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Feline Calicivirus: from Genetic Evolutionary and Clinical Diversity to Control Strategies in a Review

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Abstract

Feline calicivirus (FCV) is recognized as one of the most prevalent etiological agents of disease in cats, distinguished by its high transmissibility and a remarkable capacity for immune evasion. Following the acute phase of infection, some cats may persist as chronic carriers or experience recurrent cycles of infection with different or mutated strains, making them major Clinical manifestations of infection often include upper respiratory tract infections, oral ulcers, and excessive salivation, in addition to gingivitis and stomatitis in other cases. In some forms, it may progress to a more severe type known as Virulent Systemic Feline Calicivirus (VSFCV), which is associated with high mortality rates. Because of high transcription polymerase error rates in RNA viruses, it has given it a strong ability for genetic adaptation, allowing it to respond quickly to environmental instability. This phenomenon allows it to undergo severe genetic and antigenic diversity, leading to the development of new variants that complicate the process of controlling it later. FCV is associated with a wide range of clinical signs, starting from asymptomatic infections and relatively mild oral and respiratory illnesses to severe lameness with or without systemic infections with a high mortality rate. It is worth noting that a percentage of cats that recover from acute infection remain infected with chronic infection, and it is believed that continued evolution of the virus within these animals helps it evade the host immune response. Although these chronic cases may represent a minority, they play a pivotal role in the epidemiology and spread of the virus.

Keywords: Cat, Feline calicivirus, Review

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Introduction

Feline calicivirus (FCV) is among the most prevalent and clinically significant viruses, representing a major cause of disease in cats worldwide (1). It is recognized now for its involvement in a wide variety of clinical conditions. Feline calicivirus demonstrates extensive variability at the genetic, antigenic, and phenotypic levels. Due to their rapid mutation, like any other RNA viruses. This results from continuous evolutionary processes, including point mutations and genetic recombination(2). Although it has traditionally been identified as a primary cause of upper respiratory tract disease (URTD) and oral lesions, FCV is now recognized as involved in a wide range of clinical conditions. Characterized by rapid mutation, this RNA virus demonstrates extensive variability at the genetic, antigenic, and phenotypic levels (3). This diversity arises from continuous evolutionary processes, including point mutations and genetic recombination. These evolutionary (4) dynamics have led to a wide variety of viral pathotypes, each with distinct tissue tropisms and levels of virulence. Consequently, Feline Calicivirus (FCV infections can present in multiple forms, ranging from acute febrile lameness and dermatitis to more severe tissues such as pneumonia, enteric problems, and gestational complications (5). A worrying concern is the emergence of a highly contagious and systemic disease, which is often fatal and characterized by a systemic inflammatory response (6). The ability of the virus to undergo rapid antigenic diversity can facilitates immune evasion and persistent infections also in addition to raises critical concerns regarding the long-term effectiveness of current vaccination protocols (7) This artical review aims to attend on the latest updates about feline calicivirus FCV infection in cat to more understand about their molecular biology, epidemiology, and pathogenesis in the final improve disease management and control.

Etiology

Feline calicivirus (FCV) belongs to Vesivirus within the family Caliciviridae. This virus is characterized by having a single strand of positive-sense RNA. It is considered a highly specialized feline pathogen. Its primary infiltration was through mucosal surfaces, with it having a specific Infiltration for epithelial and endothelial tissues, and it possesses modern evolutionary mechanisms to escape (8). Structurally, the Feline Calicivirus (FCV) consists of a non-enveloped. single-stranded RNA genome of approximately 7.7 kb which facilitates the synthesis of nonstructural proteins alongside VP1 and VP2 capsid proteins (9)The VP1 protein, encoded by the second open reading frame (ORF2), constitutes the fundamental building block of the viral capsid, wherein 180 copies are arranged in T=3 icosahedral symmetry to form a spherical virion approximately 38 nm in diameter (10) Structurally, the protein is divided into the shell domain (S) featuring a β-jellyroll fold, and the protruding domain (P) comprising the stem (P1) and the terminal head (P2), which contains domain E responsible for binding to the host cell receptor fJAM-A (feline junctional adhesion molecule-A) (11) . Functionally, VP1 plays a dual role: it mediates host cell recognition and viral attachment, which explains the correlation between tight junction disruption and the

