In animal studies, in the setting of endotoxin inhalation, loss of ACE2 function in lung led to activation of the des-Arg9 bradykinin /bradykinin receptor B1 axis and subsequent release of proinflammatory chemokines from airway epithelia, increased neutrophil infiltration, and exaggerated lung inflammation and injury. It seems that a reduction in pulmonary ACE2 activity contributes to the pathogenesis of lung inflammation, in part because of exaggerated bradykinin signaling [5]. Bradykinin is a potent pro-inflammatory and vasodilator peptide and a component of the contact system has important pathophysiological role in septic shock in general [6].

Of note, ibuprofen has been noted to augment some effects of bradykinin in vivo [7]. We suggest that this effect may be a further and additional mechanism of ibuprofen’s potentially adverse effect in COVID-19 infection and is to be investigated.
References
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