Journal of Clinical Respiratory: Open Access

Regulation of Angiotensin-Converting Enzyme 2 in COVID-19 Patients

Hala Ahmed*

Department of Rhinology, University of Mohaghegh Ardabili, Ardebil, Iran

Commentary

Received: 1-Aug-2022, Manuscript No. JCROA-22-66479; **Editor assigned:** 03-Aug-2022, Pre QC No. JCROA-22-66479 (PQ); **Reviewed:** 17-Aug-22, **QC No**. JCROA-22-66479;
Revised: 24-Aug-2022, **Manuscript No.** JCROA-22-66479 (R); **Published:** 31-Aug-2022, DOI: 10.4172/jclinresp.4.S1.002

***For Correspondence:**
Hala Ahmed, Department of Rhinology, University of Mohaghegh Ardabili, Ardebil, Iran

E-mail: halahmed@gmail.com

ABOUT THE STUDY

Derived from previous SARS-CoV structural analyses of atomic-level interactions between the Receptor-Binding Domain (RBD), which is a key part of a virus located on its spike domain that allows it to dock to body receptors to gain entry into cells and lead to infection, ACE2 was predicted to be the receptor for SARS-CoV-2 in the current pandemic in January 2020. SARS-CoV-2 accesses host cells through a functional receptor on cell surfaces called Angiotensin-Converting Enzyme 2 (ACE2), which is abundantly expressed in the heart, kidneys, and lungs and released into plasma. The Renin-Angiotensin-Aldosterone System is regulated by ACE2 (RAAS). SARS-CoV-2 disrupts the ACE/ACE2 balance and activates the RAAS, leading to COVID-19 development, particularly in individuals with comorbidities such hypertension, diabetes, and cardiovascular disease. As a result, ACE2 expression could have contradictory consequences, boosting SARS-CoV-2 pathogenicity while also reducing viral infection.

The recently released SARS-CoV-2 genomic sequence with RBD and ACE2. These are also key targets in the prevention and treatment of viral infections, such as COVID-19, the coronavirus that causes Severe Acute Respiratory Syndrome (SARS-CoV-2). Researchers then published studies on viral infectivity in HeLa cells in early February. The next step in determining SARS-route CoV-2's of entrance was the release of the solved structure of the SARS-CoV-2 S protein, which was published online in mid-February 2020. Cryo-EM was used to illustrate the prefusion configuration of the SARS-CoV-2 trimeric S protein with one of the three S1 RBDs in the receptor-

accessible up-conformation. These results reveal that when the S1 subunit interacts to its receptor, the prefusion conformation rearranges, resulting in the S1 subunit shedding and the remaining S2 subunit placement in a stable postfusion shape, as expected from studies of other class I viral membrane fusion proteins. Because of the great structural similarities between SARS-CoV and SARS-CoV-2, the cryo-EM structure added to the growing evidence that ACE2 is the host receptor for SARS-CoV-2. The S protein of SARS-CoV-2 binds ACE2 with a 20-fold higher affinity than SARS-CoV, according to non-imaging data. Other studies, on the other hand, discovered that SARS-Cov-2 and SARS-CoV have identical binding affinities for hACE2 SARS-CoV-2 can enter lung cells attributable to the ACE2 receptor where the virus, which is cell-free and phagocytosed by macrophages, can travel to other organs and infect ACE2-expressing cells at locals, resulting in multi-organ damage.

Viral penetration into host cells is the first step in viral infection to spike glycoprotein on the coronavirus's envelope can attach to specific receptors on host cell membranes. Previous research has revealed that ACE2 is a functional receptor for SARS-CoV. Fever, dry cough, myalgia, tiredness, and dyspnea are the most prevalent COVID-19 symptoms. Sputum production, headache, stomach discomfort, diarrhoea, nausea, vomiting, dizziness, anosmia, dysgeusia, and liver function abnormalities have also been recorded, which could be explained by the fact that SARS-CoV-2 targets multiple ACE2-expressing tissues. During the COVID-19, ACE2 expression was found in Type II Alveolar cells (AT2), bronchial transient epithelial secretory cells, respiratory epithelial cells, myocardial cells, endothelial cells, and artery smooth muscle cells, esophageal epithelial cells, neurons and glia, tongue epithelial cells, stomach, cholangiocytes, adipose tissue, pancreatic exocrine glands and islets

In conclusion, the RBD is a key component of the viral spike glycoprotein seen on coronaviruses like SARS-CoV-2, which produces COVID-19. The RBD's attachment to the spike domain is a crucial stage in coronavirus infection because it permits the virus to bind to target body receptors (such as ACE2 on respiratory epithelial cells) and enter cells.