in creating the icosahedral symmetry of the virus. The assembly of the various subunits consists of AB dimers assembled at pentameric vertices, and CC dimers assembled along the two-fold axes of the icosahedron, producing a well-ordered Capsid that helps provide capsid stability in order to facilitate the assembly and infectivity of the virus. This quaternary organization is functionally relevant, as it mediates asymmetrical conformational rearrangements during receptor engagement. Specifically, AB dimers at the fivefold axes undergo an approximately 15° anticlockwise rotation, whereas CC dimers at the twofold axes tilt outward from the particle surface through movements within their protruding (P) domains [14]. Localized structural flexibility within VP1 permits receptor interaction without compromising overall capsid stability.

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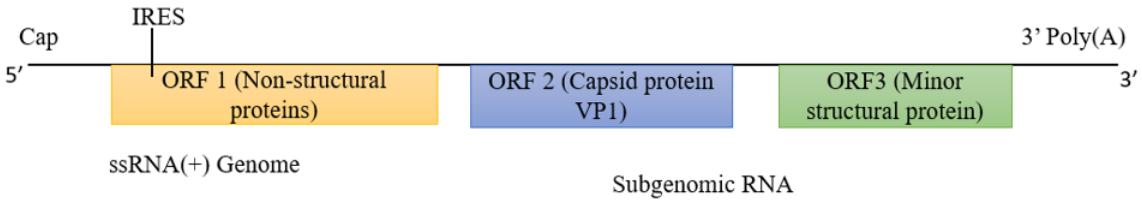

Fig 2. A linear schematic of the ssRNA genome of calicivirus to show the ORF structure along the linear ssRNA genome, with ORF1 being the non-structural ORF, and ORFs 2 and 3 specifying capsid protein VP1 (ORF2) and other low abundance structural proteins, respectively. The ORF structures are located at the 5' end of the ssRNA linear genome, while the ORFs 3 shares the 3' end of the ssRNA linear genome.

The P domain of VP1, which confers antigenic specificity and receptor-binding capacity, is subdivided into proximal (P1) and distal (P2) regions. The P2 subdomain forms the distal tips of the spike-like projections extending from the virion surface. Atomic modeling indicates that receptor-binding interfaces are concentrated within hypervariable loop regions of P2, which constitute the structural stem of these spikes. This configuration provides a mechanistic explanation for strain-specific tropism and the rapid emergence of antigenic variants through immune-driven selection [11,14]. Beneath the protruding P domain, the shell (S) domain forms the continuous structural scaffold underlying the capsid surface.

Feline calicivirus (FCV) uses an uncoating mechanism that permits effective transfer of viral genomic RNA to a host cell's cytoplasm. FCV binds its cellular receptor, feline junctional adhesion molecule-A (fJAM-A), initiating conformational changes in the viral capsid, which exposes minor capsid protein VP2 on the surface of the virus and rearranges major capsid protein VP1.These coordinated conformational changes generate an opening in the capsid that allows the FCV to interact with and permeate the endosomal membrane. The FCV's penetration of the endosomal membrane releases the viral RNA genome into the cytoplasm of the host cell, thereby facilitating replication of the viral genome [26].

Another mechanism by which FCV optimizes its replication is by regulating the transcription of subgenomic RNA in a manner that separates the synthesis of early non-structural proteins and late structural proteins in time. VP1 is subject to proteolytic cleavage by the FCV 3C-like protease during subgenomic transcription, which exposes its N-terminal region and allows it to interact with host molecular chaperone Hsp90 [21]. The regular need of FCV for both viral proteolysis and host proteostatic machinery demonstrates the complex molecular dependency between FCV and the host cell, as well as the complex regulatory framework governing calicivirus infections.

FCV demonstrates significant dynamics at the virion level as seen by using integrative structural- and molecular-based methodologies. The FCV particle does demonstrate T = 3 icosahedral symmetry, but it maintains a high degree of conformational flexibility. A specialized "portal-like" assembly is proposed to facilitate efficient translocation of genomic RNA during virion construction. Engagement with the appropriate receptor during the process of host entry triggers substantial capsid rearrangements, enabling the release of the viral genome into the cytoplasm and altering the antigenic architecture of the capsid via the altered exposure of surface epitopes.

Pathogenesis:

The pathogenesis associated with FCV is influenced by numerous viral and host factors. The replication of FCV within a host cell causes direct cellular injury (cytopathic) and strain-specific virulence factors aid in evasion of the host immune system and in directing virulence factors toward specific target tissues. Additionally, dysregulated host inflammatory cascades during the acute stages of FCV infection also contribute to tissue injury; thus, FCV virulence depends upon a delicate balance of viral pathogenic mechanisms and host immune modulation.

I: Cell Preference and Early Replication of FCV

The main target for FCV is predominantly epithelial cells in the upper respiratory tract and

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lining of the mouth after entering through the nose or mouth into the body. After invading a host cell, the virus takes its RNA genome and uses it as a "template" for creating replicate viral particles through production in the cytoplasm. This process is called "replication," and the replicating viral RNA creates "non-structural proteins" through a process known as translation. FCV also produces a "negative-strand intermediate" during replication that is used to generate new copies of its genome. Electron microscopy studies have shown that FCV can be found in the tonsils as well as in the lining of oral and respiratory epithelial cells, and in some cases even within the endothelial cells of small blood vessels. This evidence demonstrates that FCV has the ability to spread from its site of infection (i.e., the mouth and/or upper respiratory tract) and circulate throughout the entire body (i.e., beyond its site of infection) [21].

