



## Pathology and pathogenicity of SARS-CoV-2

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### ABSTRACT

*Acute respiratory distress syndrome (ARDS) is a severe form of acute respiratory distress SARS-CoV-2, also known as coronavirus illness of 2019, is a beta coronavirus that causes infectious respiratory disease (COVID-19). While substantial research has revealed fundamental information about COVID-19 clinical features, the illness pathophysiology remains unknown. The virus's Receptor-Binding Domain (RBD) binds with a high affinity to the human Angiotensin-Converting Enzyme-2 (ACE-2) receptor; according to structural analyses and biochemical assays; however, the mechanism is unknown. An overview of significant results in the pathophysiology of the SARS-CoV-2 virus, as well as major pathogenicity pathways, is addressed here. Studies shed further information on how to prevent further spread of the SARS-CoV-2 virus-caused COVID-19 illness, as well as how to treat it if a similar viral epidemic strikes in the future.*

**Keywords:** SARS-CoV-2, Coronavirus, COVID-19, pathology, pathogenicity

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### INTRODUCTION

In December 2019, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was initially detected in Wuhan, Hubei Province, China, and was later declared a pandemic in March 2020. SARS-CoV-2 shares 79.5 percent of the genetic sequence of the SARS-CoV coronavirus that triggered the 2002–2003 outbreaks, according to genomic characterization. All coronaviruses that have caused human illness (SARS-CoV, MERS-CoV, HKU1, HCoV-NL63, HCoV-OC43, and HCoV-229E) have had animal spillover; however, there is no evidence to support a zoonotic scenario for the origin of SARS-CoV-2 to yet. In addition to these viruses, research show that numerous additional known coronaviruses are circulating in animals but have not yet infected humans.

### Pathology

Coronaviruses, named after the crown-like protein spikes on their surface, are members of the Coronaviridae family of positive-sense, single-stranded RNA viruses. Coronaviridae are a family of infectious viruses that can cause illnesses ranging from minor respiratory symptoms to more serious disorders like the SARS-CoV-2 virus-caused 2019 new coronavirus disease (abbreviated as COVID-19). SARS-CoV-2, like SARS-CoV and MERS-CoV, is a developing respiratory beta coronavirus. The SARS-CoV-2 virus-caused COVID-19 illness is clinically defined by the patient experiencing flu-like symptoms such as fever, cough, and shortness of breath during the first 2 to 14 days of the viral incubation period, with droplets and close contact being the major mechanisms of transmission. As the COVID-19 illness progresses, older adults and persons of any age with severe underlying medical problems such as chronic lung disease and diabetes experience hypoxic respiratory failure and even death.

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The pathological abnormalities of SARS-CoV-2 in the COVID-19 patients' bodily systems remain unknown. The first findings obtained from investigations on clinical features of COVID-19 show that the effect of SARS-CoV-2 is not limited to the respiratory tract, but may also infect several bodily systems, necessitating a multidisciplinary approach to containing the pandemic. This portion of the study highlights the pathological aspects of human body systems (Table 1), as well as the findings of examination techniques used to confirm the diagnosis of SARS-CoV-2 virus-caused COVID-19 illness that have been described in part.

**Table 1.** SARS-CoV-2 virus pathological characteristics in the bodily systems of COVID-19 patients

Body system	Pathological features
Digestive	Fecal sample positive for SARS-CoV-2 RNA.
Immune	Lymphopenia and exacerbated inflammation.
Respiratory	In the lungs, pneumocyte injury is shown by the production of hyaline membranes, interstitial lymphocyte infiltration, and multinucleated syncytial cells.
Cardiovascular	Myocardial and blood vessels injury
Reproductive	Male patients had a lower blood testosterone to luteinizing hormone ratio.
Urinary	Proteinuria, high serum creatinine and blood urea nitrogen levels, tubular necrosis, luminal brush boundary sloughing, and vacuole degeneration are all symptoms of tubular necrosis.
Integumentary	Ischemic changes in the fingers and toes.
Skeletal	Joint problems such as arthritis
Nervous	Acute viral encephalitis, acute toxic encephalitis, and acute cerebrovascular illness are all examples of acute cerebrovascular disease.
Other	Alanine aminotransferase and aspartate aminotransferase levels in serum bilirubin are abnormal.

### Pathogenicity

The current reports describing the clinical features of COVID-19 illness have aided in a better understanding of the disease pathology in individual individuals' bodily systems. However, more research on the pathogenicity of the SARS-CoV-2 virus is needed to provide more knowledge into how to combat the COVID-19 pandemic or a similar viral epidemic in the future. The SARS-CoV-2 virus has a complicated pathogenicity profile, with several variables contributing to severe harm in the respiratory tract and other body systems. Coronaviruses have crown-shaped protein spikes on their surface, which distinguish them morphologically. The SARS-CoV-2 virus surface protein spikes contain a variable receptor-binding domain (RBD) that allows the virus to bind with a high affinity to the Angiotensin-Converting Enzyme-2 (ACE-2) receptor found in the human respiratory tract, lungs, gastrointestinal tract, kidneys, and heart, according to structural studies and biochemical experiments; however, the mechanism is unknown. The presence of type 2 transmembrane serine protease (TMPRSS2) in target cells, in addition to the ACE-2 receptor, has been hypothesized to aid SARS-CoV-2 viral entry, as it does in influenza and human metapneumovirus.

TMPS2 proteolysis to the complex produced when the SARS-CoV-2 RBD binds to the host ACE-2 receptor promotes cleavage of the host target cell receptor and activates the viral surface protein, to summarize. It may be premature to draw any conclusions about the aforementioned notions at this time; nonetheless, the presence of both the ACE-2 receptor and TMPRSS2 in the target cell determines the host's vulnerability to the SARS-CoV-2 virus. As the COVID-19 illness progresses, the SARS-CoV-2 virus impairs the human immune system, causing lymphopenia, a large loss in lymphocytes, and exacerbating inflammation.

### CONCLUSION

To summarize, there is currently no vaccine or specialized treatment available to prevent SARS-CoV-2 virus infection or to treat COVID-19 sickness. The fundamental information on COVID-19's clinical features has aided in the adoption of symptomatic treatment and oxygen therapy to improve the patient's immune function thus far. It's worth noting that the presence of the ACE-2 receptor and TMPRSS2 in the host target cell is required for SARS-CoV-2 viral pathogenicity. Advanced in vitro and in vivo investigations in the disciplines of SARS-CoV-2 virus pathogenicity are required to completely understand the pathogenesis of COVID-19 illness or if a comparable viral epidemic occurs in the future.

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