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# Interaction of the Human Coronavirus with its Host

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## Description

Infection with the human coronavirus (HCoV) causes respiratory illnesses that range from mild to severe. In the last 15 years, two zoonotic, extremely pathogenic HCoVs have emerged: the severe acute respiratory syndrome coronavirus (SARS-CoV) and the Middle East respiratory syndrome coronavirus (MERS-CoV) (MERS-CoV). HCoV replication is influenced by a variety of host variables and results in significant changes in cellular structure and physiology. Activation of key signalling pathways during HCoV infection influences antiviral immune response induction and leads to HCoV pathogenesis. Some essential features of the complex HCoV-host relationship have begun to be revealed in mechanistic depth in recent research.

Coronaviruses are enclosed viruses with non-segmented, single-stranded RNA genomes that are positive-sense. Six coronaviruses have been shown to infect human hosts and cause respiratory disorders, in addition to infecting a range of economically important vertebrates (such as pigs and chickens). The zoonotic and extremely pathogenic coronaviruses severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) are among them, causing regional and global epidemics.

In many ways, the pandemic of the new severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has posed a serious threat to the world. Despite being in development, effective treatment and preventive measures, such as medicines and vaccinations, are currently unavailable. In the fight against SARS-CoV-2, a thorough understanding of the virus's life cycle and interactions with hosts is critical. The present state of SARS-CoV-2 research, as well as the epidemic condition and epidemiological characteristics of the induced disease COVID-19, were briefly summarised in this study. SARS-CoV-2 biology was also discussed, including the virus's origin, evolution, and receptor recognition mechanism. We discussed the protein structures of SARS-CoV-2 and the development of structure-based therapies such as antibodies, antiviral drugs, and vaccines, as well as the limitations and future prospects of SARS-CoV-2 research. We hope that the information in this assessment will be useful in the global fight against SARS-CoV-2 infection [1].

SARS-CoV-2 appears to be a natural reservoir in bats. According to one study, betacoronavirus isolated from pangolins shows a sequence resemblance to the currently infected human strain of up to 99 percent. SARS-CoV-2 and the coronavirus from a pangolin in Malaysia have a substantial genetic similarity, according to another study. In terms of E, M, N, and S genes, the similarity between these two viruses is 100, 98.6, 97.8, and 90.7 percent, respectively, implying that pangolins could be the intermediate host. Dogs, chickens, ducks, and pigs are among the animals that cannot tolerate illness when they are in close proximity to humans. In cats and ferrets, SARS-CoV-2 replicates well. The golden hamster can also transmit SARS-CoV-2.

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### The SARS-CoV-2 virus's structure

Coronaviruses are members of the Coronavirinae subfamily of the Coronaviridae family, and there are four genera in the subfamily: Alphacoronavirus, Betacoronavirus, Gammacoronavirus, and Deltacoronavirus. CoVs have a single-stranded positive-sense RNA (+ssRNA) genome that is larger than any other RNA virus (27–32 kb). Outside the genome, the nucleocapsid protein (N) forms the capsid, and the genome is further packed by an envelope that is made up of three structural proteins: membrane protein (M), spike protein (S), and envelope protein. The genome size of SARS-CoV-2, which was recently sequenced as a member of the coronavirus family, is around 29.9 kb. SARS-CoV-2 has sixteen non-structural proteins (nsp16) and four structural proteins (S, E, M, and N). Nsp1 is a protein that plays a role in RNA processing and replication. The domain Nsp14 is a proofreading exoribonuclease. Mn(2+)-dependent endoribonuclease activity is found in Nsp15. Nsp16 is a methyltransferase of 2'-O-ribose. According to one study, NSP has effects on splicing, translation, and protein trafficking that suppresses host defences. NSP16 interacts to the mRNA recognition domains of the U1 and U2 snRNAs to decrease mRNA splicing after infection with SARS-CoV-2. NSP1 binds to 18S ribosomal RNA in the ribosome's mRNA entry channel, interfering with mRNA translation. NSP8 and NSP9 bind to the 7SL RNA at the Signal Recognition Particle, causing protein trafficking to the cell membrane to be disrupted [2].

Remdesivir is an adenosine analogue that inhibits RdRp effectively. In vitro, Remdesivir was found to be effective at inhibiting SARS-CoV-2 replication. In cultured cells, nonhuman primate models, and mice, Remdesivir has broad-spectrum antiviral activity against RNA virus infection. Remdesivir operates as an adenosine analogue following virus entry, integrating into nascent viral RNA to stop replication before the RNA matures. Remdesivir is a prodrug of sorts. It would convert to the triphosphate form (RTP) and become active in target cells. Remdesivir, like other nucleotide analogue prodrugs, inhibits RdRp function by covalently binding the primer strand and terminating the RNA chain. The nsp12-nsp7-nsp8 complex takes on the role of RNA polymerase when ATP is added. The RNA polymerization activity would be greatly decreased if the active triphosphate form of remdesivir (RTP) was added. The apo RdRp's structure is made up of nsp12, nsp7, and nsp8. 14-base RNA in the template strand and 11-base RNA in the primer strand make up the template-RTP RdRp complex [3]. The remdesivir in the complex is in the monophosphate form (RMP). The primer strand, three magnesium ions, and a pyrophosphate are all covalently bound to the RMP. The three magnesium ions cluster close to the active site and aid catalysis. The RMP is found in the middle of the catalytic active site. The active site of the catalytic enzyme is made up of seven motifs. RMP and the base of the primer strand in the upstream have base-stacking interactions. RMP and the template strand's uridine base have hydrogen bonds as well. Interactions between RMP and side chains are also present (K545 and R555). Nsp12 has twenty-nine residues that directly contribute in RNA binding. The RNA interactions are not mediated by any residue from nsp7 or nsp8. Favipiravir, like remdesivir, is an inhibitor of the RdRp. Favipiravir has a structure that is similar to endogenous guanine. As the first anti-SARS-CoV-2 chemical tested in China, favipiravir shown to have few negative side effects [4,5].

N3, a mechanism-based inhibitor discovered by computer-aided drug design, may fit inside the major protein's substrate-binding pocket and is a powerful irreversible inhibitor of the main protein. Two Mpro-N3 complexes join together to form a dimer (the two complexes are named protomer A and protomer B, respectively). Domain IIII refers to the three domains found in each protomer. Domain I and domain II both feature a -barrel structure that is antiparallel. Domain III has five -helices that interact in an antiparallel fashion

to generate a globular cluster structure. A lengthy loop connects domain III to domain II. The substrate binding site is located in the gap between domains I and II.

## Conflict of Interest

None.

## References


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