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THE EVOLUTION OF SARS-COV-2, A REVIEW OF GENETIC MUTATION

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ABSTRACT

SARS-CoV-2 emerged from zoonotic coronaviruses and is a novel beta-coronavirus which causes severe respiratory disease (pneumonia and lung failure), termed COVID-19. This paper describes SARS-CoV-2 genetic features (mutations and molecular epidemiology) but highlights its key differences from animal coronaviruses. We conducted a synthesis of the knowledge regarding clinical, genetic and pathological features of animal coronaviruses in comparison to SARS-CoV-2, along with recent evidence of interspecies transmission and recombination of animal coronaviruses to inform a One Health perspective of SARS-CoV-2 infection. We also take a closer look at the likely animal reservoirs and zoonotic origins of this novel virus that could help to curb disease transmission and minimize disease impact.

Keywords: SARS-CoV2, Cannine, COVID-19, Genetic Mutation, Genotyping

1. INTRODUCTION

COVID-19 trends

This report describes the first pandemic of coronavirus due to infection with the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that leads to coronavirus disease 2019 (COVID-19), and originated from an epidemic at Wuhan, a city in China.

That said, different countries have varying levels of cricket infection and death. COVID-19 is a whole new level of novel for a human virus. As a result, the information we have on this virus today is mixed, and there are still some things that we do not understand. Simply put, potential alterations in the transmission

modes of the virus The next 1–2 years are uncertain [1]. The cycle of future epidemics will continue to happen until we have an effective vaccine that stops the disease or sufficient levels of the world population become infected (60% to 70% of the world population) and thereby have herd immunity [2]. Until then, millions will carry on living with "Corona-phobia" .

Thus, our ability to predict and plan for this situation until safe and effective vaccines become available or herd immunity is reached is paramount [3]. COVID-19 is highly transmissible compared to the SARS epidemic in 2003 and the MERS epidemic in 2012 [4]. For the very high rate of asymptomatic diseases, it is a major fault to consider only people with fever and cough to be patient; keeping the mask ALWAYS on is very important to avoid the spread of silent carriers. Which basically comes down to protecting in particular the intangible, plan for it and you have one of the best-ever defense strategies. In addition, COVID-19 has raised pre-symptomatic transmission during the 1 to 3 days preceding symptom onset, generating 2.5 to 3.5 secondary cases per infected person. As of now the global death rate of COVID-19 has so far oversized the death rate of the Spanish flu pandemic which was 2.5%. More than 1,000 people in the U.S. have died, as the number of confirmed cases hits a daily record. The death rate was 3.4% in China [5].

Herd immunity builds up gradually in the population through multiple epidemics, resulting in the epidemic being over when the herd immunity threshold is reached (60% −70%), and lasting from 18 to 24 months [6]. However, there is still much confusion regarding COVID-19. Although COVID-19 infection generates >90% of the antibodies, additional data are needed to determine if they work in the prevention of re-infection. Despite high levels of antibody production, there are multiple examples of limited long-lived immunity, therefore it is only logical that antibody levels drop over the course of several months to a few years, and re-exposure may cause reinfection. Uncertainty in the development and duration of protective immunity and the difficulty to induce neutralizing antibodies represent serious challenges to the complete elimination of COVID-19.

At the end of this pandemic, the virus was expected to be less virulent and contagious because the development of herd immunity would probably make it more closely related to a seasonal coronavirus (like the seasonal flu). Globally, the S and V variant groups of SARS-CoV-2 were most prevalent until early April 2020 [7], while the G, GR, and GH groups are currently widespread. The GR group is mainly found in Africa, India and Russia, while the GH group is mainly found in North America, Europe and Middle East. Replicating and transmitting rapidly than other strains, the GH group virus likely has better binding to the host cell [8]. The Centre for Infectious Disease Research and Policy tracked three scenarios for the COVID-19 pandemic. In the first scenario, several minor pandemics hit much more frequently, every year or season at most, and minimal spread can be achieved through strict quarantine during peaks of outbreaks, only to reemerge when some restrictions are loosened [9]. The second involves a serious new pandemic hitting in the fall, the most alarming of the three possibilities. The third scenario is the most favorable one, and it is the one that multiple experts desire. In spite of its big slaughter over the early course of the pandemic, least damage came from mostly 4 small outbreaks since. Thus, the third scenario is the most desirable, especially because the pandemic could be controlled by strict quarantine measures, regardless of the spread of contagious diseases in each region. Also, retaining this pattern for two years or so makes flare-up of immunization achievable.

During the 2009 pandemic influenza, the number of patients increased in September when schools started after the summer holidays and then peaked by late October to early November. However, there were antivirals available then, and vaccination also helped control the pandemic. On the flip side, COVID-19 is worse because there is no specific treatment or vaccine available right now. Here, COVID-19 is still in the first phase of the pandemic and will remain there until there is a treatment and a vaccination.