pathogenesis of oral and cutaneous ulcerations. Moreover, the protein exhibits substantial antigenic variation (regions A–F), contributing to the pronounced genetic and antigenic heterogeneity observed among FCV strains, thereby complicating the development of broadly protective vaccines and presenting a considerable impediment to achieving comprehensive cross-protective immunity (2). The VP2 protein is encoded by the third open reading frame (ORF3) and is classified as a minor capsid protein essential for completing the infectious cycle rather than for constructing the overall virion architecture. Cryo-electron microscopy (cryo-EM) analyses reveal that upon fJAM-A receptor engagement, VP2 assembles into a large portal-like complex composed of 12 copies arranged helically or cylindrically at a unique three-fold symmetry axis within the capsid(11). Its primary mechanistic function resides in facilitating viral uncoating; receptor binding induces conformational changes in VP1, leading to the formation of a pore within the capsid shell directly beneath the VP2 portal, thereby enabling the viral genome to escape the endosome and translocate into the host cell cytoplasm following clathrin-mediated endocytosis. Furthermore, the molecular interplay between VP1 and VP2 is indispensable for maintaining the structural integrity and functional stability of the virion, and previous studies have demonstrated that VP2 plays a critical role in the self-assembly of VP1 into virus-like particles (VLPs) with the correct morphological appearance (12)

Pathogenesis

Feline calicivirus (FCV) primarily infects the oropharyngeal epithelium, causes epithelial necrosis and transient viremia, and in some strains extends to vascular and systemic disease through broader cell tropism and immune evasion. The oropharyngeal tissues serve as the initial site for viral replication. FCV entry through the conjunctival, oral, or nasal pathways. Early replication in the oral and upper respiratory epithelium causes necrosis of epithelial cells, formation of vesicles and ulcers on the tongue and oral mucosa, with neutrophilic inflammation healing over 2–3 weeks (13)

Around 3–4 days post-infection, a transient viremia distributes virus to other tissues, lungs, joints, and sometimes viscera (8). While most cats eliminate the virus within approximately 30 days, a subset establishes long-term carrier status through colonization of the tonsils and other tissues, resulting in viral shedding that can persist for months to years (14). Receptors, cell tropism, and virulent systemic disease (VS-FCV). The entry of FCV into host cells is mediated by the feline junctional adhesion molecule A (fJAM-A), which is located at the tight junctions of both endothelial and epithelial tissues as well as on leukocytes and platelets, thereby facilitating systemic spread of the virus through the bloodstream (13). Highly virulent strains target vascular and surface cells, triggering widespread vasculitis and compromising junctional integrity. This disruption results in significant fluid leakage (edema) and tissue death across multiple organs, often leading to high fatality rates and extensive mucosal erosions (15). The level of viral pathogenicity is primarily determined by specific residues in the P2 subdomain of the VP1 capsid protein. However, these specific residues vary between strains, and a universal mutation marker for virulence has not yet been identified (16). In addition to the high mutation rate of FCV, which

drives genetic and antigenic diversity and immune evasion, the nonstructural proteins (P30, P32, P39) contribute to viral persistence by inducing autophagy and promoting the autophagic degradation of RIG-I. This interaction contributes to the suppression of type I interferon signalling, creating a favourable environment for viral replication and facilitating the inhibition of innate immune responses (9).

