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Feline Calicivirus (FCV): From Molecular Characteristics to Vaccine Development Prospects

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Abstract Feline calicivirus (FCV) is a significant pathogen affecting the health of feline animals, characterized by a high degree of genetic variability, which complicates vaccine prevention and control strategies. This study provides a comprehensive review of FCV's molecular characteristics, epidemiology, infection mechanisms, host immune responses, and the current state of vaccine development. It systematically analyzes the structure and genomic composition of FCV, reveals key molecular mechanisms in the virus replication cycle, and discusses the impact of virus variability and genetic polymorphism on vaccine design. Additionally, this study reviews the modes of FCV transmission, clinical symptoms, diagnostic methods, and the host's immune response to FCV. Furthermore, in light of the limitations of existing vaccines, this research explores the prospects of emerging vaccine technologies, such as genetically engineered vaccines, virus-like particle (VLP) vaccines, DNA vaccines, and mRNA vaccines. It further identifies the challenges and research directions in the future development of FCV vaccines, underscores the importance of an integrated epidemic control strategy, and proposes key points for future research.

Keywords Feline calicivirus (FCV); Molecular characteristics; Immune response; Vaccine development; Virus variation

Feline calicivirus (FCV), a member of the Caliciviridae family, is a non-enveloped, single-stranded positive-sense RNA virus. It infects cats worldwide, causing a broad spectrum of diseases ranging from mild upper respiratory tract infections to fatal systemic illnesses (Gui, 2023). FCV infection can lead to clinical symptoms such as oral ulcers, rhinitis, and conjunctivitis (Gao et al., 2022). In severe cases, it can also result in pneumonia, skin lesions, and rare systemic diseases. One of the distinctive features of FCV is its rapid mutation rate, allowing the virus to adapt to immune pressure and vaccine selection, significantly complicating treatment and control efforts. Moreover, FCV can persist within the host even after symptoms subside, potentially turning cats into asymptomatic carriers, further increasing the risk of virus transmission.

Research on FCV holds great significance in both veterinary medicine and public health. On one hand, it directly impacts the health of pet cats, particularly in communal cat environments, where disease outbreaks pose serious health and welfare concerns. On the other hand, the high variability and prevalence of FCV make it an ideal model for studying virus evolution, host adaptability, and vaccine design. Existing vaccines, while partially alleviating clinical symptoms, cannot completely prevent virus transmission and mutation, necessitating in-depth investigation into the biological characteristics of FCV for the development of more effective vaccines (Guo et al., 2022). Furthermore, research on FCV contributes to understanding other Caliciviridae family viruses, such as noroviruses, which is of significant importance for human public health. Thus, comprehensive studies on FCV not only have direct practical applications in the management of cat health but also hold profound theoretical implications for a wide range of biomedical research fields.

This study aims to comprehensively review the molecular characteristics, infection mechanisms, and immune responses of FCV. It will also provide a detailed assessment of current and emerging vaccine strategies. Through the analysis of existing literature, we hope to offer a reference framework for researchers, veterinarians, and cat caretakers regarding FCV control strategies, while also guiding future research directions. Ultimately, by advancing FCV vaccine development, we aspire to reduce the impact of this virus on cats and their owners.

1 Molecular Characteristics of FCV

1.1 Virus structure and genome composition

Feline calicivirus (FCV) has a relatively simple genome structure, consisting of approximately 7.7 kb of single-stranded positive-sense RNA. The genome's end carries non-coding regions that play a regulatory role in the virus's replication and protein synthesis (Urban and Luttermann, 2020). FCV's genome primarily includes three open reading frames (ORFs). ORF1 encodes non-structural proteins involved in virus replication and processing. ORF2 and ORF3 encode the major structural protein VP1 and the minor structural protein VP2, respectively. VP1 is the primary protein on the virus particle's surface, directly participating in virus attachment and host cell recognition, while VP2 plays a role in virus assembly and release. The RNA genome and protein shell of FCV form the non-enveloped virus particle together, with a diameter of approximately 30-40 nm, featuring characteristic cup-shaped depressions on the particle surface (Figure 1).