SARS-CoV-2

Origin and evolution of SARS-CoV-2

SARS-CoV-2 genetic characterization showed a 96% nucleotide identity to a bat-derived strain called Beta CoV/RaTG13/2013. Other studies show that many SARS-CoV-like viruses that belong to subgenus Sarbecovirus have been reported in Rhinolophus bats in South China. These viruses show genetic diversity and rapid recombination which increases the risk of transmission to humans [10]. The Genomic Identity of this Pangolin coronavirus is 91.02% and 90.55% with SARS-CoV-2 and BatCoV RaTG13. Pangolin-CoV is phylogenetically closest related to SARS-CoV-2 after RaTG13. Secondly, the phylogenetic relationship of the S1 protein of pangolin-CoV to SARS-CoV-2 was found to be closer than that to RaTG13. For pangolin-CoV and SARS-CoV-2 the five essential amino acid residues involved in the binding with human ACE2 are the same while RaTG13 has four amino acid changes. Unlike SARS-CoV-2, both Pangolin-CoV and RaTG13 do not exhibit a putative furin recognition sequence motif at the S1/S2 cleavage site. This suggests that pangolins could be an animal reservoir of the human-like coronaviruses [11].

SNP sequencing showed more prevalence of L (70%) than S lineage (30%), and phylogenetic analysis suggested that S lineage was closely related to the coronaviruses in mammals [12]. Phylogenetic analyses showed that the genetic differences of PEDV and TGEV were only 42.8% and 43.5%, respectively, from SARS-CoV-2, while genomic similarities of PHEV and PDCoV with SARS-CoV-2 were 49.2–49.3% and 40.3–40.4%, respectively. In addition, although it is unlikely that SARS-CoV-2 evolved from pig coronaviruses, SARS-CoV-2 RBD may interact with swine ACE2, because of the high similarity of the 5 binding residues of viral to ACE2 residues in human ACE2 [13].

Birds can serve as a natural seed bank for evolution of gamma coronaviruses and delta coronaviruses and continuous evolution and spread of coronaviruses. For attachment and entry, IBV is usually binding to cellular receptors through sialic acid. They observe a genetic similarity of 43.0–43.2% between SARS-CoV-2 and IBV, which is extremely significant [14].

They belong to the genus Beta coronavirus and are classified as bovine coronaviruses. CoV, which is much like human CoV alf44/US/94, a strain isolated from kids and of potential, public health importance. In addition to BCoV being associated with human beta coronavirus OC43 which is a major pathogen of the common cold, studies suggest that BCoV might be an ancestor of human CoV or that they share a common ancestor [16]. BCoV has a wide host range as it infects dogs, chickens, and giraffes. A genomic study suggested that BCoV is only 49.2–49.3% genetically similar to SARS-CoV-2 [15].

Genotypes of SARS-CoV-2

In a recent study 1234 mutations were detected by sequencing 12343 SARS-CoV-2 genes sequenced from patients at six locations. The COVID-19 mortality

rates in 28 nations were grouped into three categories using hierarchical clustering of mutation frequency [17],. Alleged associations of nucleotide mutations in 11,083 genes encoding Nsps and severity of COVID-19 The 11083G mutation was frequently detected in symptomatic patients while infection with 11083T variants was associated with asymptomatic infection.

Genetic mutations of SARS-CoV-2: Analysis of the genomes of 30,366 SARS-CoV-2 isolates found 11 alterations in genes occurring with a frequency of greater than 10%. From the eleven mutations detected, eight of them were nonsynonymous: C1059T, G11083T, C14408T, A23403G, G25563T, G28881A, G28882A, and G28883C while the other three were synonymous mutations that caused no complementary amino acid change. The main mutations C14408T and A23403G (1,3) were mutationally correlated (based on Fisher's Exact test, P 10% of the genomes of SARS-CoV-2 isolates in the ORF1ab region. Frequency of the C3037T variant in the Nsp3-encoding region (29.3%) The other three mutations, all with a frequency greater than 10% and that involve changes to the amino acid coding areas, are located in Nsp2, Nsp6 and Nsp12. The Nsp2 C1059T mutation leads to the amino acid substitution T266I; however, the functional relevance remains unknown [18]. G11083T mutation → L36F mutation in Nsp6 → stimulation of the vesicle formation near the microtubule regulatory center and membrane proliferation. C14408T and C14805T resides in Nsp12 that is essential for protein replication and pathogenesis. The C14408T mutation influences the P232L missense mutation and is considered be important for viral transmission based on the rapid rise of its frequency.