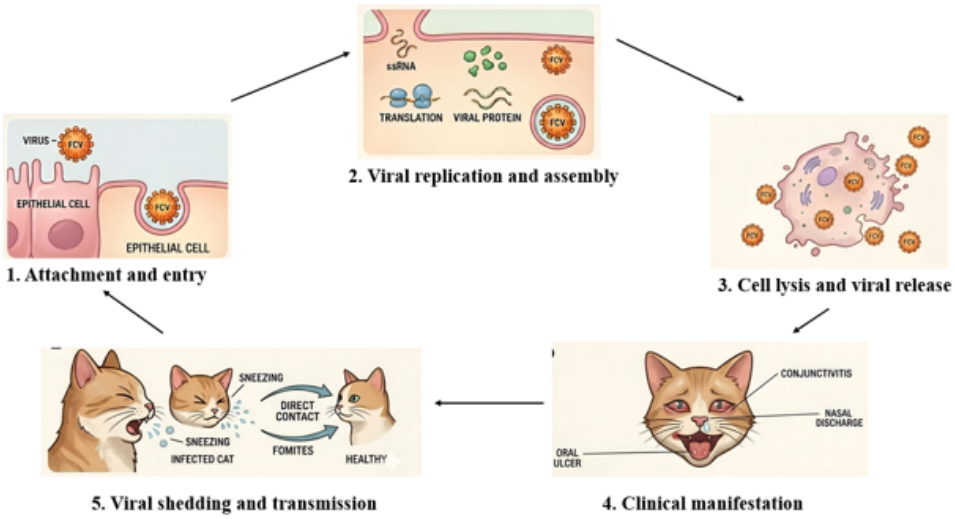

Fig 3. FCV Infection Dynamics: This is an illustration demonstrating the stages of an FCV virus infecting a host cell (Attachment, replication, lysis) taken along with the clinical signs of infection that may be seen throughout the whole cat. The illustrations show how the FCV virus is able to shed from the respiratory secretions and mouth sores that develop, so it has constant access to a cat population.

II. Virulent Systemic Disease (VSD) Lesions

Acute viral disease caused by FCV causes more severe clinical signs than classic FCV infections. In addition, the virulent strain of FCV has a greater ability than the classic strain to infect multiple cell types than the classic strain of infection within a given tissue. In one Australian study, the virulent strain was found to be able to penetrate into multiple types of cells within the same tissue, thus allowing extensive viral replication within those multiple cells, thereby resulting in significantly greater viral loads in that tissue than found in cases of infection with the classic stain of FCV. Once the viral load becomes very high, the virus can then enter the host's circulation, where it can travel to all parts of the body and infect multiple organs. Conditions that impair the host's ability to fight infection, including pre-existing medical conditions, will also make it easier for the virus to become Systemically spread throughout the body. The clinical signs associated with acute virulent FCV are quite severe and may include: skin ulcers, swelling or edema of the skin, jaundice, and multi-organ dysfunction. The clinical manifestations associated with Systemically spread acute virulent FCV are the results of substantial replication of the virus in the affected tissues and destruction of the endothelial cells of blood vessels leading to bleeding within the blood vessels [26].

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III. Innate Immune Modulation and Autophagy

Feline calicivirus (FCV) has been found to strategically alter the host's immune response by activating the cellular autophagy pathway in a manner that ultimately benefits its own replication. A considerable amount of research demonstrates that FCV infection actively stimulates autophagy in infected cells, as evidenced by the elevated accumulation of LC3-II and an increased presence of double-membrane autophagosomes. Multiple non-structural proteins (NSPs) produced from the FCV open reading frame-1 (ORF1), especially p30, p48, and most importantly p39, have been recognized as strong inducers of the autophagic response. Among these, p39 has a particularly significant functional role, as its ability to trigger autophagy directly contributes to enhanced viral replication; experimental activation of autophagy results in greater viral output, while blocking early autophagy-associated protective proteins markedly reduces viral production [7]. In addition to facilitating replication, FCV leverages the autophagy pathway to suppress innate antiviral defenses within infected cells. The p39 protein specifically interacts with the autophagy adaptor p62, promoting the selective degradation of RIG-I, a cytosolic RNA receptor responsible for sensing viral presence, which in turn diminishes downstream signaling pathways involved in type I interferon synthesis [7]. Together, these findings are consistent with and reinforce earlier research on FCV–host interactions, indicating that autophagy represents a central immune evasion mechanism used by FCV to create a cellular environment favorable for replication while simultaneously escaping host immune detection [16].

IV.Autophagy-Dependent Targeting of RIG-I by FCV P39

RIG-I is an important antiviral receptor against numerous types of viral infection; however, after an infection is established RIG-I is specifically targeted for degradation. Viral protein (p39) mediates RIG-I's degradation by promoting the interaction between RIG-I and the autophagy adaptor (p62) in order to direct RIG-I into the autophagic pathway for degradation. The ejection of RIG-I from the system prevents RIG-I from activating transcription of type I interferon gene, IFN-β. Since type I interferon would inhibit viral replication, the continual replication of viral RNA occurs even after viral RNA has been initially detected by pattern recognition receptors (PRR) in the newly infected host.