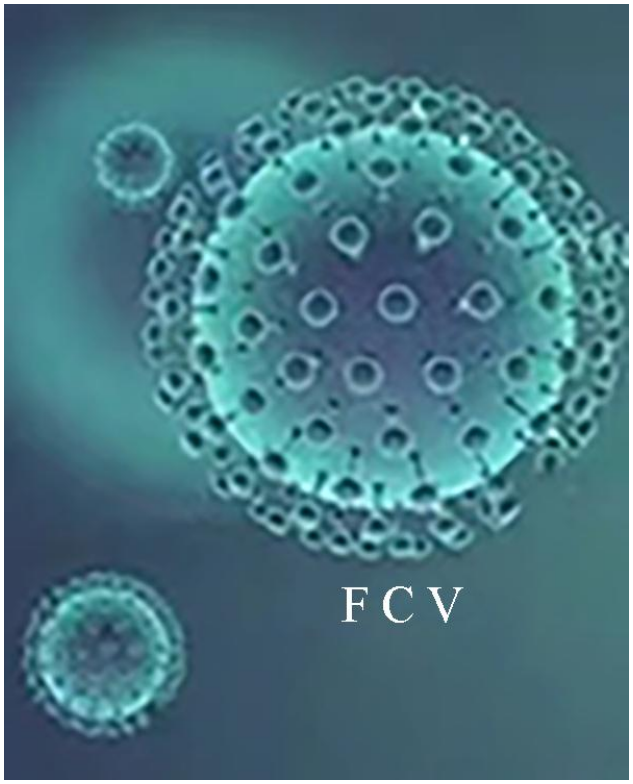

Figure 1 Feline calicivirus

1.2 Molecular mechanisms of the virus replication cycle

The replication cycle of FCV begins with the binding of the virus to the surface receptor of the host cell, a process primarily mediated by the capsid protein VP1. Once inside the cell, the viral RNA genome is released into the cytoplasm and undergoes direct translation on the host cell's ribosomes, producing viral non-structural proteins. These non-structural proteins, including replication enzymes and other necessary enzymes, collectively facilitate the replication of the virus RNA genome within the host cell. The replicated genome serves as the genetic material for new virus particles and, simultaneously, as mRNA for the continued translation of more viral proteins. After the synthesis of new virus RNA and proteins, virus particles assemble maturely in the cytoplasm and are released extracellularly through changes in the cell membrane, completing one replication cycle.

1.3 Virus variability and genetic polymorphism

The variability of FCV is a central issue in its molecular characteristics. Due to the relatively high error rate of RNA-dependent RNA polymerase (RdRp) during the replication process and the lack of a proofreading mechanism, FCV exhibits a high mutation rate. This high mutation rate allows FCV to rapidly adapt to environmental pressures, such as host immune responses and vaccine pressures. Virus variability not only affects its antigenicity, leading to immune escape, but may also influence the virus's pathogenicity and transmission

capability. The presence of genetic polymorphism implies that multiple variants of FCV can be detected at different geographic locations and time points. This polymorphism poses a challenge for vaccine design and disease management strategies because a single vaccine may struggle to provide comprehensive protection. Therefore, studying FCV variability and polymorphism is crucial for understanding how the virus evades the host immune system and for designing vaccines capable of addressing various variant strains.

2 Epidemiology and Transmission of FCV

2.1 Global epidemiological situation of FCV

Feline calicivirus (FCV) is a widely distributed virus that affects almost all regions with cat populations worldwide. Due to its high genetic diversity, different viral strains can be detected globally. Epidemiological studies indicate significant variations in FCV infection rates among different cat populations, ranging from pet cats to stray cats and those in shelters. The prevalence and transmission speed of FCV increase significantly, particularly in communal environments such as cat breeding facilities, shelters, and pet stores. This widespread infection not only poses a significant threat to the health of feline populations but also places higher demands on veterinary public health management.

2.2 Transmission routes and host range

FCV primarily spreads through viral particles in saliva, eye and nasal secretions, with close contact being the main transmission route (Radford et al., 2021). The virus can also be indirectly transmitted through shared items like food and water bowls, cat litter, and even human hands and clothing. Since the virus can survive in the environment for several weeks, cats without direct contact may still become infected with FCV. Additionally, mother cats can vertically transmit the virus to their offspring. FCV's host range is relatively narrow, mainly limited to felines, but certain strains may have infectivity to other animals. However, there is currently no evidence indicating FCV can infect humans.

2.3 Factors influencing outbreaks and transmission

Outbreaks of FCV are often associated with high cat density, low vaccination rates, the virulence of viral strains, and the immune status of the host. In densely populated urban areas, the high mobility of cats and increased contact with wildlife elevate the risk of FCV transmission. While vaccination can control outbreaks to some extent, the diversity of viral strains and limitations in vaccine coverage mean that vaccinated cats may still become carriers and spreaders of the virus (Figure 2). Furthermore, environmental factors such as hygiene conditions, stress levels, and nutritional status within cat populations significantly affect FCV transmission. Therefore, effective control of FCV requires a comprehensive consideration of the interactions between host, environment, and pathogen, along with the implementation of appropriate preventive and control measures.

