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Research on the Differences in Invasion and Replication of Coronaviruses in Feline and Human Cells

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Abstract: The coronavirus has significantly impacted people's daily lives, inducing inflammatory responses in both the respiratory and digestive systems, particularly in the case of SARS-CoV-2. Coronaviruses are classified into four subtypes, with α-coronaviruses and β-coronaviruses primarily infecting mammals. Most of these viruses are single-stranded RNA viruses that utilize angiotensin-converting enzyme (ACE) as their receptor. However, coronaviruses affecting humans originally originate from animals. Animals such as bats and camels serve as reservoirs, transmitting the virus to humans. These animals exhibit specific immune responses to resist the virus and, in some cases, even coexist with it.What are the underlying mechanisms behind this phenomenon? What differences between animal and human coronaviruses contribute to this adaptation? Can these findings be leveraged to improve current treatments for coronavirus infections?This study will use feline coronavirus (FCoV) as an example to compare the invasion mechanisms, replication processes, and clinical manifestations of human coronaviruses (HCoV) and FCoV. Unlike HCoV, which utilizes ACE as its receptor, FCoV binds to aminopeptidase N (APN) and contains 29,585 amino acids, including 13 open reading frames. Since APN primarily accumulates in the intestines, FCoV predominantly targets the digestive system, whereas HCoV mainly affects respiratory function. By examining the differences between HCoV and FCoV and analyzing the consequences of these distinctions, more effective therapeutic strategies for HCoV can be developed, along with improved preventive measures.

Keywords: Feline Coronavirus, Human Coronavirus, SARS-CoV2, Angiotensin Converting Enzyme (ACE), Aminopeptidase N(APN)

1. Introduction

Over the past decade, human health worldwide has been increasingly threatened by coronaviruses. For instance, in 2019, SARS-CoV-2 posed a severe threat to global public health and caused substantial economic losses, particularly in Wuhan, China. Similarly, the SARS outbreak in 2003 and the MERS outbreak in 2011 resulted in significant harm to human health. These three viruses belong to the family of single-stranded RNA viruses and can cause clinical symptoms such as fever, nasal congestion, and cough. Collectively, they are classified as coronaviruses. [1].

Coronaviruses are classified into four subtypes: α-coronaviruses and β-coronaviruses primarily infect mammalian host cells, whereas γ-coronaviruses and δ-coronaviruses mainly target avian host cells. However, a limited number of mammalian species may also be susceptible to γ- and δ-coronaviruses. The β-coronavirus subtype includes SARS-CoV, SARS-CoV-2, and MERS-CoV [2].

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Studies have identified bats as intermediate hosts of these viruses [3]. Interestingly, coronaviruses can coexist with bats as well as other mammals, such as camels and cats. Coronaviruses that specifically infect the intestinal epithelial cells of cats are collectively referred to as "feline coronaviruses (FCoV)" by researchers [1]. In 1976, researchers first isolated FCoV from local peritoneal cells. They subsequently propagated and cultured the virus using feline kidney cells (CRFK), naming it feline infectious peritonitis virus (FIPV) [4].

FIPV is a highly lethal virus that causes ascites, pleural effusion, fever, and organ granulomas in cats. It is a subtype of FCoV, with the other major subtype being FECV. Currently, there is no effective treatment for FIPV. FCoV belongs to the Coronaviridae family and, like human-infecting coronaviruses such as SARS-CoV-2, is a single-stranded RNA virus. Both FCoV and SARS-CoV-2 recognize host cell membranes through their spike (S) protein, facilitating viral entry by mediating membrane fusion via the S2 subunit and subsequent endocytosis [5].

This article compares the differences in the invasion and replication mechanisms of feline coronaviruses and human coronaviruses, as well as the influence of various host cell conditions on the viral genome. By enhancing the understanding of coronavirus structure and invasion mechanisms, this study aims to facilitate the development of more effective preventive and therapeutic strategies.

