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Short Communication

Relationship between Coronavirus and Angiotensin-Converting Enzyme 2 (ACE2)

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DESCRIPTION

The SARS-CoV-2 spike glycoprotein, which binds to Angiotensin-Converting Enzyme 2 (ACE2), could be a target for therapeutic development, antibodies, and vaccines. ACE2, a homolog of Angiotensin-Converting Enzyme (ACE), is found in a number of human organs and tissues and has a wide range of biological activities, including the ability to counteract the detrimental effects of the renin-angiotensin system (RAS) in a variety of disorders. ACE2 is largely expressed on type II alveolar epithelial cells in the respiratory system, but weakly expressed on the surface of epithelial cells of the oral and nasal mucosa and nasopharynx, indicating that the lungs are the primary target of SARS -CoV-2 [1-5].

The ACE2 receptor allows SARS-CoV-2 to enter lung cells. It is possible that restoring the balance between the RAS and ACE2 will help prevent organ damage. ACE2 is observed in various degrees in nearly all human organs. The virus, which is cell-free and phagocytosed by macrophages, can travel to other organs and infect ACE2-expressing cells at local locales, resulting in multi-organ damage. The same receptor, angiotensin-converting enzyme 2, is used by both 2019-nCoV and SARS-CoV to enter the host cell of ACE2. The RAS is a complex network that affect the functioning of numerous organs, including heart, blood vessels, and kidneys, and plays an important role in maintaining blood pressure, electrolyte, and fluid balance. By lowering the amount of Ang II and raising the amount of Ang (1–7), ACE2 modulates the ACE action. As a result, ACE/ACE2 is required for tissue liquid volume and electrolyte homeostasis. Tissue tolerance to COVID-19 may be increased by ACE2 expression and its regulation by circumstances and underlying comorbidities. SARS-CoV-2 also affects the ACE/ACE2 physiological balance and activates the Ang II/AT1R pathways, resulting in serious clinical consequences. The most typical bioactive peptide in the RAS, angiotensin II (Ang-II), plays a key role in the advancement of cardiovascular disorders such as hypertension, myocardial infarction, and heart failure. The SARS-CoV-2 spike glycoprotein has a 10- to 20-fold stronger binding affinity for ACE2 than SARS-CoV. After binding, the virus's membrane merging with the host cell is initiated, and viral RNA is released into the cytoplasm, resulting in infection. Infection with SARS-CoV demands intact ACE2 or its Trans membrane domain to be absorbed with the virus. For entrance into host cells, both SARS-CoV-2 and SARS-CoV employ the ACE2 receptor. The ACE2 counteracts the adverse effects of the Renin-Angiotensin System (RAS), which plays crucial roles in maintaining the body's physiological and pathophysiological equilibrium. Because ACE2 is highly expressed in a variety of organs and tissues, SARS-CoV-2 affects not only the lungs, but also other organs with high ACE2 expression. COVID-19 pathophysiology is complicated, involving a number of variables. Studying about ACE2 can help to control various diseases as well as new treatment can be discovered with great abilities.

CONCLUSION

SARS-spike CoV-2's glycoprotein could be a target for the development of particular medicines, antibodies, and vaccines. In COVID-19, restoring the balance between the RAS and ACE2 should help reduce organ damage. The disruption of the local equilibrium between the RAS and the ACE2 axis caused by ACE2 down regulation in organs following virus infection may be linked to organ damage. ACE2 down regulation and the imbalance between the RAS and ACE2 after infection may further contribute to multiple organ injury in COVID-19, in addition to the immediate viral impacts and inflammatory and immunological factors associated with COVID-19 pathogenesis.

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