Mutations in accessory and structural proteins: Gradual mutations mostly occur in G25563T of the auxiliary protein, A23403G of the S protein and G28881A, G28882A and G28883C of the N protein (from GGG to AAC) G25563T within the 3a ORF, encoding a unique membrane protein with three-membrane type and is important for virulence. More than 10% of the S and N protein amino acid substitutions occur in the structural proteins S, E, M, and N. The D614G substitution in the S protein results from the A23403G mutation, which possibly is one of the most impactful characterized mutations thus far. Similar to the C14408T mutation in Nsp12, the A23403G mutation in the S protein is present in 70.46% of cases [19].

N protein plays essential roles in regulating the metabolism of the infected cells and assembles the viral components essential for viral replication and transcription. The presence of three concurrent mutations in the N protein (G2881A, G2882A, G2883C) has been recorded. G28881A and G28882A cause R204K replacements and G28883C causes G205R substitutions [20].

Coronaviruses in animals and their evolving genetic traits

With an unusual virus in human population, SARS-CoV-2 is a zoonotic virus of unknown origin. Coronaviruses represent major veterinary diseases and targeted countermeasures exist for multiple species. In livestock and companion animals, coronaviruses cause gastrointestinal, respiratory and systemic diseases [21]. The animal coronaviruses have evolved and changed, providing a model for preparing for future adaptations that will likely happen with SARS-CoV-2.

Coronaviruses in dogs

The first reports of canine coronavirus (CCoV) emerged in the year 1974 [22]. When dogs are solely infected with canine coronavirus (CCoV), it rarely causes morbidity or mortality and generally produces mild to moderate gastrointestinal

illness. However, it was noted that canine coronavirus might be deadly in the case of a co-infection with canine parvovirus 2 [23]. Animal experiments have shown that CCoV predominantly targets enterocytes but it does also impact other organs such as the lungs, liver and tonsils, Since 2009, fatal coronavirus infections have been recorded. Canines with systemic diseases, characterized by fever, lethargy, neurological signs, and diarrhea were also positive for CCoV. This virus, called pantropic CCoV , the cause for this has been the pantropic CCoV that has caused the CCoV infection into tissue beyond gastrointestinal nausea/diarrhea [24].

CCoV exists in only two serotypes, type I and type II, the latter further subdivided into type IIa and IIb. CCoV type I has been shown to be a recombination product of CCoV and FCoV. In the S protein, 81.76% sequence homology with an FCoV type I (UCD1) and 54.31% with another CCoV strain (K378) was recorded for a CCoV strain of FCoV (Elmo/02). In contrast, the FCoV-like CCoV and the reference CCoV were grouped into CCoV type. This resulted in TGEV-like CCoV, CCoV type IIb, with a CCoV type II backbone partially substituted for the TGEV S1 region by intra-species recombination between CCoV type II and TGEV. Although recombination events were shown to not be a major determinant of pathogenesis in CCoV, genetic polymorphism or divergence could be one of the reasons for the virulence of CCoV type II strains. The BGF strain of CCoV type II has longer ORF3b sequences than other CCoV type II strains, including the CCoV type II CB/05 strain, which has a deletion in ORF3b. These mutations result in the unusual pathogenicity of the two strains, accompanied by severe gastrointestinal signs in the BGF strain or multiple organ tropism in the CB/05 strain [25]. The strain is called "pantropic CCoV" due to its broad tissue tropism and the CB/05 strain exhibits more variable tropism 46. Although the exact mechanism of multi-organ tropism is still unknown, this virus showed continuous transmission in Europe, China, and Brazil [26].

Coronaviruses in cats

FCoV was first reported in 1968, FCoV exists as two serotypes: type I and type II [27]. FCoV is classified into two biotypes according to the disparity in clinical manifestation, feline enteric coronavirus (FECV) and feline infectious peritonitis virus (FIPV). FCoV type II originates from the recombination event between FCoV and CCo. Many studies have shown recombinant between CCoV and FCoV in the ORF1, S and M genes . It has shown that the S protein is the main recombination hotspot. Consequently, they provide differences in neutralisation and cell receptors used for infection between type I and type II strains of FCoV) [28]. It was widely accepted that FCoV used feline APN (fAPN) to enter feline cells, as coronaviruses originally used aminopeptidase N (APN) as receptor. While FCoV type I does not utilise fAPN, this entry receptor is used exclusively by FCoV type II. Feline C-type lectin dendritic cell-specific intercellular adhesion molecule-3-grabbing nonintegrin (fDC-SIGN) was proposed as a co-receptor for FCoV types I and II, but the exact cellular receptor for FCoV type I has not been identified so far. Characteristics of the homo-zenogeneous structure of the cellular receptor may facilitate the adaptation of FCoV type II over type I, independent of the in vitro culture characteristics of FCoV type II [29].

