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Plasma ACE2 activity is persistently elevated following SARS-CoV-2 infection: implications for COVID-19 pathogenesis and consequences

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Plasma ACE2 activity is persistently elevated in patients after COVID-19 infection. Larger studies are needed to determine if this identifies people at risk of prolonged illness following COVID-19. https://bit.ly/2XQlrYF

Cite this article as: Patel SK, Juno JA, Lee WS, *et al*. Plasma ACE2 activity is persistently elevated following SARS-CoV-2 infection: implications for COVID-19 pathogenesis and consequences. *Eur Respir J* 2021; 57: 2003730 [https://doi.org/10.1183/13993003.03730-2020].

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To the Editor:

Coronavirus disease 2019 (COVID-19) causes persistent endothelial inflammation, lung, cardiovascular, kidney and neurological complications, and thromboembolic phenomena of unclear pathogenesis [1]. Severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) utilises the catalytic site of full-length membrane-bound angiotensin converting enzyme 2 (ACE2) for host cell entry [2], which is thought to downregulate membrane-bound ACE2, and thus contribute to ongoing inflammation due to loss of a degradative pathway for angiotensin II. In healthy individuals, ACE2 exists primarily in its membrane-bound form with very low levels of the catalytically active ectodomain of ACE2 present in the circulation [3]. However, in patients with cardiovascular disease, there is increased "shedding" of ACE2, and higher circulating levels are associated with downregulation of membrane-bound ACE2 [4].

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