Figure 2 illustrates the infection, transmission, and recovery process of Feline calicivirus (FCV). Different stages of cat images represent various states of virus infection, and arrows indicate possible pathways of virus transmission.

Part A depicts the process of acute infection. Cats that have just been infected show clinical symptoms and viral shedding. Over time, even if clinical symptoms disappear, cats may still be in a state of viral shedding, but the quantity decreases. Approximately 30 days later, some cats may transition to an apparently healthy state but continue to shed the virus.

Part B portrays the scenario of chronic infection. Some clinically seemingly healthy cats may be persistent shedders of FCV, meaning they show no symptoms but continue to shed the same strain of the virus. Additionally, some cats may become healthy carriers due to continuous infection with different strains or other viral variants.

Part C represents cats recovering health through immune intervention (e.g., vaccination). After vaccination, these cats clear the virus, cease viral shedding, and thus recover health.

In summary, this process diagram clearly illustrates the complexity of FCV infection and demonstrates the different possibilities from acute infection to potential chronic carrier states, and finally to recovery through

immune intervention. The diagram also emphasizes the importance of managing FCV transmission within cat populations, as cats may become carriers and spreaders of the virus even without obvious symptoms. Therefore, regular vaccination and monitoring are crucial for controlling the spread of FCV.

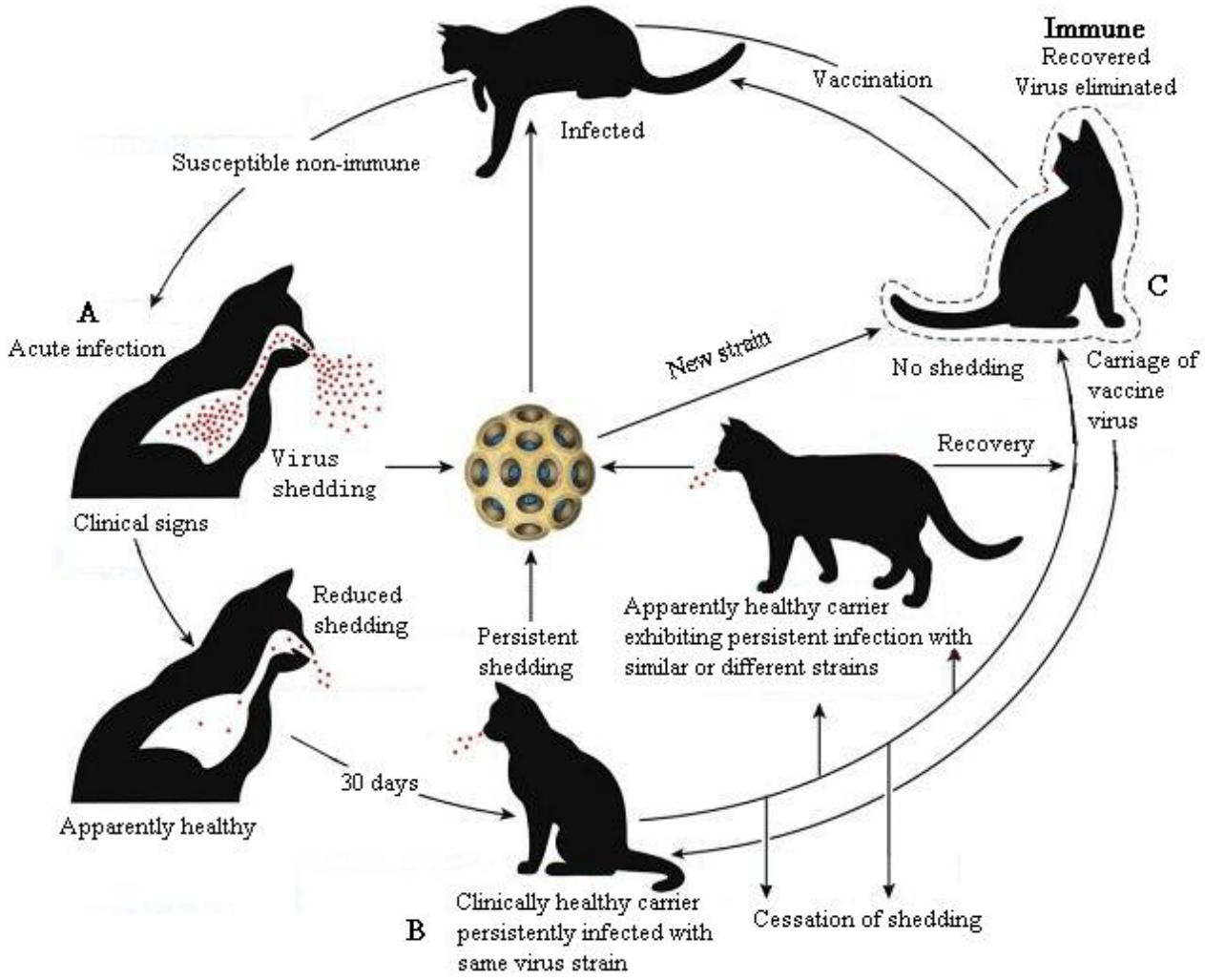

Figure 2 Infection, transmission and recovery process of feline calicivirus

3 Mechanisms of FCV Infection

3.1 Host cell recognition and virus entry

The recognition and entry of Feline calicivirus (FCV) into its host cells involve a precise molecular process. FCV utilizes its surface VP1 protein to bind to receptors on the surface of host cells, a process akin to a key entering a lock. For FCV, these receptors typically consist of complex glycan structures on the surface of cat cells. Once bound to the receptor, FCV enters the host cell through endocytosis. The virus is enveloped in vesicles formed by the cell membrane. After entering the cell, the acidic internal environment prompts the vesicle to release the virus into the cytoplasm. At this point, the virus's RNA genome is released and prepared for the next replication step.

3.2 Virus replication and assembly

After the virus RNA enters the host cell, it initiates the synthesis of viral proteins using the host's translation machinery (Li et al., 2022). FCV's RNA genome directly serves as messenger RNA (mRNA) in protein synthesis, with the initial synthesis being non-structural proteins, including key enzyme proteins like RNA-dependent RNA polymerase (RdRp). These non-structural proteins collaborate to form the replication complex, initiating the virus RNA replication process. As new virus RNA and structural proteins are produced, new virus particles begin assembling inside the cell. The assembly process mainly occurs in the cytoplasm, where newly synthesized virus RNA is enveloped in newly formed protein shells, forming immature virus particles.

3.3 Strategies for viruses to evade the host immune system