V.Additional Immune-Evasion Mechanisms

The viral PP (proteinase-polymerase) contributes another mechanism for disruption of host shutoff functions through assisting in the dismantling of the host's cellular mRNA and by the FCV protein p30, which decreases mRNA expression of interferon alpha/beta receptor subunit 1 (IFNAR1) thus reducing the efficiency of interferon signalling. These combined mechanisms allow FCV to have a considerable advantage over the host in replication rate compared to when the host has an inappropriately activated and functioning adaptive immune response.

VI.Integrated Model of Disease

Evidence suggests that this model describes the ability of FCV to establish high levels of reproduction in the mucosal tissue. The FCV has developed methods to escape from the innate immune system of its host. For example, it is able to induce the autophagic destruction of RIG-I, which is a critical antiviral molecule in the host. In addition, FCV has the ability to inhibit the expression of various anti-viral genes in the host. The result of these two events disrupts the normal early signalling pathways of the antiviral response of the host, thus providing a better environment for the virus to replicate. Some FCV genotypes exhibit characteristics of being highly virulent and/or invasive due to their ability to infect vascular endothelial cells. Infection of vascular endothelial cells by FCV results in a dysregulated release of cytokines, extensive damage to blood vessels and systemic inflammation. These pathogenic processes are associated with the severe clinical signs and high mortality rates seen in VSD outbreaks.

Clinical Disease

Feline calicivirus is known to cause a very wide range of clinical signs and symptoms. For example, it can result in mild cases of upper respiratory disease, or lead to a severe, rapidly progressive systemic disease, based on factors intrinsic to the host. Host factors including the amount of genetic variation present within the feline calicivirus, as well as host characteristics such as the host's age, immunocompetence, etc. will all affect the severity and virulence of disease [1].

Approximately 2–6 days after infected cats are exposed, the virus begins replicating in the tissues of the oropharynx and nose, resulting in the abrupt development of various clinical signs: fever; serous to mucopurulent nasal discharge; conjunctivitis; vesicular and/or ulcerative lesions on

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the tongue and hard palate. The virus replicates within the epithelial cells, resulting in the lysis of those cells, followed by the development of ulcers. The development of these ulcers creates a microbial environment that allows for the secondary colonization by bacteria and May, in and of itself, cause additional damage to the underlying mucosal surface.

Although most FIIV infections resolve without treatment, some strains of FIIV have been shown by molecular testing to possess the ability to persist in the oral cavity and oropharynx, shedding viral particles over extended periods of time, and therefore, potentially lead to long-term infection. This long-term viral persistence is influenced by the extensive genetic diversity of FIIV strains in circulation (e.g., differences in the capsid region) and the capsid's ability to bind to different receptors, as well as by their ability to infect various types of tissues within the host. In addition, continuous replication of FIIV in the oral cavity will ultimately lead to unabated antigenic stimulation, which is believed to be a major contributor to the development of chronic immune-mediated inflammatory disorders, such as lymphoplasmacytic gingivostomatitis [17,19].

If you are a cat owner, you may know that your feline friend can develop a lameness syndrome associated with feline calicivirus (FCV) infections. Limping syndrome has a sudden onset with fever and multiple joint dysfunction occurring within a few days of having fever, nasal discharge, and excessive drooling (sometimes called "kissing" due to the large volume of saliva produced). A cat suffering from limping syndrome may also be reluctant to walk, have a short- or long-leg lameness, and show signs of discomfort in their joints. In most cases, there has been no evidence that the condition is caused by bacterial infection, and limping syndrome often resolves on its own without any specific treatment. The self-limiting nature of limping syndrome along with the fact that there is no evidence of bacterial arthritis, suggests that the joint lesions associated with limping syndrome are primarily due to immune-mediated or inflammatory mechanisms as a result of FCV infection [22].

There are multiple variations of Feline calicivirus (FCV), with the virulent systemic disease (VSD) variation of FCV being considered the most lethal to cats. The virulent form is extremely aggressive, resulting in a high fever and visible swelling of the face and legs. It causes systemic effects on the body causing serious clinical signs making it a serious and potentially fatal disease., skin lesions, jaundice, difficulty breathing, multiple organs affected, and very high mortality. While most classical strains of FCV cause respiratory disease only, VSD strains infect a wider variety of tissues, cause much higher viral loads in multiple organs, and cause viremia (presence of the virus in the bloodstream), leading to dissemination via the blood. Disease progression and final outcome depend on host factors, especially the type and degree of immune response. Therefore not all infected cats will die from VSD, even during outbreaks, indicating that VSD pathogenesis has multiple causal factors. Given the rapid progression of VSD, the effect on multiple body systems, and potentially high mortality, VSD is regarded as the most aggressive and advanced form of FCV infection [26].

High mortality rate (greater than 50% in cats infected) demonstrates increased virulence in strains compared to previously known strains, and indicates that vaccinated cats are at a greater risk than previously thought for dying from virus infection.

