

## RdRp and SARS-CoV-2

Alfio Ferlito\*

Department of Surgical Sciences, University of Udine School of Medicine, Udine, Italy

\*Correspondence to: Alfio Ferlito, Department of Surgical Sciences, University of Udine School of Medicine, Udine, Italy; E-mail: alfio.ferlito@uniud.it

Received: November 02, 2020; Accepted: November 16, 2020; Published: November 23, 2020

Copyright: © 2020 Ferlito A. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Editorial Note

Coronavirus makes changes in body cells that cause cells not to recognise it. The resulting viral messenger RNA is now considered as part of the cell's own code and not foreign due the modifications which deceive the cell. The transmission mechanisms of COVID-19 include the infusion of the virus inside the body causes the immune defenses to a "cytokine storm", an over-activation of white blood cells, which release too-great amounts of cytokines into the blood.

Human coronaviruses are made up of various non-structural proteins like the proteases nsp3 and nsp5, as well as RdRp. They are designed with an efficient proofreading mechanism that can usually discard unwanted nucleoside analogs. RNA-dependent RNA polymerase (RdRp) occurs within the RNA genome of viruses, and is a highly flexible enzyme that assists in RNA synthesis by catalyzing the phosphodiester bond-dependent RNA-template formation. Inhibitors of RdRp have also been studied and shown remarkable performance in the reduction of SARS-CoV-2 as they have been found to enhance their antiviral activity by overcoming this proofreading process.

The mutants can survive harsh environmental factors as the mutation was caused under extreme pressure by physiological defense of the host organisms for these viruses. Increased mutation rates support the population of RNA virus as they are considered to be harmful to host cells leading to the progeny of lethal and resistant virus without the interference from immune cells of organisms.

RdRp inhibitors have been studied and unique progress in reducing SARS-CoV-2 has been demonstrated due to the lack of exonuclease activity during the RNA synthesis process results in a high copying error rate for RdRps, which is estimated to be around  $10^{-4}$ .

The RdRp inhibitor was successfully used to treat SARS-CoV-2 and prevented the subjects from witnessing any of the classic symptoms associated with this respiratory disorder as the test subjects administering the RdRp inhibitor had decreased pulmonary infiltrates on their radiographs and reduced virus titers in the obtained bronchoalveolar lavage samples.

The use of several different RdRp inhibitors in the treatment of the novel SARS-CoV-2 continues to show promising results. A nanoLCMS/MS acquisition method was developed to detect SARS-CoV-2 marker peptides in nasopharyngeal swabs spiked with different quantities of purified SARS-CoV-2 viral content using the tandem mass spectrometry detected the microbiota signal present in these samples is small and can be excluded when interpreting proteomic shotgun data obtained from a restricted peptidome landscape window. Further research is required to explore the mechanisms responsible for reducing the antiviral activity of SARS-CoV-2. The therapeutic importance of the prevention of RdRp activity has contributed to the development of many inhibitors that have been used to treat a wide variety of RNA viruses including Ebola, the human immunodeficiency virus (HIV) and Zika virus.