2. The Invasion Mechanism of Coronaviruses

HCoV belongs to enveloped RNA viruses, which mainly cause respiratory and intestinal dysfunction in animals and humans [6]. Similarly, FCoV, also an enveloped RNA virus, can cause intestinal and abdominal infections in cats and belongs to the α-coronavirus genus.FCoV is divided into two subtypes. The first subtype, Feline Enteric Coronavirus, is a low-virulence or avirulent virus that primarily causes diarrhea in cats and is not lethal. The second subtype, Feline Infectious Peritonitis Virus, is a highly virulent virus that arises from mutations in FECV [7]. The transmission mode of FCoV is fecal-oral transmission, and it spreads through contaminated feces, urine, saliva, and nasal secretions. Therefore, kittens and cats in environments with numerous cats are more susceptible to infection. Once it invades the host cell, FIPV causes systemic infection and triggers an inflammatory response in many organs and tissues, with the most severe infection occurring in the peritoneum of cats [8]. The mutation of the S gene enables the transmission from FECV to FIPV, with two key amino acid differences: methionine (M) at position 1058 versus leucine (L) or serine (S) at position 1060 versus alanine (A) [9]. Similar to HCoV, FCoV also contains spike protein, membrane protein, envelope protein, and nucleocapsid protein (commonly referred to by their first letters). The spike (S) protein, which plays a critical role in viral invasion, consists of two subunits, S1 and S2. S1 specifically binds to the receptor, while S2 is responsible for fusing with the host cell membrane. The S protein of FCoV, with a rod-like conformation, comprises three regions: the extracellular domain, the transmembrane domain, and a small intracellular domain. The extracellular domain contains most of the protein's functional elements and is composed of both the S1 and S2 subunits. The S1 subunit contains the receptor-binding domain (RBD) and two subdomains: the C-terminal domain (CTD) and the N-terminal domain (NTD). The CTD is responsible for binding to the protein receptor on the host cell, while the NTD binds carbohydrates. The S2 protein contains a fusion peptide and a heptad repeat sequence.

FCoV fuses with the epithelial cells of the cat’s small intestine via its S protein to gain entry into the host. In contrast, mutated FECV—a subtype of FCoV—transforms into FIPV. Upon tissue entry, macrophages mistakenly identify the virus as a foreign body and phagocytize it [7]. Current evidence indicates that a mutation in the S protein gene of FCoV, specifically a substitution from methionine to leucine, may trigger the emergence of FIPV [10]. FCoV comprises two serotypes: type I and type II. The S protein structures of these serotypes differ; type I FCoV possesses two binding sites (S1/S2 and S2’), whereas type II FCoV features only the S2’ cleavage site. The receptor for type I FCoV

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remains unidentified, while it is established that type II FCoV uses aminopeptidase N (APN) for cellular entry. However, the precise molecular interactions between the S protein and APN are still unclear. In the intestine, APN plays a role in the maturation of small proteins and facilitates peptide absorption, and although it is predominantly localized in the digestive system, APN is also present in organs such as the kidney and respiratory tract, where its functions vary according to its location.Moreover, there are significant discrepancies in the amino acid sequences of the S protein between FCoV and HCoV; for example, the sequence similarity between HCoV-NL63 and other coronaviruses is only about 10%. FCoV can utilize C-type lectins as cofactors to aid in cell invasion, and similarly, apart from their primary structural receptors, some HCoVs can also infect cells via C-type lectin-mediated pathways [4].

Following specific receptor recognition, FCoV requires two additional steps for complete host cell entry: protease activation (i.e., S protein cleavage) and endosomal acidification. Furin-like proteases activate the FCoV S protein at the S1/S2 site, while cathepsin cleaves the S2' site via the endosomal entry pathway [10]. Although other proteases may also be involved, their roles remain unclear. Subsequently, the endosomal pH decreases—a necessary condition for membrane fusion between the virus and the host cell. It is still uncertain whether this acidification occurs simultaneously with or after protease activation. Once these steps are completed, the S fusion peptide is exposed, acting as a key regulator of the invasion process. Additionally, while calcium ions drive S-mediated invasion in HCoV-SARS-CoV, their role in FCoV infection remains enigmatic [4].

The primary difference in the invasion process between human coronaviruses and FCoV lies in the distinct receptor-binding sites on host cell surfaces. In human coronaviruses, the S1 subunit specifically recognizes the ACE2 receptor, which is abundantly expressed on lung cells. This widespread ACE2 expression in pulmonary tissues likely explains the propensity of human coronaviruses to infect the lungs and cause respiratory diseases [11]. In contrast, APN is expressed in granulocyte–monocyte lineage progenitor cells, fibroblasts, neurons, kidneys, respiratory tract, and especially in digestive epithelial cells, where both the high expression level and the extensive surface area of the digestive tract provide ample targets for viral entry. This distribution may underlie the predominance of gastrointestinal symptoms in FCoV infections. Once the virus binds to its receptor through precise intermolecular interactions, the S2 subunit undergoes a conformational change to form a stable hairpin structure. This rearrangement shortens the distance between the viral envelope and the host cell membrane, ultimately promoting membrane fusion and allowing the viral genetic material to enter the host cell to initiate infection [4].