Laboratory abnormalities from most acute infections are relatively mild; they typically consist of short-lived fluctuations in the number of white blood cells, small elevations in liver enzyme levels, and somewhat less common laboratory abnormality findings; in contrast, laboratory abnormalities in cats with a diagnosis of viral systemic disease (VSD) are greatly increased compared with the degree of abnormalities seen in most acute infections (i.e. lymphopenia, thrombocytopenia, hyperbilirubinemia) and they generally show significant evidence of extensive vascular and tissue damage from the virus and the resulting viral disease [1]. Typically, the diagnosis of a cat with a viral disease will be made by PCR identification or viral culture from an oral swab; however, confirmation of VSD is concluded upon the use of immunohistochemistry on affected tissues. This virus may survive for a period of weeks in the environment, and many adult cats without signs of clinical disease (asymptomatic carriers) are common in the feline population and serve as virus shedders for the continued transmission of the virus within the feline population.

Feline calicivirus (FCV) is a viral agent that can result in either localized mucosal disease or more severe systemic disease (less common), affecting multiple organs at once. Most infected cats exhibit mild illness with clinical signs localised to either the respiratory tract and/or oral cavity. However, there are a few strains of FCV that have

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been shown to have very aggressive systemic disease and result in widespread organ dysfunction and high rate of death in affected cats. Very importantly, because FCV can persist in carrier kitties after recovery and because there is significant genetic and antigenic diversity in the currently circulated strains of FCV, there are major obstacles to controlling the disease and the result is that FCV causes repeated outbreaks in the feline population [18,22].

Epidemiology

Feline calicivirus (FCV) is pervasive in both wild and domestic cats globally and has been shown to vary significantly in the frequency of infection by geography [1]. Approximately 25-50% of cats tested will have evidence on serological testing of past exposure to FCV (e.g., the presence of antibodies in their blood), but seroprevalence tends to be much higher in environments with large numbers of cats in close contact, such as shelters, catteries, and breeding facilities when compared to homes with a single cat. There are clear regional differences in seroprevalence rates, with high seroprevalence rates seen in Milan (85.4% of stray cats) and very low seroprevalence rates seen in South Korea (0.0-0.3%) and Japan (1.1%). The same geographic disparity of seroprevalence is observed in China, where the Shandong Province has a seroprevalence rate of 70.7%, whereas neighbouring regions such as Guangxi and Beijing have seroprevalence rates of 23-44% [5]. Phylogenetic analysis of viral sequences from Shandong indicates the circulation of multiple lineages of virulent FCV, demonstrating the significant genetic diversity within the FCV population, and that the epidemiology of FCV is heterogeneous and strongly impacted by environmental and regional factors.

The epidemiology of feline calicivirus (FCV) involves having a still unknown number of chronic carrier cats that look well but continue to shed (pass) and pass the virus on to other cats. Infected cats can be infectious for a long period, both before and after showing signs of being ill, and so are continually reinfecting the next susceptible cat that comes into contact with them. This is a major concern in high-density animals in shelters and catteries due to the close grouping of cats and the number of cats moving through foster homes and facilities, providing an ideal environment for continued transmission of the FCV to other cats [26]

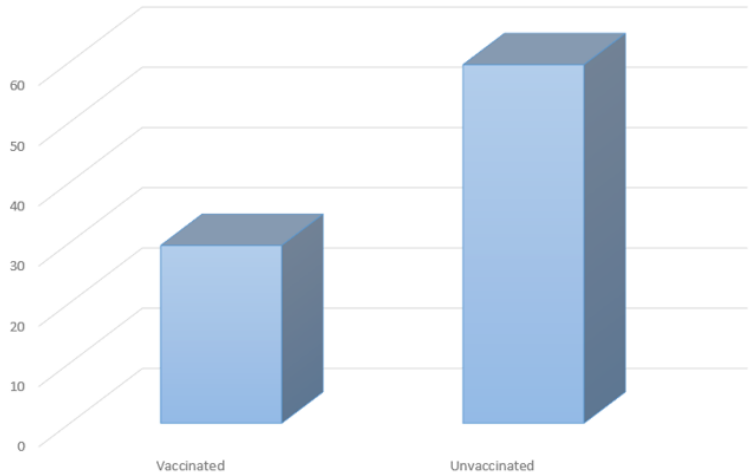

Fig 4. Comparison of vaccinated and unvaccinated FCV infection rates in various studies [7,28,34,42,46]

Researchers look at the differences between strains of a virus in order to estimate how easily a person may have become infected with that virus. The FCV exhibits a high degree of genetic variability compared to other viruses and this variability is largely due to the way in which FCV replicates, which results in the generation of new strains at regular intervals. At least three major genogroups (GI, GII, and GIII) of FCV have been identified through molecular epidemiological

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analysis, and new variations continue to be generated through recombination events [3]. The evolution of virulent systemic strains (VS-FCV), which have a mortality rate of more than 50% due to severe multisystemic infections, indicates the rapid evolution of the virus and its ability to affect the disease burden [4,6].

