Letter to Editor

Ambient Temperature Interferes with COVID-19

Dear Editor,

Angiotensin-converting enzyme 2 (ACE2) is a blood pressure regulating enzyme that attaches to the outer surface in cells of the lungs, arteries, kidney, heart, and intestines.[1] In addition to blood pressure regulating, ACE2 is involved in the pathophysiological processes by converting angiotensin isozymes during cell injury.[2] ACE2 converts angiotensin II to angiotensin-(1-7).[3] ACE2 also is a receptor for some coronaviruses including human coronavirus NL63 (HCoV-NL63), severe acute respiratory syndrome-associated coronavirus (SARS-CoV), and SARS-CoV-2, the infectious agent of COVID-19 (coronavirus Disease 2019) worldwide.[4] Many hypotheses have been made about COVID-19. Underlying diseases such as hypertension, diabetes, and cancer, and environmental factors such as preventative methods and ambient temperature have been suggested to be involved into outbreak of COVID-19. Because of the unclear treatment results and the panic following COVID-19, some news about prevention and treatment strategies have been quickly spread among people through virtual networks. One of these news is the effect of heat and ambient temperature on the contagion of SARS-CoV-2. This paper has surveyed the scientific possibility of intervention of ambient temperature on COVID-19.

Previous studies have shown that cells in the human body biosynthesize heat shock proteins when exposed to adverse environmental conditions such as low and high temperatures.[5] Heat shock protein contributes to the robustness and survival of cellular protein structures by its chaperone function.[5] Previous studies show that heat shock protein 72 (HSP72) is expressed in human cells under changes of temperature out of physiologic ranges.[5] In regards to COVID-19, HSP72 increases the gene expression of ACE2 as the SARS-CoV-2 virus receptor.[4]

On the other hand, ACE2 converts angiotensin II to angiotensin-(1-7) that affects phosphorylation and activation of AKT, AMPK, and Sirt1 molecules through Mas membrane receptors.[6] Mentioned proteins activate the phospho-endothelial nitric oxide synthase that increases intracellular nitric oxide.[2,3] After binding the SARS-CoV-2 virus to ACE2, because of decreased ACE2 levels, angiotensin II is not converted to angiotensin-(1-7) (1 and 3). Following the decreased level of angiotensin-(1-7), nitrogen radical production, and viral genome degradation will cease.[7] On the other hand, in ACE2 deficiency conditions, the cellular signaling between angiotensin II and angiotensin-(1-7) will create an imbalance toward angiotensin II signaling.[1] Angiotensin II increases inflammation response by producing inflammatory cytokines and reactive oxygen species, which causes pneumonia and cell death.[1]

In conclusion, staying in a hot environment, such as the dry and wet sauna, or cold environment such as cold water pool for at least 15 min can cause stress shock in the cells and gene expression of HSP72 (3 and 6). HSP72 increases the ACE2 gene expression, as the receptor of SARS-COV-2 virus, inflammation, cell death, and finally pneumonia.[6] Therefore, avoiding of stress shocks such as heat and cold is recommended to reduce the risk of SARS-COV-2 infection.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Received: 20 Apr 20 **Accepted:** 08 May 20
Published: 22 Jul 20

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Access this article online

Quick Response Code:

Website: www.ijpvmjournal.net/www.ijpm.ir

DOI: 10.4103/ijpvm.IJPVM_204_20

How to cite this article: Ahmadi Hedayati M. Ambient temperature interferes with COVID-19. Int J Prev Med 2020;11:109.