3. Differences in the Replication Mechanisms of Coronaviruses in Feline and Human Cells

The replication mechanism of HCoV can be roughly divided into five steps: first, the virus fuses with the host cell membrane to achieve entry; next, the viral genome is translated into polyproteins that are subsequently cleaved into functional proteins; then, transcription occurs to generate subgenomic RNAs (sgRNAs); following this, both structural and nonstructural proteins (NSP) are synthesized; and finally, new virions are assembled and released from the host cell [12].

HCoV, such as SARS-CoV-2, has a genome length of 29.9 kb and encodes 28 main proteins. After the S protein on the surface of the virus specifically recognizes the ACE2 enzyme on the surface of the host cell, the virus enters the host cell and initiates protein translation [13].

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Table 1: The difference between HCoV and FCoV in Invasion Mechanisms

	HCoV	FCoV
Differences in the Main Infected Organs and Cells	Mainly causes respiratory and intestinal infections	Mainly causes intestinal and abdominal cavity infections in cats
Type of Coronavirus	β coronavirus	α coronavirus
Differences in Transmission Modes	Contact transmission	Fecal-oral transmission
Differences in S Protein Structure	Only has S1 and S2	The S protein of the FCoV type I virus has two cleavage sites, S1/S2 and S2', and the FCoV type II virus has only the S2' cleavage site.
Differences in Receptors	ACE2	APN

The genome of the SARS-CoV-2 virus contains 13 open reading frames (ORFs). These ORFs are capable of translating genes into structural and nonstructural proteins. Genes outside the ORFs cannot be translated into proteins. First, in cells infected by SARS-CoV-2, approximately two-thirds of the 5' end RNA positive-sense genomic RNA (gRNA) is directly translated from the overlapping ORF1a and ORF1b into two polyproteins. ORF1a is translated into polyprotein 1a (pp1a), consisting of 4,405 amino acids, while ORF1b synthesizes polyprotein 1b (pp1b), which contains 7,096 amino acids. Second, pp1a is cleaved into 10 nonstructural proteins (NSPs), whereas pp1b is cleaved into 15 NSPs. Among them, the papain-like protease, activated by NSP3, hydrolyzes the viral polyprotein precursors pp1a and pp1ab, generating NSP1, NSP2, and NSP3. The chymotrypsin-like protease, activated by NSP5, cleaves pp1a and pp1ab at 11 different sites, generating 13 NSPs (NSP4 to NSP16). Then, the remaining 30% of the genome, located at the 3' end, is transcribed into 11 subgenomic RNAs (sgRNAs), which encode the four structural proteins (S protein, M protein, E protein, and N protein) and six accessory proteins. After synthesis, the structural proteins are transported from the endoplasmic reticulum to the Golgi apparatus. During this process, the virus undergoes a series of modifications to reach maturity, though the details may vary depending on the host cell type, viral species, and interactions between the host cell and the virus. For example, the S protein of SARS-CoV-2 requires specific enzymes (such as TMPRSS2) for later cleavage and activation to facilitate cell fusion. Once mature, the viral genome is packaged into budding vesicles, which are then released extracellularly as new virions through exocytosis, completing the viral replication cycle. [15].

Structural proteins are essential for constructing the viral structure. The Spike (S) protein consists of a major extracellular domain and a short cytoplasmic tail, forming a trimeric transmembrane protein. It can be cleaved into two subunits. The S protein, located on the virus's surface, specifically binds to the cell receptor angiotensin-converting enzyme 2 (ACE2) and facilitates the membrane fusion between the virus and the host cell [12]. The M protein is the most abundant structural protein and constitutes the majority of the viral membrane. During viral assembly, it interacts with the S, E, and N proteins to form a stable complex, collectively promoting the formation of the viral envelope and the budding process. It is a crucial regulatory protein in the virus assembly process. The E protein is the smallest transmembrane structural protein. It consists of three domains: the transmembrane domain, the intracellular domain, and the extracellular domain. The transmembrane domain helps the virus anchor to the host cell membrane and regulates ion concentration to create a suitable

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environment for viral formation. The intracellular domain participates in RNA formation, while the extracellular domain regulates the host cell's immune response. The N protein binds to the viral RNA to form a ribonucleoprotein complex, protecting the RNA from degradation. Accessory proteins, including 3a, 3c, 6, 7a, 7b, 8, and 9b, are a group of highly variable virus-specific proteins that help regulate the host's response to infection [9]. However, the molecular functions of many accessory proteins remain largely unknown [16].