A number of risk factors have been shown to be important in the spread and maintenance of FCV in feline populations. High-density housing conditions, such as shelters and catteries, have been shown to have a significantly higher infection rate than individually housed domestic cats [3]. The age of the animal is also an important factor, as kittens under 12 weeks of age are highly susceptible after the maternal-derived antibody levels have waned. Stress-related conditions, such as overcrowding and co-infection with other pathogens, such as feline herpesvirus-1, have also been shown to increase susceptibility and exacerbate the disease. Carrier status, which has been shown to exist in up to one-third of infected cats, has been shown to play a role in the maintenance of the disease within the population [1].

In addition, there has been evidence of seasonal variation in the incidence of FCV infections, with increased rates of infection being reported during the winter and spring months, which could be related to increased co-infections and environmental stress during these periods [5].

Transmission and Stability of Feline Calicivirus (FCV):

Feline calicivirus (FCV) is a highly contagious viral infection that spreads from one infected cat to another mainly through bodily secretions such as blood and saliva. Close physical interactions between a healthy and infected cat during the acute phase of the disease are thought to be the most common and effective method of FCV transmission. FCV may also be found in urine, feces, and blood (i.e., non-oral and non-respiratory secretions) when an infected cat is shedding FCV. The presence of FCV in urine and feces is particularly significant because both body fluids are readily available in high-density cat populations such as catteries or when several cats share an environment [37,45].Although most infected cats will completely recover without any lasting complications and remove all traces of FCV after the clinical signs resolve, a small subset of cats may continue to shed FCV after the signs have resolved. Some of these cats shed FCV continuously, whereas others shed FCV occasionally. The continued presence of FCV carriers creates an ongoing source of FCV infection and contributes to its continued presence in the cat population [37].

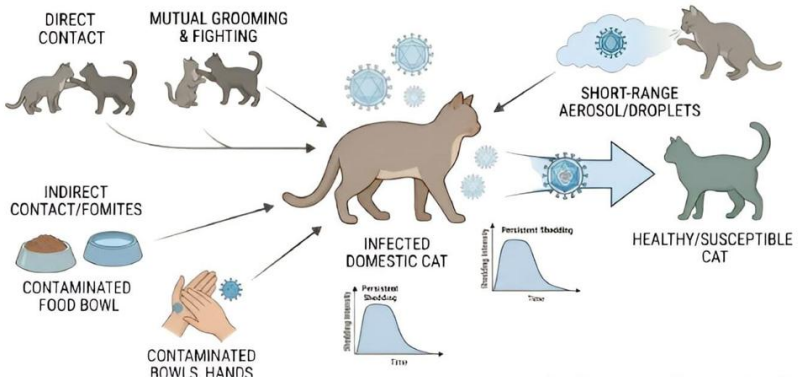

Fig 5. The various methods that Feline calicivirus (FCV) can be transmitted in include: via direct touch (e.g., during grooming or fighting), short-distance air travel, and indirectly through contact with contaminated objects (e.g., food containers or hands). When an exposed cat comes upon secretions, or an object that is infected with the virus, they may get infected with FCV.

Feline calicivirus (FCV) can be transmitted without actual contact. This can lead to FCV continuing to be passed around and persisting in domestic cat populations. FCV is able to contaminate many different types of surfaces and objects, like food and water bowls, litter trays, cages and bedding. Environmental contamination of surfaces or objects means that FCV can be passed in other ways than just from cat to cat directly; therefore, it increases the chance to be exposed to FCV while in shared environments. Furthermore, humans also can transmit FCV to other cats through

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their hands, clothes or equipment. This can occur if many cats are handled or moved regularly, thus, increasing the possibility of cross-contaminating [37,45]. FCV is also capable of being transmitted through short-distance aerosols. An infected cat with mouth ulcers or upper respiratory disease can aerosolize FCV into the environment if they produce droplets that are spread in the air. The aerosolization of FCV may pose a greater risk to nearby susceptible cats. However, aerosolization as a means of disease transmission is significantly greater in crowded environments because of the close proximity of infected to uninfected cats, thus, increasing the possibility of inhalation exposure. There are also other potential routes of FCV transmission, such as through insects (e.g., fleas), but this is poorly defined and considered inconsequential compared to the transmission routes of direct contact between cats or environmental contamination [45].

Feline calicivirus (FCV) is a highly resilient virus that is capable of remaining viable for extended periods, both in the environment as well as in cat populations, and is a significant factor in the epidemiology of FCV due to its long environmental persistence and widespread distribution among cats. As a non-enveloped virus, FCV has a high resistance to desiccation, allowing FCV to maintain infectious particles for long periods on dry surfaces. Experimental studies and field observations have shown that under various ambient conditions, FCV may survive anywhere from multiple days to multiple weeks post-infection. In favourable environmental conditions (e.g., low ambient temperature and high humidity), the virus has been demonstrated to survive and remain viable for up to 30 days after an infected cat has shed the virus, particularly in the absence of effective disinfection [39,45].

The ability of FCV to survive for extended periods in the environment, increases the likelihood that a susceptible cat may be exposed to infectious FCV material from contaminated surfaces long after the infected cat has been removed from the environment. Thus, environmental persistence of FCV is critical in maintaining endemic cycles of infection and presents significant challenges for the implementation of effective infection control and biosecurity practices in affected environments.

