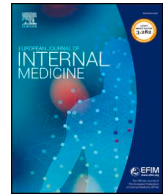




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Letter to the Editor

Is low sodium intake a risk factor for severe and fatal COVID-19 infection?



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As of March 16th, the number of deaths due to the coronavirus 2019 (COVID-19) outside of China has surpassed those in China. Intriguingly, recent calculations that adjust for an incubation period of up to 14 days estimate that the mortality rates from COVID-19 infection in China are nearly three times lower than outside of China [1]. It should be realized that, amongst others differences, China is known for having very high sodium intake compared to other countries in the world [2]. Human pathogenic coronaviruses bind to their target cells through angiotensin-converting enzyme 2 receptor (ACE2), which is expressed in epithelial cells of the lung, intestine, kidney, and blood vessels [3]. Importantly, animal-based studies have demonstrated that the tissue expression of the ACE2 receptor is down-regulated in response to a high dietary sodium intake [4,5].

Combined, these experimental and epidemiological data led us to hypothesize that low sodium balance may augment cellular damage at a certain virus load and increase the risk of developing severe and fatal COVID-19 infection. If this hypothesis were to be confirmed, it could lead to a conflict because, besides being a target for COVID-19, ACE2 also has anti-inflammatory properties and has been shown to protect against acid aspiration-induced acute respiratory distress syndrome [6].

Besides habitual dietary salt intake, more acute changes in sodium balance might also influence ACE2 receptor expression. Intermittent sodium loss, either due to diarrhea, vomiting or perspiration could put patients that acquire COVID-19 infection at higher risk for development of a more severe or fatal course of disease. It seems wise to monitor sodium intake and start with sodium and fluid resuscitation early in the

course of more severe COVID-19 infection, and perhaps also to refrain from robust sodium restriction during the current COVID-19 outbreak.

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