## **Pathology of Corona Virus**

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Editorial

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## **Description**

Coronavirus Disease 2019 (COVID-19) has become a serious ill health causing severe acute respiratory disease in humans. It has already spread rapidly around the globe since its first identification in Wuhan, China, in December 2019. The causative virus is known as Severe Acute Respiratory Syndrome Corona Virus 2 (SARS-CoV-2), and the World Health Organization (WHO) named the new epidemic disease Coronavirus Disease (COVID-19). The incidence of COVID-19 continues to extend with quite three million confirmed cases and over 244,000 deaths worldwide.

Therefore, within the absence of pharmaceutical interventions, the implementation of precautions and hygienic measures are going to be essential to regulate and to attenuate human transmission of the virus. Coronaviruses were distinguished as enveloped, single-stranded, positivestrand RNA viruses classified within the Nidovirales order. This coronavirus family consists of pathogens of many animal species and of humans, including the recently isolated Severe Acute Respiratory Syndrome Corona Virus 2 (SARS-CoV-2). This is divided into two main parts in which the first concerns the animal coronaviruses and their pathogenesis, with an emphasis on the functions of individual viral genes and the second consists of the newly described human emerging pathogen, SARS-CoV.

## **Pathogenesis**

Coronaviruses may infect many species of animals which also includes humans, causing acute and chronic diseases. MHV consists of strains, which provide models systems for the study of viral tropism and pathogenesis in several organs systems, including the central nervous system, the liver, and the lung, and has been cited as providing one among the few animal models for the study of chronic demyelinating diseases like MS.

The coronavirus spike protein is a type I glycoprotein which forms the peplomers on coronavirus particles. Some coronaviruses spikes are cleaved into two subunits by a furin-like enzymatic activity during processing in the Golgi. The prototype MHV spike is 180 kDa; for many MHV strains, it's cleaved into two noncovalently associated subunits of about 90 kDa. The amino-terminal S1 subunit, which is believed to make the globular head of the mature protein, contains a Receptor Binding Domain (RBD) within the primary 330 amino acids. The RBDs of HCoV-229E and SARS-CoV spikes are also found in S1, although not at the amino termini.

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Coronaviruses attach to a specific cellular receptor through the spike protein. The first identified coronavirus receptor was CEACAM 1, utilized by MHV. Viral attachment triggers a conformational change within the spike protein that promotes the fusion of viral and cellular membranes. While there are not any crystal structures available for any coronavirus spike, it's believed that it's going to undergo changes almost like those of other type I fusion proteins, such as influenza virus hemagglutinin and human immunodeficiency virus gp120, so as to mediate fusion of viral and cellular membranes.

The use of recombinant coronaviruses, including MHV, TGEV, and IBV, has definitively demonstrated that the spike is a major determinant of tropism and pathogenicity. In the case of TGEV, the replacement of the spike gene of an attenuated respiratory strain of TGEV with the spike gene from a virulent enteric strain renders the virus enterotropic. The JHM strain is very neurotropic, causing severe, usually fatal encephalitis and tiny if any hepatitis, while the A59 strain causes moderate hepatitis and is merely weakly neurovirulent.

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