Fortuitously, Feline Calicivirus (FCV) is highly resistant to many common disinfectants and many commercial disinfectants have little or no virucidal activity against FCV. The decreased susceptibility of the virus to being inactivated by chemicals has much to do with the structural design of the viral capsid, which is highly stable and structurally sound. The stable configuration of the capsid associated with FCV allows this virus to survive environmental stressors, and to remain outside of a host for much longer than most viruses [25,39].The continued presence of FCV in the cat population and the continued spread of FCV infection stems from many interrelated epidemiological factors: the ability of FCV to be shed for long periods of time from infected cats, the establishment of cats with persistent or intermittent carrier states, the ability to transmit FCV indirectly by contaminated fomites, and the ability of FCV to remain viable for extended periods of time in the environment. Taken together, the effects of these factors allow for the continued spread of FCV; even in the presence of routine hygiene and routine vaccination. Therefore, prevention and control of FCV infection in cats pose a serious challenge because of the influence of the above factors [23,37].

Diagnosis and Treatment

Reverse Transcription - Polymerase Chain Reaction (RTPCR) analysis from oropharyngeal or conjunctival swabs is the most commonly used method of achieving molecular confirmation of feline calicivirus (FCV) during acute infection, and is considered the most common sensitive routine diagnostic procedure for detecting FCV in clinical diagnostic practice. However, RT-PCR does not differentiate between residual live RNA and live (replication-competent) virus which can produce infectious progeny. When determination of the presence of live viral progeny (for example, during a suspected outbreak or to determine whether or not a suspected virulent strain of virus) is needed, virus isolation is used to allow for phenotypic and antigenic determination of the virus. In order to identify the strain, and epidemiological tracking of the strain of virus, a variety of molecular methods are used, including partial or whole genome sequencing of the virus. By amplification of the VP1 region of the virus present in the virus capsid and/or use of tiling amplicons, a phylogenetic comparison of the various circulating strains of virus and virulent FCV (VS-FCV) is able to be conducted. The combination of RT-PCR, virus isolation, and sequence-based typing is important in increasing the overall diagnostic efficiency and improving epidemiological resolution to provide better support

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for outbreak investigation and improve strain characterization [31].

Overall, the management of feline calicivirus (FCV) infection is based around evidence-based supportive care in both routine clinical settings and in supplemented documented case reports. No antiviral therapy has been universally endorsed, so the aim of management is to minimize the clinical signs while maintaining systemic stability. Analgesic therapy can be provided to treat the pain associated with oral ulcerations and therefore improve the voluntary intake of food. There may also be a requirement for nutritional support through assisted feeding or the administration of appetite stimulants in the event of anorexia. Patients will have their hydration maintained through oral fluid supplementation, or in cases where clinically indicated, via subcutaneous or intravenous fluid therapy. In patients that are having significant respiratory compromise, supplemental oxygen and additional forms of respiratory support will be given as necessary. Antimicrobials should only be prescribed when there is a confirmed or strong suspicion of secondary bacterial infection, and they are not recommended for routine prophylactic use. During outbreak situations, additional supportive measures as per the clinical guidelines would include nebulization or humidification, to assist with clearing airways, and topical ophthalmic medications for conjunctival or corneal involvement. In multicat households or hospital environments, strict implementation of infection control protocols is critical for containment. Such measures would normally include isolation of the infected cats from other cats and rigorous cleaning and disinfection of the hospital environment using virucidal agents proven effective against non-enveloped viruses as well as the strict control of staff and animal movement in an effort to minimize the spread of the disease.Because of how effectively the virus survives on surfaces for long periods of time, as well as how quickly it can spread between contaminated surfaces when biosecurity procedures are not enforced adequately, these steps are critical [1].

Attempts to find an antiviral agent that directly affects feline calicivirus (FCV) have shown some promise, although the results of in vitro research into several compounds (including nucleoside analogs, e.g., NITD008, and other small-molecule inhibitors) have not been consistent. In general, while many compounds show the ability to inhibit the replicative cycle of FCV in Feline cell lines, their effectiveness against FCV varies by viral strain tested and by the modulating ability of the compound on host immune systems, including cytokine production. A significant challenge in evaluating antiviral agents against FCV in culture systems is that they exhibit significant cytotoxicity at concentrations close to their antiviral activity, limiting their therapeutic potential. It is therefore difficult to define a safe, effective therapeutic window. In vitro studies have confirmed that some of these compounds can inhibit viral RNA synthesis; however, no compound has yet progressed into clinical use. Ongoing translational challenges, along with the extraordinary genotypic and phenotypic variability exhibited by the currently circulating strains of FCV, pose additional complications for drug development. Consequently, the treatment of FCV infection continues to rely heavily on supportive care and administering compounds that act as immunomodulators. In cases of FCV-associated stomatitis that are either chronic or resistant to treatment with immunomodulatory agents, the use of recombinant. Currently, no nucleoside analog—including NITD008—or any other direct-acting antiviral agent is considered suitable or approved for routine clinical use in the management of feline calicivirus infection [33].

