



Georgian National Association of Medical Rehabilitation and Sports Medicine



OPINION

Angiotensin converting enzyme 2 (ACE2) is an enzyme attached to the outer surface (cell membranes) of cells in the lungs, arteries, heart, kidney, and intestines. ACE2 lowers blood pressure by catalysing the cleavage of angiotensin II (a vasoconstrictor peptide) into angiotensin (1–7) (a vasodilator) .ACE2 also serves as the entry point into cells for some coronaviruses. The human version of the enzyme is often referred to as hACE2.

ACE2 counters the activity of the related angiotensin-converting enzyme (ACE) by reducing the amount of angiotensin-II and increasing Ang (1-7) making it a promising drug target for treating cardiovascular diseases.

Protein modeling experiments on the spike protein of the virus soon suggested that SARS-CoV-2 has sufficient affinity **to the receptor angiotensin converting enzyme 2** (ACE2) on human cells to use them as a mechanism of cell entry By January 22, 2020, a group in China working with the full virus genome and a group in the United States using reverse genetics methods independently and experimentally demonstrated that ACE2 could act as the receptor for SARS-CoV-2. Studies have shown that SARS-CoV-2 has a higher affinity to human ACE2 than the original SARS virus strain. SARS-CoV-2 may also use basigin to assist in cell entry.

As a transmembrane protein, ACE2 serves as the main entry point into cells for some coronaviruses, including HCoV-NL63; SARS-CoV (the virus that causes SARS); and SARS-CoV-2 (the virus that causes COVID-19). More specifically, the binding of the spike S1 protein of SARS-CoV and SARS-CoV2 to the enzymatic domain of ACE2 on the surface of cells results in endocytosis and translocation of both the virus and the enzyme into endosomes located within cells. This entry process also requires priming of the S protein by the host serine protease TMPRSS2, **the inhibition of which is under current investigation as a potential therapeutic.**

Angiotensin-converting enzyme-a glycoprotein, is present mainly in the lungs and in small quantities in the brush border of the epithelium of the proximal tubule of the kidneys, blood vessel endothelium and blood plasma. ACE catalyzes the conversion of angiotensin 1 to angiotensin 2 (**one of the most powerful vasoconstrictors**) and **hydrolyzes the bradykinin vasodilator to an inactive peptide.**

Coming out of the above information and our discussion, we have the opinion that the disease does not occur in the pathogenesis of microcirculatory pulmonary kalapot and the bradykinin system predominates on the abortion and in the acini (elementary link of the lungs) the microvasculature of the vessels exudes a powerful dilatation.

As a result, the physiological pressure in the capillaries and venules of the lungs drops, **which leads to gas bouts and oxygen diffusion. tissue supply with oxygen and nutrients is disrupted.**

It is logical that there is a total dysfunction of the physiological normal parameters of organs and systems of the body.



Sincerely

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