Unlike HCoV (such as SARS-CoV-2), FCoV contains 11 ORFs. While HCoV and FCoV share the same structural proteins, their non-structural proteins differ. FCoV includes accessory proteins 3a, 3b, 3c, 7a, and 7b, as well as replication proteins 1a and 1b. Its replication process is generally similar to that of HCoV; however, unlike HCoV, two polyproteins, pp1a and pp1ab, are synthesized during viral replication. Additionally, pp1a is cleaved into 11 NSPs, while pp1ab is cleaved into 16 NSPs. The structural proteins of FCoV also exhibit some differences from those of HCoV. The S protein of FCoV has lower glycosylation than that of HCoV. The E protein of FCoV lacks the ability to maintain high ion channel activity, serving primarily to assist in viral assembly and budding. In contrast, the E protein of HCoV can maintain high ion channel activity and plays a role in viral release and immune evasion. For example, the E protein of SARS-CoV induces apoptosis in host cells. Additionally, the N protein of FCoV has a weaker immune evasion capability compared to that of HCoV [4].

In terms of the accessory protein, FCoV lacks the hemagglutinin esterase (HE protein), which is contained in HCoV. Regard of the viral release, FCoV has a special feature comparing with HCoV. FCoV will be released to the basolateral part of the cell membrane, and then, the cell membranes will fuse, inducing the so-called syncytium [4]. The main replication differences between HCoV and FCoV are summarized in Table 2.

Table 2: The difference between HCoV and FCoV in Replication Mechanisms

	HCoV(Taking SARS-CoV2 as an Example)	FCoV
Genome Length	29.9kb	29.255kb
Number of Open Reading Frames	13	11
Difference in the function of structural protein	The function of structural protein is more comprehensive which is designed for adapting different host cell environment from different animals.	The function is simpler, which is designed for exclusively infect feline's cell.
Differences in the Types of accessory protein	3a, 3c, 6, 7a, 7b, 8, and 9b	3a, 3b, 3c, 7a, 7b, 1a, and 1b
Situation of Polyproteins	ORF1a is translated into pp1a, and ORF1b can synthesize pp1b	ORF rep1a and ORF rep1b can synthesize pp1a and pp1ab
Cleavage Products of Polyproteins	pp1a is cleaved into 10 NSPs, and pp1b is cleaved into 15 NSPs	pp1a is cleaved into 11 NSPs, and pp1ab is cleaved into 16 NSPs
Protein Modification During the Transport from the ER to the Golgi Apparatus	Requires specific enzymes (such as TMPRSS2)	It requires glycosylation, and the N protein binds to the newly synthesized

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Table 2: (continued).

		genomic ssRNA+ molecules.
Characteristics of virus release	New virus particles are directly released extracellularly through exocytosis.	New viruses are transported to the cell surface by secretory vesicles, and then released to the basolateral part of the cell membrane, inducing the formation of syncytia.

4. Differences in the Transmission and Clinical Manifestations of Coronaviruses in Feline and Human Cells

The main transmission routes of HCoV are respiratory droplets and close contact [13]. The common symptoms of SARS-CoV-2 infection include fever, cough, shortness of breath, and muscle aches, which are similar to those of SARS and MERS. Some patients may experience a sore throat, runny nose, headache, or confusion in the days preceding fever onset. A small number of patients exhibit hemoptysis, while a significant proportion do not develop such symptoms. Hematologically, patients may present with a low white blood cell count, decreased lymphocytes or platelets, and elevated C-reactive protein levels. Fever may be accompanied by upper respiratory tract symptoms and can lead to leukopenia or lymphocytopenia, particularly in individuals with a history of travel to epidemic areas or close contact with infected individuals. Clinical reports also suggest that male patients may experience erectile dysfunction and anhedonia. The most critical and common clinical manifestation is severe acute respiratory distress [12].