To persist in the host and continue spreading, FCV has evolved multiple mechanisms to evade the host's immune system. Firstly, the virus can change its surface antigens through frequent mutations, evading the neutralizing action of host antibodies. Secondly, FCV can interfere with the host's antiviral response, such as inhibiting the production and function of interferons, critical factors in the host's early immune response. Thirdly, FCV can directly damage the host's immune cells, such as phagocytes and T cells, reducing the effectiveness of the immune system. Additionally, FCV forms persistent infections within the host, sometimes even shedding the virus without symptoms. Such covert infections make it challenging for the immune system to completely clear the virus. These mechanisms collectively make FCV a complex and challenging pathogen, requiring a deep understanding of its interactions with the host immune system for effective control.

4 Host Immune Response to FCV

4.1 Innate immune response

When a cat is infected with feline calicivirus (FCV), the first line of defense in its immune system, the innate immune response, is rapidly activated. The innate immune system recognizes viral components, such as viral RNA, through general patterns, initiating a series of immune responses. This includes the activation of natural killer (NK) cells, which can directly kill virus-infected cells, and the production of interferons by host cells. Interferons are antiviral proteins that inhibit the replication and spread of the virus. Additionally, phagocytic cells like macrophages and dendritic cells engulf virus particles, presenting viral antigens to prepare for subsequent adaptive immune responses.

4.2 Adaptive immune response

Building upon the innate immune response, the adaptive immune response provides a more specific defense mechanism. B cells recognize and respond to specific antigens of FCV, differentiating into plasma cells that produce antibodies against FCV. These antibodies can bind to virus particles, preventing them from entering host cells and tagging them for recognition and clearance by phagocytic cells. Simultaneously, T cells are activated, especially cytotoxic T cells, which can recognize and kill host cells infected with FCV, limiting the spread of the virus.

4.3 Immunological memory and immune evasion

Once the host immune system undergoes the initial attack by FCV, it forms immunological memory against the virus. Memory B cells and memory T cells persist in the host long-term after the initial infection. When encountering the same or similar FCV antigens again, these memory cells can rapidly proliferate and initiate a quicker, more effective immune response. However, FCV's immune evasion mechanisms (Figure 3), particularly the high variability of the virus's surface proteins, allow it to evade recognition by host antibodies. This means that even in the presence of immune memory, certain variants of FCV can still cause reinfection (Zheng, 2022). Therefore, even immunized cats may not be immune to all FCV strains, posing challenges for vaccine design and immunization strategies. Researchers are actively working to understand how to stimulate broad protective immune responses through vaccination and how to enhance immune memory to combat the diversity and evasion strategies of the virus.

Figure 3 illustrates how Feline calicivirus (FCV) affects the host cell's immune response through immune evasion mechanisms. The figure describes the interferon signaling pathway, a crucial pathway for cell response to virus infection.