Prevention and Control

1. Effectiveness of Vaccination and Antigenic Variation

An assessment of feline calicivirus (FCV) infections in 516 clinically ill cats conducted in Hangzhou, China implicated an FCV rate of 43% of the feline population sampled. Phylogenetic evaluation of viruses detected in the samples available indicated considerable genetic diversity among circulating strains. Furthermore, virus neutralization assays performed on samples from vaccinated and unvaccinated cats revealed marked differences in FCV antibody responses. Collectively, these data suggest that currently available vaccines may provide only limited coverage against FCV infection due in part to antigenic diversity among circulating viral strains [34]. In a separate study of multi-cat environments, investigators reported differing patterns of FCV shedding among cats. Although some cats were observed to be persistent shedders of FCV, continuously shedding the virus for an extended period of time, other cats shed the virus intermittently, while some cats did not shed detectable virus. In addition to these different shedding patterns, an analysis of laboratory samples

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from multiple cats demonstrated that multiple FCV strains were in circulation at the same time in the same population of cats. Collectively, these findings illustrate the epidemiological complexity of FCV infections and highlight the need for comprehensive control measures. Effective methods to control FCV transmission must consider factors such as identification and monitoring of carrier cats, reduction of persisting shedders, and strict hygiene practices to minimize environmental contamination and transmission of FCV [35].

2. Effects of Modified Live Virus (MLV) Vaccination on Outcome Measures that are Clinically Important.

Vaccination with modified live viruses (MLV) can significantly decrease the severity of symptoms experienced by a cat after exposure to non-matching field strains of feline calicivirus (FCV). In addition to having milder clinical signs and less time in viremia associated with FCV infection (i.e., the state of being infected by and shedding virus) than cats that are not vaccinated, vaccinated cats will have lower amounts of viral RNA (genetic material from the virus) present at the time of infection. While vaccination does not prevent infection, it helps to reduce the severity of clinical disease due to vaccination and decreases the amount of virus shed following infection. Thus, the MLV vaccines can play an important role in controlling the impact of FCV on exposed cats [36].

3) Carrier State and Dynamics of Viral Shedding

After a cat has been infected by FCV, it is possible they will develop into a "carrier" state, where, although they have recovered from the clinical signs of their initial illness, they continue to shed the virus either continuously or intermittently. Those cats with a persistent FCV infection are reservoirs for the virus, creating continuing exposure(s) to susceptible cats, and representing a perpetual source of recirculating FCV within a multi-cat population unit. The genetic variability of FCV and the ongoing evolution of the viral population(s) within carrier cats further compound the difficulties associated with eradicating the virus, and, therefore, creating population-level control of FCV is particularly difficult [27,8]. Both vaccinated and unvaccinated cats may establish the carrier state and may also be able to shed some field strains of FCV that are capable of evading the vaccine-induced viral antibodies. The implications of these points is that there is a great need to identify and manage all carrier cats as part of any complete FCV control strategy. Combining the management of carrier cats with administration of vaccines and implementation of stringent environmental sanitation practices should decrease the transmission of the virus and limit the long-term effects of FCV in feline populations [37].

4.Environmental Stability of Feline Calicivirus and Efficacy of Surface Disinfection

FCV can survive in the environment for long periods of time. In an experiment where fecal contamination occurred on inanimate surfaces from infected cats, infectious FCV was recovered for 28 days after the last fecal sample was collected. The presence of viral RNA on surfaces does not ensure infectious virus exists. After effective disinfectants such as (5% sodium bicarbonate) or commercially available products such as (Incidin Plus) have been used, infectious virus recovery from contaminated surfaces is not possible after the surfaces are disinfected. Alternatively, surfaces that have been cleaned without being effectively disinfected may still show the presence of residual FCV RNA, even though the viral isolate(s) may no longer be viable [38]. Research evaluating the susceptibility of FCV to various disinfectants has demonstrated highly variable results for individual viral isolates. There are also several commonly used disinfectants such as quaternary ammonium compounds, alcohols, and hypochlorite bleach that do not inactivate all viral isolates equally. The efficacy of disinfectants relates to three primary factors: the choice of disinfectant, the concentration of disinfectant, and the duration of contact time with the viral isolate. The above demonstrates the importance of selecting the appropriate disinfectant and using the proper procedures to reduce environmental contamination and minimize risk for indirect transmission of FCV in multi-cat environments and/or in the veterinary clinical practice [39].

5.There has been some investigation into non-traditional chemical disinfectants that have been tested as alternatives to conventional disinfectants for disinfecting surfaces contaminated with feline calicivirus (FCV).

Recently, a study, titled “Exploring Non-Alcohol Based Disinfectants,” was done on arginine and zinc chloride ($ZnCl_2$) at a pH of approximately 11 to see whether this combination of disinfectants would effectively inactivate FCV. It showed that after just

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five minutes of being in contact with the arginine/$ZnCl_2$ combination, the FCV was reduced to nearly zero log units (i.e., when FCV was present at an average concentration greater than 100,000 per gram or per milliliter, it would be reduced to less than 1 per gram or per milliliter). This indicates that non-traditional chemical disinfectants (e.g., arginine/$ZnCl_2$ combination) might provide additional options for environment cleaning and for reducing the risk of FCV spread through indirect routes, in addition to current conventional disinfection methods [40].