The major transmission route of FCoV is fecal-oral transmission. Mutations in FCoV can lead to different clinical symptoms and pathogenic characteristics, ranging from mild gastrointestinal disease (feline enteric coronavirus, FECV), which typically presents without specific abnormalities, to severe and often fatal systemic disease (feline infectious peritonitis, FIP), caused by mutation into the virulent strain (feline infectious peritonitis virus, FIPV) along with the specific immune response of infected cats. FIP manifests in two major clinical forms: the wet form and the dry form [5]. The hallmark of the wet form is the accumulation of fibrin-rich fluid in body cavities, leading to symptoms associated with severe acute hypovolemia, increased pressure on organs within the affected cavity, and resulting in dyspnea or impaired movement. The clinical manifestations of the dry form depend on the primary site of granulomatous lesion formation. If lesions predominantly occur in the kidneys, renal function may decline. However, when granulomatous lesions develop in other organs, such as the liver, lungs, intestines, or particularly the eyes and central nervous system, extrarenal symptoms may arise. These symptoms include fever, depression, weakness, weight loss, anemia, lymphocytopenia, and elevated concentrations of serum globulin components and acute-phase proteins [5].

5. Conclusion

The differences between HCoV and FCoV lead to distinct characteristics in their transmission mechanisms and replication processes. HCoV utilizes ACE2 as its receptor, which is primarily located in the lungs, epididymis, and testicles, allowing HCoV to significantly affect the normal function of these organs. In contrast, FCoV uses APN as its receptor, which is mainly expressed in

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the intestines and also found in neurons, kidneys, and the respiratory tract. FCoV primarily causes diarrhea, with kidney failure as a possible outcome. Although HCoV primarily uses ACE2 as a receptor, which is also expressed in cats, HCoV can infect domestic cats. For example, during the COVID-19 pandemic, several reports documented cross-infection of SARS-CoV-2 between humans and cats. However, although humans also express APN, there are several differences in the amino acid sequences of the APN protein between humans and cats. As FCoV specifically recognizes the amino acid sequence of APN in cats, it cannot infect humans.

The differences in the replication processes of HCoV and FCoV can also lead to distinct viral characteristics. For example, HCoV, such as SARS-CoV-2, has 13 ORFs, allowing it to infect various animals, including humans, camels, and bats. In contrast, FCoV has only 11 ORFs, meaning it can primarily infect cats. The greater number of ORFs in HCoV helps explain its ability to infect multiple animal species, as these ORFs enable the virus to produce more structural proteins capable of recognizing different amino acid sequences in ACE2.

The differences in the virus release processes can also be attributed to their distinct living environments. Since FCoV primarily accumulates in the digestive system, where various digestive fluids are present, its release process is adapted to this acidic environment. In contrast, HCoV is localized mainly in the lungs, and its direct release process enables it to quickly infect other animals.

For FCoV, a serological test for infection revealed that all cats infected with the virus produced FCoV-specific serum antibodies. These antibodies could be detected 10 days after inoculation. There was a clear trend that animals infected with a higher dose produced higher and more persistent serum antibody titers. The induced antibodies recognize FCoV structural proteins M, N, and S in the Radioimmuno Precipitation Assay (RIPA). Antibodies against the N protein were dominant and could be detected first by this assay. Based on this, we can speculate that in the treatment of FCoV, developing drugs that specifically inhibit the synthesis of the N protein within host cells could effectively prevent viral replication. The same conclusion can be applied to the development of vaccines for HCoV, especially SARS-CoV-2. By injecting the viral nucleocapsid of SARS-CoV-2 into the human body, it can stimulate the formation of antibodies that specifically recognize these proteins, thereby providing a preventive effect.

The research on HCoV and FCoV is of great significance for the treatment and prevention of both diseases. The well-known FIPV drug GS441 is derived from Molnupiravir, a drug used to treat SARS-CoV-2. Molnupiravir targets the SARS-CoV-2 RNA-dependent RNA polymerase (RdRp) in a similar manner to other nucleoside analogs, preventing the replication and transcription of the coronavirus genome. However, the specific mechanism of action remains unclear, and we still do not fully understand why GS441, or Molnupiravir, is effective in treating FCoV. Additionally, the reasons behind the mutation and conversion of FECV to FIPV require further investigation.

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