Assignment

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**ORIGIN AND ETIOLOGY OF CORONAVIRUS (COVID-19)**

 The Coronavirus family causes illnesses ranging from the common cold to more severe diseases such as Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome(MERS) .

 They circulate in animals and some can be transmitted between animals and humans.Several coronaviruses are circulating in animals that have not yet infected humans. The new coronavirus, the seventh known to affect humans has been named COVID19.

An acute respiratory disease, caused by a novel coronavirus (SARS-CoV-2, previously known as 2019-nCoV), the coronavirus disease 2019 (COVID-19) has spread throughout China and received worldwide attention. On 30 January 2020, World Health Organization (WHO) officially declared the COVID-19 epidemic as a public health emergency of international concern. Meanwhile, several independent research groups have identified that SARS-CoV-2 belongs to β-coronavirus, with highly identical genome to bat coronavirus, pointing to bat as the natural host. The novel coronavirus uses the same receptor, angiotensin-converting enzyme 2 (ACE2) as that for SARS-CoV, and mainly spreads through the respiratory tract. Importantly, increasingly evidence showed sustained human-to-human transmission, along with many exported cases across the globe. The clinical symptoms of COVID-19 patients include fever, cough, fatigue and a small population of patients appeared gastrointestinal infection symptoms. The elderly and people with underlying diseases are susceptible to infection and prone to serious outcomes, which may be associated with acute respiratory distress syndrome (ARDS) and cytokine storm.

The SARS-CoV-2 is a β-coronavirus, which is enveloped non-segmented positive-sense RNA virus (subgenus *sarbecovirus*, *Orthocoronavirinae* subfamily) [[6](https://mmrjournal.biomedcentral.com/articles/10.1186/s40779-020-00240-0#ref-CR6)]. Coronaviruses (CoV) are divided into four genera, including α−/β−/γ−/δ-CoV. α- and β-CoV are able to infect mammals, while γ- and δ-CoV tend to infect birds. Previously, six CoVs have been identified as human-susceptible virus, among which α-CoVs HCoV-229E and HCoV-NL63, and β-CoVs HCoV-HKU1 and HCoV-OC43 with low pathogenicity, cause mild respiratory symptoms similar to a common cold, respectively. The other two known β-CoVs, SARS-CoV and MERS-CoV lead to severe and potentially fatal respiratory tract infections [[7](https://mmrjournal.biomedcentral.com/articles/10.1186/s40779-020-00240-0#ref-CR7)]. It was found that the genome sequence of SARS-CoV-2 is 96.2% identical to a bat CoV RaTG13, whereas it shares 79.5% identity to SARS-CoV. Based on virus genome sequencing results and evolutionary analysis, bat has been suspected as natural host of virus origin, and SARS-CoV-2 might be transmitted from bats via unknown intermediate hosts to infect humans. It is clear now that SARS-CoV-2 could use angiotensin-converting enzyme 2 (ACE2), the same receptor as SARS-CoV [[8](https://mmrjournal.biomedcentral.com/articles/10.1186/s40779-020-00240-0#ref-CR8)], to infect humans . The epidemic of unknown acute respiratory tract infection broke out first in Wuhan, China, since 12 December 2019, possibly related to a seafood market. Several studies suggested that bat may be the potential reservoir .

**CORONAVIRUS STRUCTURE**

The structure of SARS-CoV-2's "key" and the body's "lock" could theoretically provide a target for antiviral drugs that would stop the new coronavirus from getting into new cells. Most antiviral drugs already on the market focus on halting viral replication within the cell, so a drug that targeted viral entry would be new territory.

Even so, developing either drugs or a vaccine will be a challenging task. Treatments and vaccines not only have to prove effective against the virus, but must also be safe for people.

Coronaviruses are enveloped viruses with distinct virion morphology, displaying widely spaced, long petal-shaped spikes at the surface, that confer to the virus a crownlike appearance, origin of the name corona . The [viral envelope](https://www.sciencedirect.com/topics/immunology-and-microbiology/viral-envelope) contains a long helical [nucleocapsid](https://www.sciencedirect.com/topics/immunology-and-microbiology/virus-nucleocapsid) with single-, positive-stranded RNA 27–32 kb in size, the largest known viral-RNA genome.

