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Commentary

Current Challenges in COVID-19 Clinical Trails

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DESCRIPTION

The 2019 Coronavirus Disease (COVI-19) pandemic is already having a major impact. The pandemic has been rampant around the world for a year, with more than 150 million confirmed human infections and more than 3 million deaths. The genomic sequence of the associated pathogen SARSCoV2 (Severe Acute Respiratory Syndrome Coronavirus 2) was rapidly determined, but unknown aspects such as the origin and evolutionary trends of the virus and the efficacy of the drug against current vaccines and mutant viruses.

There are still many. This overview summarizes current knowledge and advances in COVID-19, including viral origins, infections, and infections, with the aim of gaining a better understanding of COVID-19 and providing new perspectives for future research. The advent of COVID19 is a breakthrough in human history, regardless of whether SARSCoV2 can be completely eliminated like SARSCoV or becomes a seasonal epidemic of the population like other human infectious coronaviruses.

So far, the scientific knowledge gained in response to this pandemic will help us better understand SARSCoV2 and related diseases and help manage and prevent future emerging infectious diseases.

Many scientists believed that identifying the "original host animal" was essential to contain the COVID-19 pandemic and prevent future pandemics. However, current research on the origin of the virus is still unknown. Therefore, this step is a decisive factor in the efficiency of viral entry and host orientation. It has been reported that SARSCoV1 and MERSCoV invade host cells *via* endocytosis and require cathepsin proteolysis of S proteins in endosomes to induce membrane fusion. SARSCoV2 can use similar mechanisms for cell invasion and membrane fusion.

Compared to the SARSCoV1 S protein, the unique properties of The SARSCoV2 S protein can result in differences in receptor binding capacity. This may partially explain its higher infectivity than other human coronaviruses. In addition to ACE2, other putative receptor molecules including CD209 (differentiation cluster 209) and CLEC4M (c-type lectin domain, family 4, member M) and neuropil in 1 have also been proposed. These molecules also indicate potential targets for antiviral intervention. These facts also suggest that the mechanism of SARSCoV2 infection is not fully understood. Many other questions remain unanswered, such as whether other receptors/factors are involved. Non-protein genic factors such as fatty acids can also play an important role in the interaction between the viral S protein and host receptors and should not be overlooked.

The clinical symptoms of SARSCoV2 are similar to those of SARSCoV1. The main organ of the viral infection is the lungs, and patients can develop Acute Respiratory Distress Syndrome (ARDS), which can lead to respiratory failure and even death. In particular, in addition to respiratory pathology, some clinical cases also showed other clinical symptoms. More and more clinical studies have shown that SARSCoV2 not only attacks the lungs, but also damages other organs in the human body, especially in critically ill patients. SARSCoV2 can directly infect extra pulmonary organs that express ACE2 and TMPRSS2.

In addition, SARSCOV2 infection can cause the following unexpected complications:

SARSCoV2 can also break the blood-brain barrier and invade the central nervous system by attacking the vascular system, causing some neurological complications such as Spinal Cord Injury (SCI). It has also been reported that about 10% of patients have gastrointestinal symptoms such as diarrhea.

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