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Editorial

The Role of Molecular Recognition in Covid-19 Infection, Replication, and Transmission

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Introduction

The halo or crown structure of proteinaceous spike peplomers or Glycoproteins (SGPs) detected in a transmission electron microscope image is the basis for the name Coronavirus (CoV), which is given to viruses that cause a variety of respiratory illnesses, such as COVID-19 (coronavirus disease of 2019), SARS (severe acute respiratory syndrome), and MERS (measles, encephalitis, and respiratory syndrome) (Middle East respiratory syndrome). SARS-CoV-2 has a shape, size (80 nm-120 nm), genome, and RNA-based pathogenesis similar to other CoVs. The extremely pathogenic character of SARS-CoV-2 and its recent genetic variants suggests that these infections have high binding affinities for a host cell and can successfully circumvent or inhibit cytokine (interferon (IFN))-triggered immune responses. As a result, we must address the following fundamental problems about SARS-CoV-2 tropism, replication, and release/transmission. How can SARS-CoV-2 adapt to the specific SGP genes that add a Furin Cleavage Segment (FCS) to the virus, allowing it to detect and attach to the Angiotensin-Converting Enzyme 2 Receptor (ACE2R) efficiently? Is Hemagglutinin (HA) a coreceptor for attaching to a host cell via sialic acid (Sia)? How does cleavage by Neuraminidase (NASe) or Esterase (ES) release a progeny virion? How do non-structural proteins, Nuclear Capsid (NC) and other structural proteins, and RNA generate progeny virions by bypassing the IFN-Induced Janus-Activated Kinase-Signal Transducer and Activator of Transcription (JAK-STAT) mechanism? SARS-CoV-2 was initially linked to Rhinolophus affinis, a bat species, according to a 96 percent sequence similarity between SARS-29.9 CoV-2's kb RNA and the RNA of the RaTG13 virus in R. affinis.

The similarities between the SGP amino acids of SARS-CoV-2 and Malayan pangolin CoV (pangolin-CoV) attracted further attention to the studies on the chimeric nature of the viral RNA and the origin of SARS-CoV-2.

Independent of the suggested lineage relationships between RaTG13, SARS-CoV-2, and Pangolin-CoV, investigations have validated the zoonotic evolution of SARS-CoV-2. An emerging and serious concern about COVID-19 is the propagation of SARS-CoV-2 RNA mutations in humans or animals, particularly during the development of an effective drug or vaccine against COVID-19.

Molecular Recognition

Pharmaceutical behemoths and private and public research organizations in Russia, the United States, the United Kingdom, India, Japan, China, Italy, Spain, Belgium, Germany, Australia, Singapore, and Israel began developing drugs and vaccines against COVID-19 without taking into account an RNA virus's ability to bypass antiviral drugs or modify its own genome to overcome the host's innate or adaptive immunity. SARS-CoV-2 pathogenesis is similar to influenza viruses, Human Immunodeficiency Virus (HIV), Ebola virus, SARS-CoV, and MERS-CoV in the early episodes of COVID-19.

Various antiviral drugs, such as Tamiflu and favipiravir, which are used to treat common influenza viruses; lopinavir and ritonavir, which are used to treat HIV; and remdesivir, which is used to treat Ebola virus, Marburg virus, Lass virus, Syncytial virus, Nipah virus, Junin virus, Hendra virus, and CoVs that cause SARS and MERS, have been tested in COVI Chloroquine, mefloquine, hydroxychloroquine (HCQ),

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artemisinin, clindamycin, doxycycline, and pyrimethamine were also tested in clinical trials against the malaria-causing Plasmodium falciparum (P. falciparum).

The positive and negative results of these assays signal tropism switching and virion genomic change. The reduction of RNA copying by avoiding exoribonuclease proofreading is one of the continuing treatments for a COVID-19 patient, for which the blocking of endolysosomal transport of the virus-encapsulated endosome is under contention. Furthermore, the success of remdesivir in inhibiting Ebola virus by altering its RNA is linked to the drug's success in treating the first COVID-19 case in the United States. However, negative effects of these medications, such as heart dysfunction in patients treated with chloroquine derivatives, should be carefully considered during COVID-19 management.

Molecular-level knowledge about the infection, immune response, replication, and transmission of SARS-CoV-2 is unavoidable for the development of a successful vaccine or treatment, in addition to the aforementioned testing. Because of the infection's complexity, a new platform at the intersection of virology, immunology, drug delivery, genetics, chemistry, materials science, and nano science is needed to assist researchers, physicians, and technologists collaborate and produce COVID-19 vaccines and therapies. It is critical to make effective use of the amount of knowledge available on nano materials, imaging probes, bio imaging techniques, vaccine development, and drug/gene/nanomaterial delivery in vitro and in vivo. Nonetheless, when considering virus mimicking nano viruses for in vivo applications, the toxicity of nano materials is a key problem. This article highlights the fundamental components of viral infection and host immune response molecular connections, as well as future prospects for the aforementioned interface in the pandemic battle.