Among all the viral diseases in cats, coronavirus causes the most lethal disease entity known as feline infectious peritonitis (FIP). In addition, FCoV is a virus of great therapeutic interest, because it is related with one of the deadliest diseases in cats, FIP. Contrasting with the aetiology of FIP which remains the matter of dispute, the pathophysiology of the disease is accounted for by the "internal mutation" model, which assumes that mutations arise in the S, ORF7 and ORF3

regions of FECV, regardless of type. The most important mutation is the aa substitution in the S protein that is responsible for the change in tissue tropism of enterocytes to macrophages or monocytes. The current understanding of FIP dictates that, in the face of different tissue tropism, the enteric disease caused by FCoV became a deadly systemic disease when FCoV, after first being present in the intestines of kittens, infects intacells that are vacated of their enterocyte contents, enter phagocytic cells and change submit cells with different genetic predisposition for enteritis originating from enterocytes into macrophages and other associated cells [30].

Coronaviruses in pigs

Pig coronaviruses that have received most attention include transmissible gastroenteritis virus (TGEV), porcine respiratory coronavirus (PRCV), porcine epidemic diarrhoea virus (PEDV), porcine hemagglutinating encephalomyelitis virus (PHEV), and porcine delta-coronavirus (PDCoV). While TGEV, PRCV, and PHEV have been infecting pigs for many years, PEDV and PDCoV are more recent arrivals. Swine acute diarrhoea syndrome coronavirus (SADS-CoV), a novel highly pathogenic intestinal coronavirus, has emerged in China in 2016 and a high mortality rate among pigs has been noted [31].

The first known report of TGEV, an alpha coronavirus, occurred in the United States in 1946, following an outbreak of severe diarrhea with high mortality rates in piglets. However, the clinical significance of TGEV was reduced around the world with the introduction of PRCV, where a spontaneous mutant of TGEV, once formed and deletion of a large part of S protein of TGEV. Currently, indirect transmissions of acute diarrhea in piglets due to TGEV intermittently appears on North America, Europe and Asia farms negative for both TGEV and PRCV. Three distinguishing features have previously been reported on the genomic characteristics of PRCV and TGEV. Compared with TGEV, the PRCV S gene encodes a truncated S glycoprotein due to a deletion of nucleotides 621–681. Differences in Each TGEV and PRCV ORF3 Segment The differences between TGEV and PRCV are located in each ORF3 segment. While the leader RNA-binding site (CTAAAC) preceding the ORF3a gene is changed or partly deleted in PRCV [32].

Chimeric viruses with the TGEV backbone containing the S protein of PEDV have been identified in several European countries in recent years, suggesting the possibility of such chimeric viruses entering the United States. Of these, eight dropouts with potential biological significance and 119 different amino acids representing an altered genotype were apparent among six TGEV mutants in the United States. This changed genotype bore resemblances to deletions and amino acid changes when compared with a newly identified variant of porcine respiratory coronavirus (PRCV), suggesting a possible genetic recombination between TGEV and PRCV. The TGEV genotype represented by this mutant is the currently dominant TGEV genotype in the U.S. [33].

Between 2016 and 2017, a genetically different but clinically similar novel alpha-coronavirus was reported. Background: SADS-CoV is a newly discovered lethal virus, which likely evolved from HKU2 bat coronavirus, which infects the same Rhinolophus bat species in China as SARS-CoV. SADS-CoV displays 98.48% sequence similarity with HKU2 bat coronavirus, implying common ancestry. After the initial epidemic from May 2017 until January 2019, no cases were recorded on other pig farms, but a mutant SADS-CoV/CN/GDLX/2019 was detected in February 2019. The identity of other SADS-CoV variants identified in Guangdong with the S gene of SADS-CoV/CN/GDLX/2019 was 99.2%–99.9% (all 7 S gene sequences in

GenBank), and the lowest identity was 97.5% with SADS-CoELFJ. Recombinant rSADS-CoV has been experimentally cultured in numerous mammals such human hepatoma cell lines and primate fetuses, but no high-expressing cell lines are able to produce rSADS-CoV to a high titer. In addition, the rSADS-CoV virus did not use either ACE-2, DPP4, or CD13 as entrance receptors, which are common receptors for human coronaviruses. Although SADSCoV replication has not yet been confirmed in humans, the ability of SADSCoV to replicate in primary human cells means that transmission to humans is possible [34].