Firstly, the cell recognizes the presence of the virus, leading to the production and release of Type I interferons (IFNs). These interferons bind to specific receptors on the cell surface (such as IFNAR1 and IFNAR2). When Type I interferons bind to these receptors, they activate two key enzymes inside the receptor: TYK-2 and JAK-1.

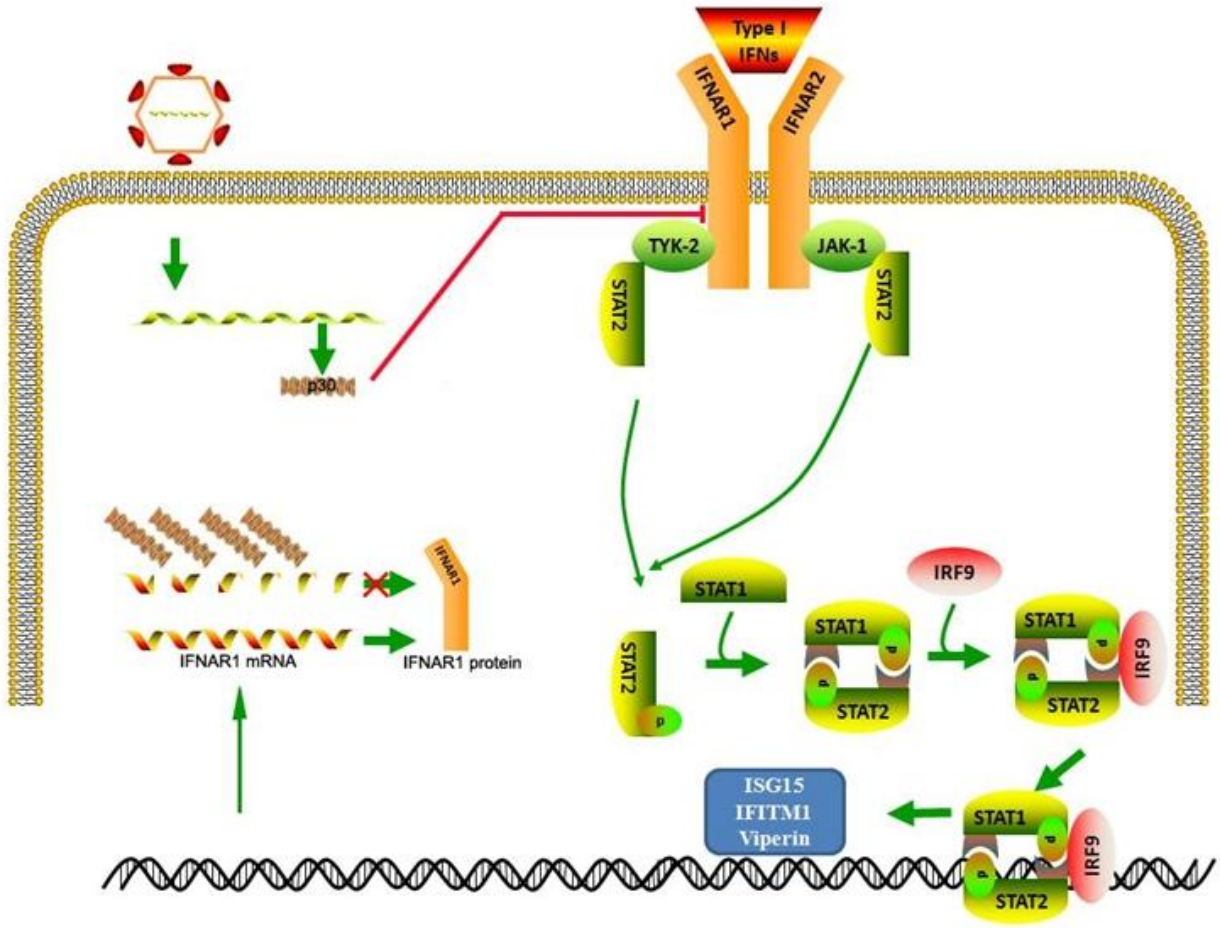

Figure 3 FCV immune escape mechanism (Tian et al., 2020)

Activation of these enzymes results in the phosphorylation of STAT1 and STAT2 proteins. Phosphorylated STAT1 and STAT2 then bind to the IRF9 protein, forming the ISGF3 complex. This complex subsequently enters the cell nucleus, promoting the expression of antiviral genes, such as ISG15, IFITM1, and Viperin. Proteins produced by these antiviral genes can inhibit virus replication and spread.

