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LETTER

Ibuprofen has Synergism with SARS-CoV-2 Infection

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Although the outbreak of viral infections among human societies is not an unknown phenomenon, but there is a growing terror due to a lack of drugs and fast global strategy against COVID-19 (Coronavirus disease 2019) [1]. There are many growing speculations about the causes of the fast outbreak, the pathology and fast death related to COVID-19. The last one was recent news from University Clinic of Vienna about possible synergism of SARS-CoV-2 RNA replication and consumption of ibuprofen in patients with high frequency of death consequence (data not published). It means Ibuprofen would be a colleague and synergism agent along with Severe Acute Respiratory Syndrome Coronavirus 2 (SARSCoV-2) on death of patients. Is that true?

Let's look at the brief description about the interaction between Ibuprofen and antiviral cell system. We know Ibuprofen as a non-steroids anti-inflammatory drug that inhibits activity of Cox1 and Cox2 enzymes [2, 3]. Cox1 and Cox2 enzymes convert acid arachidonic to prostaglandins after producing some Intermediate substrates [3]. Prostaglandins such as PGE2 andPGI2 progress the infected cells and immune system toward protection against infection [3]. So, consumption of non-steroidal anti-inflammatory drugs such as Ibuprofen in patients with COVID-19 can be due to intervention in cell defense mechanisms [4]. The point with a worth of mention is that Ibuprofen is a scavenger of Nitrogen radicals [5]. Nitrogen radicals are known to effect viral infection because of damages to viral genome [6].

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Ibuprofen on nitric oxide synthetase isoforms [7]. However, more evidence is needed, but it seems a decrease in the production of nitrogen radicals in infected cells with coronavirus can lead to an increase in viral RNA load in cells.

CONFLICT OF INTEREST

The author declares no conflict of interest, financial or otherwise.

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