Coronaviruses in chicken

After the first isolation and identification of chicken Coronavirus (IBV) in the US in 1931, multiple mutant strains have been identified worldwide. Many are recombinants with other strains, not generated by the build-up of point mutations. Point mutations and recombination could occur on both structural and non-structural proteins of IBV. These mutations and recombination events are mainly in S gene and also in polyproteins 1a and 1ab. Mutation and recombination of the S1 genes are essential for IBV immunogenicity and diversity because changes in the IBV S protein, particularly the S1 gene, underlying important viral phenotypic and virulence [14]. Although many vaccines for IBV are available, the last decade has seen the identification of new genotypes, serotypes, and pathogenic IBV variants as a result of mutation. So far, these mutations of IBV have been reported in China, Korea, and Egypt. More than seven different major IBV genotypes (GI–GVII) and 35 different lineages (1–35) as well as several other genotypes have been identified. Distribution of GI-1 (former Massachusetts; Mass), GI-13 (793/B, 4/91 or CR88), GI-19 (LX4 or QX), GI-16 (ck/ CH/ LDL/97I (LDL/97I) or Q1), GI-21 (Italy, 02) and GI-23 (Var2) to specific continents, countries, or regions. In addition, nucleotide replacement or recombination frequently occurs between the field and vaccine strain. Even the most commonly used commercial vaccination, the GI-1 genotype H120 vaccine, bears little similarity to the predominant GI-19, or other extant genotypes. As a result, it is claimed that this vaccine does not confer adequate protection against genotype infection, and several epizootics have occurred in vaccinated sheep [35].

Genome sequences of a recombinant strain of infectious bronchitis virus using the S1 gene as a genetic marker. Identification of this novel highly-pathogenic genotype, CK/CH/2010/JT-1. This indicated that mutant CK/CH/2010/JT-1 had a serotype different from H120 and 4/91, as the serum targeting H120 and 4/91 could not fully neutralize it. As a result, the H120 and 4/91 Mass strain-specific vaccines only poorly protect against CK/CH/2010/JT-1. In addition, isolates such as CK/CH/2010/JT-1 were recognized as new genomic group [36].

Inter-species transmission

SARS-CoV, distantly related to bat coronaviruses, was transmitted through masked civet to humans in 2002, MERS-CoV distant from bat coronaviruses to humans via dromedary in 2013 and SARS-CoV-2 closely related to bat coronaviruses through pangolin to humans in 2019 [37]. Third, the inter-species transmission episodes to humans were recently. The genetic basis of cross-species transmission from avian and bat source is exemplified by animal coronaviruses, especially that of swine coronaviruses, as seen with PDCoV and SADS-CoV (frequently called as SeACoV) (swine) indicating high genetic association with bat

HKU2-CoV (bat) [38]. The main reservoir hosts of delta-coronaviruses include wild avians like sparrows and bulbuls but this group of viruses can also be transmitted inter-species to some mammals, including pigs, Chinese ferret badgers, and Asian leopard cats. In an experimental setting, a strain of the novel delta-coronavirus PDCoV was transmitted from pigs to chickens. Hens co-mingled shed PDCoV and serologies positive to PDCoV. Even though SeACoV and PDCoV belong to different genera compared with SARS-CoV-2, these coronaviruses may be used as models for alphacoronavirus and deltacoronavirus inter-species transmission, respectively. Since PDCoV has been shown to transmit efficiently in an inter-species transmission experiment, the infection cycle of animal coronaviruses can be informative. In addition, with the exception of TGEV, recombinant coronaviruses (e.g., FCoV type II (FCoV + CCoV), CCoV type I (CCoV + FCoV), CCoV type IIb (CCoV type II + TGEV), and SeCoV (TGEV + PEDV)) could be regarded as the models of inter-species recombination for human betacoronaviruses, which has not been reported so far [39].

Alterations in tissue tropism

Here we discuss the change of tissue tropism due to mutation of S gene of FIPV, pantropic CCoV, PRCV, huge deletion in PEDV and genetic variability in IBV. Most changes in tissue tropism are due to mutations in the S protein that are important to bind to cellular receptor. However, regardless of genetic changes, both SARS-CoV, MERS-CoV and SARS-CoV-2 showed a broad multiorgan tropism [40]. SARS-CoV-2 caused renal failure in patients as SARS-CoV and MERS-CoV did. These observations may be pertinent to the role of IBV in renal infections. Therefore, IBV and SARS-CoV-2 initiate infection in the respiratory tract then spread to kidneys through primary viremia [40].

2. CONCLUSION

SARS-CoV-2 is classified as a beta-coronavirus. Like SARS-CoV-2, animal beta-coronaviruses, including BCoV and canine respiratory coronavirus (CRCoV), frequently exhibit respiratory symptoms and diarrhea. Consequently, animals with similar viral pathology of beta-coronavirus may serve as prospective infection models for SARS-CoV-2 .

Ferrets, due to their physiological similarities, serve as models for studying respiratory illnesses, such as influenza viruses and coronaviruses. Clinical symptoms and infection patterns may be modelled in ferrets [41]. The interspecies scenarios of PDCoV and SADS-CoV may serve to simulate the SARS-CoV-2 spillover hypothesis. Moreover, in light of antigenic diversity, vaccine development, and infection dynamics, poultry populations may serve as a valuable etiological and economic model for SARS-CoV-2. IBV has consistently progressed since its discovery [42], and the accumulation of genetic variety and differences in tissue tropism has impeded the creation of effective vaccines [43]. Furthermore, the dynamics of the chicken business might resemble those of human communities concerning population density.