However, FCV may interfere with the above signaling pathway through certain mechanisms, reducing the expression of antiviral genes. The figure indicates potential interference points of FCV with red arrows. For example, the virus may inhibit the expression of the IFNAR1 protein or directly block a part of the JAK-STAT signaling pathway, weakening the host's immune response and achieving immune evasion.

This mechanism explains how FCV can persist in the host and may lead to chronic infection. Understanding this process is crucial for developing vaccines and treatment strategies against FCV.

5 FCV-Induced Clinical Symptoms and Pathology

5.1 Typical clinical presentations

The clinical manifestations of Feline calicivirus (FCV) infection can vary from mild to severe. Typical symptoms include fever, oral ulcers, acute rhinitis, conjunctivitis, and respiratory symptoms such as sneezing and nasal discharge (Hofmann-Lehmann et al., 2022). In some cases, FCV may also lead to lameness and arthritis. In the most severe cases, FCV can cause Viral-Induced Feline Pneumonia (VFP), a rare but often fatal systemic disease characterized by high fever, severe respiratory distress, and multi-organ failure.

5.2 Pathological changes and tissue damage

Pathological changes induced by FCV primarily occur in the oral cavity, upper respiratory tract, and lungs. In the oral cavity, FCV can cause inflammation and ulcers of the oral mucosa, especially on the tongue and soft palate

(Figure 4). Symptoms in the upper respiratory tract include mucosal inflammation of the nasal cavity and pharynx, which may lead to epithelial damage and secondary bacterial infections. In cases of FCV-induced VFP, the lungs exhibit interstitial pneumonia, characterized by thickening of the alveolar walls and infiltration of mononuclear cells, potentially leading to respiratory failure.

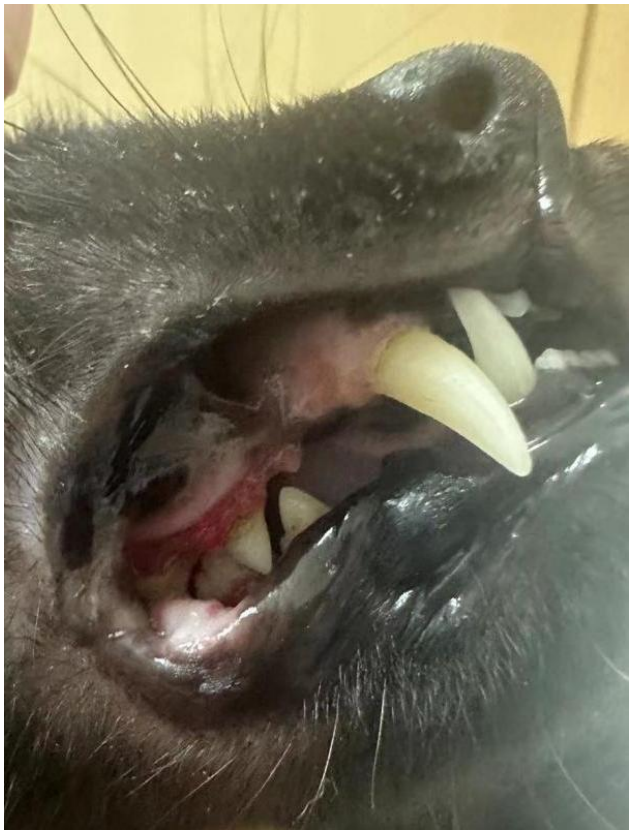

Figure 4 Pathological manifestations of FCV (Source: this hospital)

5.3 Diagnostic methods and laboratory testing

The diagnosis of FCV typically relies on the identification of clinical symptoms and results from laboratory testing. Laboratory diagnostic methods include virus isolation, PCR (Polymerase chain reaction) techniques for detecting viral RNA, and serological methods such as ELISA (Enzyme-linked immunosorbent assay) to detect specific antibodies. Virus isolation is a traditional method that requires the use of cell culture systems, while PCR technology provides a faster and more sensitive detection method. Serological testing helps determine whether the host has mounted an immune response and its immune status. Additionally, molecular techniques, such as sequence analysis, are used to identify specific genotypes of FCV strains and monitor virus variations. Through these diagnostic tools, veterinarians can assess the severity of the disease, guide treatment plans, and provide information for epidemic control.

6 Development of FCV Vaccines

6.1 Early vaccine development and use

The development of FCV vaccines began with a deep understanding of the virus's structure and biological characteristics. Early vaccines often utilized inactivated or attenuated live virus, which were administered to cats to elicit an immune response (Tizard, 2021). Inactivated vaccines involve killing virus particles with chemicals or heat while preserving their antigenic properties, while attenuated live vaccines weaken the virus's pathogenicity through continuous passages in non-target host cells. These early vaccines contributed to reducing the incidence of FCV and played a positive role in disease prevention.

