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ACE-2 expression in the small airway epithelia of smokers and COPD patients: implications for COVID-19

Janice M. Leung[1,2,3], Chen X. Yang[1,2,3], Anthony Tam[1,2,3], Tawimas Shaipanich[2,3], Tillie-Louise Hackett[2,3,4], Gurpreet K. Singhera[1,2,3], Delbert R. Dorscheid[1,2,3] and Don D. Sin[1,2,3]

Affiliations: [1]University of British Columbia (UBC) Centre for Heart Lung Innovation, Vancouver, BC, Canada. [2]Dept of Medicine (Division of Respirology), University of British Columbia, Vancouver, BC, Canada. [3]St Paul's Hospital, Providence Health Care, Vancouver, BC, Canada. [4]Dept of Anesthesia, Pharmacology and Therapeutics, University of British Columbia, Vancouver, BC, Canada.

Correspondence: Don D. Sin, UBC Centre for Heart Lung Innovation, St Paul's Hospital, 1081 Burrard Street, Vancouver, BC V6Z1Y6, Canada. E-mail: don.sin@hli.ubc.ca

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Smokers and those with COPD have increased airway expression of ACE-2, which is the entry receptor for the COVID-19 virus. This may explain the increased risk of severe COVID-19 in these subpopulations and highlight the importance of smoking cessation. https://bit.ly/3bC29es

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To the Editor:

The World Health Organization (WHO) has declared coronavirus disease 2019 (COVID-19) a pandemic [1]. COVID-19 is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). COVID-19 displays symptoms ranging from mild to severe (pneumonia) that can lead to death in some individuals [2–4]. As of 18 April 2020, there have been 2 280 945 cases of COVID-19 worldwide and 156 354 deaths [5]. SARS-CoV-2 uses the angiotensin-converting enzyme II (ACE-2) as the cellular entry receptor [6]. While the virus can infect individuals of any age, to date, most of the severe cases have been described in those >55 years of age and with significant comorbidities, such as COPD [7]. Here, we determined whether patients with COPD have increased expression of ACE-2 in bronchial epithelial cells in the lower respiratory tract.