Since the initial identification of coronavirus IBV in 1931 [44], coronaviruses have been prominent diseases in animals and the livestock sector. Although human coronaviruses were identified in the 1960s, they were mostly regarded as an underestimated category of pathogens in clinical settings until the SARS outbreak in 2002. Currently, the coronavirus is no longer an unfamiliar pathogen as it was in the

21st century; it has been recognized for at least 90 years, approaching a century. Comprehensive studies have been conducted to manage coronavirus infections in livestock, including poultry, cattle, and swine, as well as in companion animals. The trials and errors in veterinary medicine may serve as a benchmark for managing the lethal coronavirus infection in contemporary times. Likewise, the advanced technology created for managing COVID-19 and the prospective innovative vaccine platforms or antivirals may facilitate advancements in the control of animal coronavirus infections.

CONFLICT OF INTERESTS

None.

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REFERENCES

Epidemiology Working Group for NCIP Epidemic Response, Chinese Center for Disease Control and Prevention. [The epidemiological characteristics of an outbreak of 2019 novel coronavirus diseases (COVID-19) in China]. Zhonghua Liu Xing Bing Xue Za Zhi. 2020 Feb 10;41(2):145-151. Chinese. doi: 10.3760/cma.j.issn.0254-6450.2020.02.003.

Brett TS, Rohani P. Transmission dynamics reveal the impracticality of COVID-19 herd immunity strategies. Proc Natl Acad Sci U S A. 2020 Oct 13;117(41):25897-25903. doi: 10.1073/pnas.2008087117.

Guo R, Fan B, Chang X, Zhou J, Zhao Y, Shi D, Yu Z, He K, Li B. Characterization and evaluation of the pathogenicity of a natural recombinant transmissible gastroenteritis virus in China. Virology. 2020 Jun;545:24-32. doi: 10.1016/j.virol.2020.03.001.

Lee PI, Hsueh PR. Emerging threats from zoonotic coronaviruses-from SARS and MERS to 2019-nCoV. J Microbiol Immunol Infect. 2020 Jun;53(3):365-367. doi: 10.1016/j.jmii.2020.02.001.

Abdelrahman Z, Li M, Wang X. Comparative Review of SARS-CoV-2, SARS-CoV, MERS-CoV, and Influenza A Respiratory Viruses. Front Immunol. 2020 Sep 11;11:552909. doi: 10.3389/fimmu.2020.552909.

Mohammed, G., & Abdulrahman, G. (2023). Comparative ELISA Detection of SARS-COV-2 Monoclonal Antibodies in Patients` Serum, Saliva, and Nasal Fluid in Iraq. Israa University Journal for Applied Science, 6(2), 15–41. https://doi.org/10.52865/nwgt5493.

Gussow AB, Auslander N, Faure G, Wolf YI, Zhang F, Koonin EV. Genomic determinants of pathogenicity in SARS-CoV-2 and other human coronaviruses. Proc Natl Acad Sci U S A. 2020 Jun 30;117(26):15193-15199. doi: 10.1073/pnas.2008176117.

Mercatelli D, Giorgi FM. Geographic and Genomic Distribution of SARS-CoV-2 Mutations. Front Microbiol. 2020 Jul 22;11:1800. doi: 10.3389/fmicb.2020.01800.

Xu S, Li Y. Beware of the second wave of COVID-19. Lancet. 2020 Apr 25;395(10233):1321-1322. doi: 10.1016/S0140-6736(20)30845-X.

Wong ACP, Li X, Lau SKP, Woo PCY. Global Epidemiology of Bat Coronaviruses. Viruses. 2019 Feb 20;11(2):174. doi: 10.3390/v11020174.

Zhang, G., Li, B., Yoo, D., Qin, T., Zhang, X., Jia, Y., & Cui, S. (2020). Animal coronaviruses and SARS-CoV-2. Transboundary and Emerging Diseases. doi: 10.1111/tbed.13791 .

Tang X, Wu C, Li X, Song Y, Yao X, Wu X, Duan Y, Zhang H, Wang Y, Qian Z, Cui J, Lu J. On the origin and continuing evolution of SARS-CoV-2. Natl Sci Rev. 2020 Jun;7(6):1012-1023. doi: 10.1093/nsr/nwaa036.

Wen J, Cheng Y, Ling R, Dai Y, Huang B, Huang W, Zhang S, Jiang Y. Antibody-dependent enhancement of coronavirus. Int J Infect Dis. 2020 Nov;100:483-489. doi: 10.1016/j.ijid.2020.09.015.