The virions are spherical, with an envelope containing a prominent crown (‘corona’) of peplomers of S (spike) glycoprotein. HE (hemagglutinin), E (small envelope protein), and M (membrane glycoprotein). The genome is a positive-stranded RNA associated with the N (nucleocapsid phosphoprotein), composing the helical RNP (ribonucleoprotein).Each virion is about 50 to 200nm in diameter and has 4 structural proteins.The S (spike) E (Envelope) N (Nucleocapsid) M (Membrane) proteins S E And M make up the envelop while the N holds the RNA . The S protein helps the virus to attach to host cells and with action of some enzymes like ACE ,the ACE is the receptor for the virus

[Coronavirus](https://www.sciencedirect.com/topics/immunology-and-microbiology/coronavirinae) [RNA synthesis](https://www.sciencedirect.com/topics/immunology-and-microbiology/rna-synthesis) occurs in the cytoplasm via a negative-strand RNA intermediate . The viral RNA is capped at the 5′ end , where there is a ‘leader’ sequence followed by an UTR. At the 3′ end, there is a terminal UTR, followed by a poly(A) tail. The ORF 1 of the genomic RNA is translated into a polyprotein that is processed to yield the proteins that form the transcriptase–helicase complex. The genomic RNA is used as a template to synthesize negative-sense RNAs, which are in turn used to synthesize full-length genomic RNA and [subgenomic mRNAs](https://www.sciencedirect.com/topics/immunology-and-microbiology/subgenomic-mrna). The mRNAs direct translation of the [viral structural and nonstructural proteins](https://www.sciencedirect.com/topics/immunology-and-microbiology/viral-nonstructural-proteins). Progeny viruses assemble and bud in vesicles between the [endoplasmic reticulum](https://www.sciencedirect.com/topics/immunology-and-microbiology/endoplasmic-reticulum) and the Golgi apparatus, later released by [exocytosis](https://www.sciencedirect.com/topics/immunology-and-microbiology/exocytosis).

 **Pathogenesis**

The severe symptoms of COVID-19 are associated with an increasing numbers and rate of fatalities specially in the epidemic region of China. On January 22, 2020, the China National Health Commisson reported the details of the first 17 deaths and on January 25, 2020 the death cases increased to 56 deaths [[8](https://www.sciencedirect.com/science/article/pii/S0896841120300469%22%20%5Cl%20%22bib8)]. The percentage of death among the reported 2684 cases of COVID-19 was approximately 2.84% as of Jan 25, 2020 and the median age of the deaths was 75 (range 48–89) years [[8](https://www.sciencedirect.com/science/article/pii/S0896841120300469#bib8)].

Patients infected with COVID-19 showed higher leukocyte numbers, abnormal respiratory findings, and increased levels of plasma pro-inflammatory cytokines. One of the COVID-19 case reports showed a patient at 5 days of fever presented with a cough, coarse breathing sounds of both lungs, and a body temperature of 39.0 °C. The patient's sputum showed positive real-time polymerase chain reaction results that confirmed COVID-19 infection [[14](https://www.sciencedirect.com/science/article/pii/S0896841120300469%22%20%5Cl%20%22bib14)]. The laboratory studies showed leucopenia with leukocyte counts of 2.91 × 10^9 cells/L of which 70.0% were neutrophils. Additionally, a value of 16.16 mg/L of blood C-reactive protein was noted which is above the normal range (0–10 mg/L). High erythrocyte sedimentation rate and D-dimer were also observed [[14](https://www.sciencedirect.com/science/article/pii/S0896841120300469#bib14)]. The main pathogenesis of COVID-19 infection as a respiratory system targeting virus was severe pneumonia, RNAaemia, combined with the incidence of ground-glass opacities, and acute cardiac injury [[6](https://www.sciencedirect.com/science/article/pii/S0896841120300469%22%20%5Cl%20%22bib6)]. Significantly high blood levels of cytokines and chemokines were noted in patients with COVID-19 infection that included IL1-β, IL1RA, IL7, IL8, IL9, IL10, basic FGF2, GCSF, GMCSF, IFNγ, IP10, MCP1, MIP1α, MIP1β, PDGFB, TNFα, and VEGFA. Some of the severe cases that were admitted to the intensive care unit showed high levels of pro-inflammatory cytokines including IL2, IL7, IL10, GCSF, IP10, MCP1, MIP1α, and TNFα that are reasoned to promote disease severity [[6](https://www.sciencedirect.com/science/article/pii/S0896841120300469#bib6)].