Genetic Diversity and Evolution

1. Genomic Distribution, Presence, And Evolution Of Novel FCV Strains From Emerging Novel Geographical Regions.

Recent studies have confirmed the existence of genetically diverse, potentially virulent, as well as genetically distinct strains of feline calicivirus (FCV), particularly in terms of the VP1 capsid gene's sequence and the range and number of nonclinical/clinical strains present, from 2018-2022 within China and other parts of the world. The collection of 152 cat fecal specimens from a location within DaLing yielded approximately 29% FCV positive specimens in total (i.e., 152 total samples; approx. 29% FCV positive); eleven total isolate sequences were generated and showed that seven isolates belonged to genogroup G1. Of G2, four belonged to the G2 gene pool. One isolate from the cohort was identified as a recombinant of previously reported Chinese isolates and therefore represents continued evolution of the virus in this region. The presence of the predominant G2 strain in DaLing may also be due to the geographical area in addition to the ongoing transmission of the established strains of the virus, as both the established strains and established sequences of the G1 and G2 gene pools co-exist in this area of China [41].

The same analysis of the VP1 sequence of 52 FCV positive pooled cat fecal samples from Kunshan showed that both G1 and G2 strains were present; again G2 strain predominated with 52 total specimens being FCV positive. Comparison of the variations between these two geographical locations indicates the presence of multiple amino acid differences at the site of the hypervariable region E of VP1; many of which were previously described to be associated with virulence. Therefore, the findings demonstrate a high degree of genetic divergence from the reference vaccine strain; indicating that there are continuing challenges to controlling FCV disease, as novel genogroups continue to develop through on-going antigenic diversity and differences in genotype and phenotype across multiple geographical regions [42].

2.Virulence-Associated Amino Acids, Gene Recombination and Natural Selection

In a study entitled "Phylogenetic and Isolation Analyses of Several FCV Isolates from China" (2019-2021), FCV was isolated from 26 individuals in a phylogenetic study from G-I, G-II. Analysis of the capsid protein confirms that evolutionary change has occurred through natural selection. For example, the HB7 strain has 3 unique amino acid residues that are in common (homologous) with those of VSD (virulent systemic disease) strains, suggesting that the HB7 strain may have evolved either by convergent evolution or through recombination with a VSD strain (or both). Overall, the results of this study provide support for the theory that evolutionary pressures, as well as molecular changes, may allow FCV strains to develop enhanced virulence over time, illustrating how the evolutionary forces and molecular changes can interact to influence the emergence of virulent FCV strains with increased pathogenicity [40].

3.The temporal and spatial structure of populations of feline calicivirus (FCV) through phylogenetic study has revealed an extraordinarily high degree of diversity among co-circulating viruses in addition to the genetic variation present within each virus.

For example, data collected on FCV in the UK demonstrated the existence of strain diversity among multiple strains of FCV that were circulating at the same time but could not be identified as having a dominant strain (>5%) nationally or on a regional level (>14%). Interestingly, while 11 distinct strains of FCV were sampled, some of these strains appeared to have restricted geographic ranges thus indicating patterns of localized circulation of viruses [41].

A recent study conducted in Switzerland analyzed 66 FCV isolates, nearly covering the entire VP1 gene,

and classified strains based on approximately 20% genetic distance within the hypervariable E region. From this analysis, 52 distinct strains were identified, many of which were represented by a single individual cat. Interestingly, the phylogenetic relationships of the strains were strongly associated

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with cat breed or pedigree status, whereas no correlation was observed between geographic distance and genetic relatedness, suggesting that viral evolution is influenced more by host factors than by spatial separation [41].

Further investigations into quasispecies dynamics revealed that temporally separated FCV isolates did not form distinct clusters in phylogenetic analyses. In other words, strains collected at different time points exhibited genetic distances similar to those observed within populations sampled simultaneously. This indicates a continual turnover of viral populations and ongoing mutation, rather than the persistence of older viral lineages over time [27].

The mutation rates reported for FCV, particularly from isolates collected from 3–10-year-old FIV-infected cats, rank among the highest described for RNA viruses. Both intra-host and inter-host substitution rates are remarkably high, demonstrating the rapid evolution of FCV and underscoring the challenges this presents for long-term viral control, vaccine efficacy, and epidemiological management [1].

Conclusion

FCV is a significant viral pathogen for domestic cats worldwide, with a high degree of genetic variation and high mutation rate. FCV is a single-stranded RNA virus that has structural features that cause ongoing evolution. The emergence of new strains of FCV continue to occur with different levels of pathogenicity, as well as different modes of infection and transmission among cats. Understanding the structure of FCV, how it infects cats, how it spreads, and how it is transmitted will help to explain the continued persistence of FCV in domestic cat populations. All four of these factors will affect the level of disease; the susceptibility of a cat to develop an FCV infection; and the overall transmission dynamics of FCV among cats.

Vaccines remain the primary method for preventing and controlling diseases but genetic diversity and antigenic diversity of FCV could make existing vaccines ineffective in preventing viruses circulating in real time. Constantly monitoring the evolution of viruses will be critical for expanding on the advancements currently occurring to enhance and develop vaccines and develop management strategies for a disease. Increasing prevention of FCV, monitoring the evolution of FCV viruses and enhancing ways to provide better immunity from vaccines will aid markedly in improving the health of cats.

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