Zhang T, Wu Q, Zhang Z. Probable Pangolin Origin of SARS-CoV-2 Associated with the COVID-19 Outbreak. Curr Biol. 2020 Apr 6;30(7):1346-1351.e2. doi: 10.1016/j.cub.2020.03.022. Epub 2020 Mar 19. Erratum in: Curr Biol. 2020 Apr 20;30(8):1578. doi: 10.1016/j.cub.2020.03.063.

Zhang X, Deng T, Lu J, Zhao P, Chen L, Qian M, Guo Y, Qiao H, Xu Y, Wang Y, Li X, Zhang G, Wang Z, Bian C. Molecular characterization of variant infectious bronchitis virus in China, 2019: Implications for control programmes. Transbound Emerg Dis. 2020 May;67(3):1349-1355. doi: 10.1111/tbed.13477.

Hasoksuz M, Alekseev K, Vlasova A, Zhang X, Spiro D, Halpin R, Wang S, Ghedin E, Saif LJ. Biologic, antigenic, and full-length genomic characterization of a bovine-like coronavirus isolated from a giraffe. J Virol. 2007 May;81(10):4981-90. doi: 10.1128/JVI.02361-06.

Toyoshima, Y., Nemoto, K., Matsumoto, S. et al. SARS-CoV-2 genomic variations associated with mortality rate of COVID-19. J Hum Genet 65, 1075–1082 (2020). https://doi.org/10.1038/s10038-020-0808-9.

Chen Y, Liu Q, Guo D. Emerging coronaviruses: Genome structure, replication, and pathogenesis. J Med Virol. 2020 Apr;92(4):418-423. doi: 10.1002/jmv.25681. Epub 2020 Feb 7. Erratum in: J Med Virol. 2020 Oct;92(10):2249. doi: 10.1002/jmv.26234.

Ugurel OM, Ata O, Turgut-Balik D. An updated analysis of variations in SARS-CoV-2 genome. Turk J Biol. 2020 Jun 21;44(3):157-167. doi: 10.3906/biy-2005-111.

Kang S, Yang M, Hong Z, Zhang L, Huang Z, Chen X, He S, Zhou Z, Zhou Z, Chen Q, Yan Y, Zhang C, Shan H, Chen S. Crystal structure of SARS-CoV-2 nucleocapsid protein RNA binding domain reveals potential unique drug targeting sites. Acta Pharm Sin B. 2020 Jul;10(7):1228-1238. doi: 10.1016/j.apsb.2020.04.009.

Domańska-Blicharz K, Woźniakowski G, Konopka B, Niemczuk K, Welz M, Rola J, Socha W, Orłowska A, Antas M, Śmietanka K, Cuvelier-Mizak B. Animal Coronaviruses in the Light of COVID-19. J Vet Res. 2020 Aug 2;64(3):333-345. doi: 10.2478/jvetres-2020-0050.

Ward JM, Munn RJ, Gribble DH, Dungworth DL. An observation of feline infectious peritonitis. Vet Rec. 1968 Oct 19;83(16):416-7. doi: 10.1136/vr.83.16.416.

Decaro N, Buonavoglia C. An update on canine coronaviruses: viral evolution and pathobiology. Vet Microbiol. 2008 Dec 10;132(3-4):221-34. doi: 10.1016/j.vetmic.2008.06.007.

Buonavoglia C, Decaro N, Martella V, Elia G, Campolo M, Desario C, Castagnaro M, Tempesta M. Canine coronavirus highly pathogenic for dogs. Emerg Infect Dis. 2006 Mar;12(3):492-4. doi: 10.3201/eid1203.050839.

Sanchez-Morgado JM, Poynter S, Morris TH. Molecular characterization of a virulent canine coronavirus BGF strain. Virus Res. 2004 Aug;104(1):27-31. doi: 10.1016/j.virusres.2004.02.038.

Alfano F, Fusco G, Mari V, Occhiogrosso L, Miletti G, Brunetti R, Galiero G, Desario C, Cirilli M, Decaro N. Circulation of pantropic canine coronavirus in autochthonous and imported dogs, Italy. Transbound Emerg Dis. 2020 Sep;67(5):1991-1999. doi: 10.1111/tbed.13542.

Shiba N, Maeda K, Kato H, Mochizuki M, Iwata H. Differentiation of feline coronavirus type I and II infections by virus neutralization test. Vet Microbiol. 2007 Oct 6;124(3-4):348-52. doi: 10.1016/j.vetmic.2007.04.031.

Terada Y, Matsui N, Noguchi K, Kuwata R, Shimoda H, Soma T, Mochizuki M, Maeda K. Emergence of pathogenic coronaviruses in cats by homologous recombination between feline and canine coronaviruses. PLoS One. 2014 Sep 2;9(9):e106534. doi: 10.1371/journal.pone.0106534.

Jaimes JA, Millet JK, Stout AE, André NM, Whittaker GR. A Tale of Two Viruses: The Distinct Spike Glycoproteins of Feline Coronaviruses. Viruses. 2020 Jan 10;12(1):83. doi: 10.3390/v12010083.