Clinical signs

The classical feline calicivirus (commonly referred to as "cat flu") is classified among the common viral respiratory diseases. It is characterized by a set of clinical signs, including fever, sneezing, nasal and ocular discharge, and conjunctivitis. Painful ulcers in the mouth and tongue are also observed, along with gingivitis or stomatitis, which often lead to loss of appetite and lethargy. In some cases, the disease may progress to pneumonia or lameness syndrome (17). Chronic gingivostomatitis is considered one of the major complications In addition, infected cats often remain lifelong carriers of the virus with prolonged shedding in the oropharyngeal region making them an important source of infection transmission (18) limping syndrome can occur alone or alongside respiratory and oral symptoms(19)Paw and mouth disease ulcers and lesions on footpads and in the mouth, sometimes with systemic illness (20). Although the viral systemic form is rare, it poses a serious threat due to its high mortality rates even among vaccinated cats. Clinical manifestations include fever, swelling of the limbs and extremities, and the development of extensive skin and mucous membrane ulcers, potentially leading to jaundice and organ failure. This strain is often associated with densely populated environments such as shelters or treatment centres (14). This diversity allows for a clinical spectrum ranging from asymptomatic carriage to several distinct manifestations. These include classic upper respiratory and oral disease characterized by sneezing, conjunctivitis, and ulcers of the tongue and palate (8), limping syndrome and associated arthritis (21). A strong association with chronic gingivostomatitis (2). and the most severe form, virulent systemic disease (VS-FCV), which presents with edema, cutaneous and lingual ulceration, multi-organ necrosis, and high mortality, even among vaccinated adult cats (15). Furthermore, some infected cats become long-term carriers, with the virus persisting in tonsillar and retropharyngeal tissues and undergoing prolonged shedding for months to years, thereby sustaining a high prevalence of infection in shelters and multi-cat colonies (22).

Epidemiology

It is primarily spread through direct contact with infected cats via bodily fluids, especially nasal and oral secretions. The likelihood of infection increases in crowded cat environments such as shelters and veterinary clinics. Infection rates vary significantly between regions, ranging from 30% to over 70%, depending on factors such as vaccination status, age, sex, environment, and breed. Notably, certain breeds exhibit more pronounced respiratory symptoms and heightened susceptibility to infection (22). Molecularly, the virus exhibits significant genetic diversity, leading to continuous evolution and genetic recombination, which helps it survive and spread among cats

(14). Although vaccines reduce the likelihood of infection and disease severity, they do not completely prevent infection or transmission due to antigenic variation and the emergence of new highly virulent strains (23). The virus can cause chronic infection in some cats making them a persistent source of transmission Furthermore outbreaks of highly virulent systemic disease have been associated with viral strains that are difficult to distinguish complicating disease control (16) In general the mechanism of virus transmission is influenced by multiple factors including genetic diversity host characteristics, and environmental conditions, highlighting the need for continuous monitoring, the development of more effective vaccines, and the application of specific preventive strategies to limit spread (7)

Prevention and Control

systemic disease Prevention primarily relies on vaccination, which reduces clinical severity but does not fully prevent infection or viral shedding due to FCV's high genetic variability and mutability (19). New vaccine candidates. such as virus-like particle (VLP) vaccines based on broadly neutralizing strains like DL39. show providing cross-protection against multiple FCV genotypes and reducing viral shedding and clinical symptoms (24) vaccines the dominant neutralizing epitope region (CDE) of the VP1 protein also stimulates strong humoral and cellular immunity, offering a potential low-cost and safe alternative for FCV prevention (25) Rapid and sensitive detection methods, including CRISPR/Cas13a-based lateral flow dipsticks have been developed early diagnosis that help quick control of FCV spreation (26) Environmental disinfection using ozone has demonstrated effective virucidal activity against FCV, supporting its use in controlling environmental contamination in multi-cat settings (27) Overall, FCV control requires a combination of vaccination, early detection, and environmental hygiene measures, with ongoing research focused on improving vaccine efficacy against diverse viral strains The disease cannot be eradicated, but vaccination, strict hygiene, and good population management markedly reduce its occurrence and outbreaks Core vaccination against FCV is recommended for all cats that can Vaccines reduce the severity of viral . and duration of RNAemia, but do not fully prevent infection or shedding (19). When disease occurs in fully vaccinated groups due to antigenic diversity, modified-live and inactivated vaccines can be used, meaning changing the vaccine strain or using multivalent/bivalent formulations can be helpful. (27) In multicat settings (shelters, catteries), control relies on smaller groups, minimizing new introductions, quarantining new cats, rapid vaccination upon intake, and strict separation of sick cats (13). High-density groups are at particular risk, including for virulent systemic FCV (VS-FCV). Annual boosters for prime dose vaccination are often advised in groups (19). Intake quarantine limits the introduction of new strains, especially from shelters and strays. Group size reduction decreases transmission. House cats can be individually housed in shelters when possible (17). Rapid diagnostic testing Early detection & isolation, PCR, Nano-PCR CRISPR/Cas13a dipstick for quick field use (28) some disinfectants can kill the virus at specific concentrations, such as sodium hypochlorite 2700 ppm (1 min) accelerated hydrogen peroxide 35,000 ppm (10 min) potassium peroxymonosulfate 1%