6.2 Efficacy and limitations of current vaccines

Despite the contributions of early vaccines to FCV control, the efficacy of existing vaccines faces several challenges. Firstly, due to the high variability of FCV strains, current vaccines cannot provide comprehensive protection, especially against newly emerging variants. Additionally, vaccines may not completely prevent the shedding and transmission of the virus, leading to vaccinated cats still potentially becoming carriers. Furthermore, prolonged reliance on the same vaccine may result in a singular immune response, reducing the defense capability against diverse virus strains. Therefore, there is still room for improvement in the breadth and duration of immune protection induced by existing vaccines.

6.3 Vaccination strategies and immunization programs

Effective vaccination strategies are crucial for the successful implementation of FCV vaccines. To maximize vaccine effectiveness, veterinarians and cat caretakers need to follow scientifically-based immunization programs. This typically involves vaccinating kittens early (usually starting at 6-8 weeks of age) and providing necessary booster shots to ensure a long-lasting immune response to FCV. Additionally, regular booster vaccinations can help maintain immune levels, especially in high-risk environments. Vaccination schedules should be personalized based on the prevalence of the virus, the health status of the cats, and potential exposure risks. With a deeper understanding of FCV immune mechanisms, new vaccine technologies, such as nucleic acid vaccines or protein-engineered subunit vaccines, are expected to enhance vaccine effectiveness and applicability.

7 Emerging Vaccine Technologies and Prospects

7.1 Genetic engineering vaccines

With the advancement of molecular biology techniques, genetic engineering vaccines have become a frontier in FCV vaccine development. These vaccines are typically designed to include only one or a few key antigenic protein genes of the virus, without the viral genetic material that causes disease. These genes are inserted into a safe viral or bacterial vector, allowing the expression of FCV antigenic proteins in the host, thereby eliciting an immune response. Genetic engineering vaccines have advantages such as high safety, strong specificity of immune response, and the potential to target multiple strains of FCV through design.

7.2 Virus-like particles (VLPs) and recombinant protein vaccines

Virus-like particles (VLPs) are an innovative vaccine strategy composed of the viral protein shell without the viral genetic material, making them incapable of replication or causing disease. VLPs can mimic the structure of authentic viruses, eliciting a robust immune response (Lu et al., 2018). Recombinant protein vaccines use genetic engineering techniques to produce antigenic proteins of FCV in bacteria or yeast. After purification, these proteins are used as vaccines, directly stimulating the host's antibody response to FCV. The development of VLPs and recombinant protein vaccines provides a safer and more controlled option for FCV prevention.

7.3 Development progress of DNA vaccines and mRNA vaccines

DNA vaccines and mRNA vaccines have become hot topics in vaccine development in recent years. DNA vaccines involve directly injecting DNA containing genes encoding FCV antigens into the host, prompting host cells to produce FCV antigenic proteins and activate an immune response. mRNA vaccines, on the other hand, deliver mRNA molecules containing sequences encoding FCV antigens, utilizing the host's cellular machinery for translation and expression of antigenic proteins. Both types of vaccines can be rapidly designed and produced, especially demonstrating advantages in addressing the rapid mutation of the virus. As research deepens and technologies mature, DNA and mRNA vaccines are expected to be effective tools against FCV and other pathogens.

As vaccine technologies continue to advance, future vaccines against FCV may become more efficient, broad-spectrum, and conducive to large-scale production. The development of these emerging technologies brings new hope for the control and prevention of FCV and other infectious diseases. Researchers must continue to explore the immune escape mechanisms of FCV and host immune responses to lay the scientific foundation for designing the next generation of vaccines.

8 Conclusion

One of the biggest challenges in the development of Feline calicivirus (FCV) vaccines is the high variability of the virus. The genetic diversity of FCV leads to the production of various antigenic variants, making it difficult for existing vaccines to provide comprehensive protection. Additionally, vaccine-induced immune responses may gradually weaken over time, requiring periodic booster shots (Bergmann et al., 2019). Formulating effective vaccine administration strategies to cover cats of different ages, health conditions, and living environments is also a current challenge in vaccine development. Furthermore, factors such as vaccine safety, cost-effectiveness, and ease of administration must be considered when promoting vaccine use.

For FCV, an integrated virus control strategy is particularly important in addition to vaccination. This includes regular monitoring of the infection status of cat populations, improving living environments, reducing contact between cats, and implementing effective isolation measures to control virus spread. Developing broad-spectrum vaccines and increasing vaccination rates are crucial components of an integrated strategy. Additionally, public education and professional veterinary training are key elements in improving the effectiveness of virus control.