Jaimes JA, Whittaker GR. Feline coronavirus: Insights into viral pathogenesis based on the spike protein structure and function. Virology. 2018 Apr;517:108-121. doi: 10.1016/j.virol.2017.12.027.

Vlasova, A. N., Wang, Q., Jung, K., Langel, S. N., Yashpal Singh Malik, & Saif, L. J. (2020). Porcine Coronaviruses. Livestock Diseases and Management, 79–110. https://doi.org/10.1007/978-981-15-0402-0_4.

Zhang X, Zhu Y, Zhu X, Chen J, Shi H, Shi D, Dong H, Feng L. ORF3a deletion in field strains of porcine-transmissible gastroenteritis virus in China: A hint of association with porcine respiratory coronavirus. Transbound Emerg Dis. 2017 Jun;64(3):698-702. doi: 10.1111/tbed.12634.

Chen F, Knutson TP, Rossow S, Saif LJ, Marthaler DG. Decline of transmissible gastroenteritis virus and its complex evolutionary relationship with porcine respiratory coronavirus in the United States. Sci Rep. 2019 Mar 8;9(1):3953. doi: 10.1038/s41598-019-40564-z.

Two consecutive proline substitutions in the fusion peptide of swine acute diarrhea syndrome coronavirus spike protein reduce cell-cell fusion. (2023). Journal of Preventive Veterinary Medicine, 47(4), 185–189. https://doi.org/10.13041/jpvm.2023.47.4.185

Ma T, Xu L, Ren M, Shen J, Han Z, Sun J, Zhao Y, Liu S. Novel genotype of infectious bronchitis virus isolated in China. Vet Microbiol. 2019 Mar;230:178-186. doi: 10.1016/j.vetmic.2019.01.020.

Zhou H, Zhang M, Tian X, Shao H, Qian K, Ye J, Qin A. Identification of a novel recombinant virulent avian infectious bronchitis virus. Vet Microbiol. 2017 Feb;199:120-127. doi: 10.1016/j.vetmic.2016.12.038.

Lam TT, Jia N, Zhang YW, Shum MH, Jiang JF, Zhu HC, Tong YG, Shi YX, Ni XB, Liao YS, Li WJ, Jiang BG, Wei W, Yuan TT, Zheng K, Cui XM, Li J, Pei GQ, Qiang X, Cheung WY, Li LF, Sun FF, Qin S, Huang JC, Leung GM, Holmes EC, Hu YL, Guan Y, Cao WC. Identifying SARS-CoV-2-related coronaviruses in Malayan pangolins. Nature. 2020 Jul;583(7815):282-285. doi: 10.1038/s41586-020-2169-0.

Yang YL, Yu JQ, Huang YW. Swine enteric alphacoronavirus (swine acute diarrhea syndrome coronavirus): An update three years after its discovery. Virus Res. 2020 Aug;285:198024. doi: 10.1016/j.virusres.2020.198024.

Boley PA, Alhamo MA, Lossie G, Yadav KK, Vasquez-Lee M, Saif LJ, Kenney SP. Porcine Deltacoronavirus Infection and Transmission in Poultry, United States1. Emerg Infect Dis. 2020 Feb;26(2):255-265. doi: 10.3201/eid2602.190346.

Puelles VG, Lütgehetmann M, Lindenmeyer MT, Sperhake JP, Wong MN, Allweiss L, Chilla S, Heinemann A, Wanner N, Liu S, Braun F, Lu S, Pfefferle S, Schröder AS, Edler C, Gross O, Glatzel M, Wichmann D, Wiech T, Kluge S, Pueschel K, Aepfelbacher M, Huber TB. Multiorgan and Renal Tropism of SARS-CoV-2. N Engl J Med. 2020 Aug 6;383(6):590-592. doi: 10.1056/NEJMc2011400.

Belser JA, Katz JM, Tumpey TM. The ferret as a model organism to study influenza A virus infection. Dis Model Mech. 2011 Sep;4(5):575-9. doi: 10.1242/dmm.007823.

M Najimudeen S, H Hassan MS, C Cork S, Abdul-Careem MF. Infectious Bronchitis Coronavirus Infection in Chickens: Multiple System Disease with Immune Suppression. Pathogens. 2020 Sep 24;9(10):779. doi: 10.3390/pathogens9100779.

Decaro N, Martella V, Saif LJ, Buonavoglia C. COVID-19 from veterinary medicine and one health perspectives: What animal coronaviruses have taught us. Res Vet Sci. 2020 Aug;131:21-23. doi: 10.1016/j.rvsc.2020.04.009.

Cook JK, Jackwood M, Jones RC. The long view: 40 years of infectious bronchitis research. Avian Pathol. 2012;41(3):239-50. doi: 10.1080/03079457.2012.680432.