(10 min aldehydes 2% ppm chlorine dioxide Ensuring thorough cleaning and removal of organic matter is a critical prerequisite for effective disinfection(29)Several compounds (e.g., nitazoxanide, 2′-C-methylcytidine) show in-vitro anti-FCV activity and PMO therapy has shown promising survival benefits in outbreaks, but are not standard therapies yet (8).

Conclusion

FCV is a major feline pathogen notable for its high genetic variability, serving as a model for viral evolution. It causes respiratory disease and virulent systemic infections, highlighting the need for broadly cross-protective vaccines. Accurate diagnosis integrates clinical, molecular, and epidemiological data. Effective control requires a multifaceted approach combining supportive care, strict disinfection, isolation, and quarantine measures. Further research is essential to develop targeted antiviral therapies.

Conflicts of interest

The authors declare that there is no conflict of interest.

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فيروس الكاليسي عند القطط: من التطور الجيني والتنوع السريري إلى استراتيجيات السيطرة ـ مراجعة شاملة

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الخلاصة

يُعتبر فيروس الكاليسي عند القطط (FCV) أحد أكثر العوامل المسببة للأمراض انتشاراً بين القطط، ويتميز بقدرته العالية على الانتقال وقدرته المذهلة على المراوغة المناعية. فبعد المرحلة الحادة من العدوى، قد يستمر بعض القطط كحاملين مزمنين للفيروس أو يواجهون دورات متكررة من العدوى بسلالات مختلفة أو متحورة، مما يجعلهم مصدراً رئيسياً للعدوى تشمل المظاهر السريرية للإصابة عادةً التهابات الجهاز التنفسي العلوي، وتقرحات الفم، وسيلان اللعاب المفرط، بالإضافة إلى التهاب اللثة والتهاب الفم في حالات أخرى. وفي بعض الأشكال، قد يتطور المرض إلى نوع أكثر خطورة يُعرف بفيروس الكاليسي الجهازي شديد الضراوة(VSFCV) ، والذي يرتبط بمعدلات وفيات عالية ونتيجة لارتفاع معدلات خطأ إنزيم بوليميراز النسخ في الفيروسات التي تعتمد على الحمض النووي الريبوزي(RNA) ، فقد اكتسب الفيروس قدرة قوية على التكيف الجيني، مما يسمح له بالاستجابة بسرعة لعدم الاستقرار البيئي. وتسمح هذه الظاهرة للفيروس بالخضوع لتنوع جيني و مناعي شديد، مما يؤدي إلى ظهور متغيرات جديدة تعقد عملية السيطرة عليه لاحقاً يرتبط فيروس الكاليسي بمجموعة واسعة من الأعراض السريرية، بدءاً من العدوى غير المصحوبة بأعراض والأمراض الفموية والتنفسية الخفيفة نسبيًا، وصولاً إلى العرج الشديد مع أو بدون إصابات جهازية ذات معدل وفيات مرتفع. ومن الجدير بالذكر أن نسبة من القطط التي تتعافى من العدوى الحادة تظل مصابة بعدوى مزمنة، ويُعتقد أن التطور المستمر للفيروس داخل هذه الحيوانات يساعده على التهرب من الاستجابة المناعية للمضيف. ورغم أن هذه الحالات المزمنة قد تمثل أقلية، إلا أنها تلعب دوراً محورياً في وبائية الفيروس وانتشاره.

الكلمات المفتاحية: قطط, فايروس كاليسي عند القطط, مراجعة.