Future research on FCV needs to advance on multiple fronts simultaneously. Firstly, in-depth studies on the molecular biology and immune escape mechanisms of FCV will provide more targets for vaccine design. Secondly, the development of new vaccine technologies, such as mRNA vaccines or VLPs vaccines, may provide stronger immune protection and better cross-protection. Thirdly, studying the dynamics of feline population immunity and the immune network will guide effective population immunity strategies. Lastly, interdisciplinary collaboration, including the integration of molecular biology, immunology, epidemiology, and veterinary clinical practice, will contribute to a comprehensive understanding of FCV and the formulation of more effective control measures. Through these comprehensive research efforts, researchers can expect greater progress in the prevention and control of FCV in the future.

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References

Bergmann M., Speck S., Rieger A., Truyen U., and Hartmann K., 2019, Antibody response to feline calicivirus vaccination in healthy adult cats, Viruses, 11(8): 702.
https://doi.org/10.3390/v11080702
PMid:31370359 PMCid:PMC6723298

Gao C.X., Chen B.J., Mi J., Han J.J., Lian S.C., Ma W.R., 2022, Epidemiological investigation and analysis of feline calicivirus in Xi'an, Chinese Journal of Veterinary Medicine, 58(3): 101-105.

Guo Y.B., Liu H.K., Wang Q, Yi S.S., Niu J.T., Li D.L., Cui Z.D., Wang K., Shao H.Z., and Hu G.X., 2022, Assessment of the protective efficacy of a feline calicivirus inactivated vaccine using in vivo FCV CH-JL2 infection, Pak. Vet. J., 42(3): 328-333.

Gui Y.P., Liu J., Bai Y.L., Wang J., and Zhao H.J., 2023, Research progress on the characteristics of virulent systemic feline calicivirus, Chinese Journal of Virology, 39(4): 1093-1098.

Hofmann-Lehmann R., Hosie M.J., Hartmann K., Egberink H., Truyen U., Tasker S., Belák S., Boucraut-Baralon C., Frymus T., Lloret A., Marsilio F., Pennisi M.G., Addie D.D., Lutz H., Thiry E., Radford A.D., and Möstl K., 2022, Calicivirus infection in cats, Viruses, 14(5): 937.
https://doi.org/10.3390/v14050937
PMid:35632680 PMCid:PMC9145992

Li S., Zang M.H., and Zhou Z.L., 2022, iTRAQ-based exploration of differentially expressed proteins in host cells infected with different virulence of feline caliciviruses, Chinese Journal of Preventive Veterinary Medicine, 44(12): 1261-1268.

Lu Z., Ledgerwood E.D., Hinchman M.M., Dick R., and Parker J.S., 2018, Conserved surface residues on the feline calicivirus capsid are essential for interaction with its receptor feline junctional adhesion molecule A (fJAM-A), J. Virol., 92(8): 10-1128.
https://doi.org/10.1128/JVI.00035-18
PMid:29386293 PMCid:PMC5874407

Radford A., Afonso M., and Sykes J.E., 2021, Feline calicivirus infections, In: Sykes J.E. (ed.), Greene's Infectious Diseases of the Dog and Cat, WB Saunders, pp.443-454.
https://doi.org/10.1016/B978-0-323-50934-3.00035-5

Tian J., Kang H.T., Huang J.P., Li Z.J., Pan Y.D., Li Y., Chen S., Zhang J.K., Yin H., and Qu L.D., 2020, Feline calicivirus strain 2280 p30 antagonizes type I

interferon-mediated antiviral innate immunity through directly degrading IFNAR1 mRNA, PLoS Pathog., 16(10): e100894.
https://doi.org/10.1371/journal.ppat.1008944
PMid:33075108 PMCid:PMC7571719

Tizard I.R., 2021, Feline vaccines, In: Vaccines for Veterinarians, pp.167-187.e1.
https://doi.org/10.1016/B978-0-323-68299-2.00023-X
PMCid:PMC7348621

Urban C., and Luttermann C., 2020, Major capsid protein synthesis from the genomic RNA of feline calicivirus, J. Virol., 94(15): 10-1128.
https://doi.org/10.1128/JVI.00280-20
PMid:32404528 PMCid:PMC7375370

Zheng Y.T., Liu D., Xu X.Y., Yang Y.P., Kang H.T., Jiang Q., Yang M.F., Liu J.S., and Qu L.D., 2022, Genetic variation analysis of calicivirus isolates from immuned cats with calicivirus vaccine, Chinese Journal of Preventive Veterinary Medicine, 44(5): 555-559, 571.