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## Letter to the Editor

## COVID-19 exposure and obstructive sleep apnea: a therapeutic approach



The rate of COVID-19 transmission has elevated the morbidity and mortality rate across the world. Respiratory failure is a predominant cause of COVID-19 related deaths. COVID-19 enters into the host cell through binding with angiotensin-converting enzyme-2 (ACE-2) receptor [1]. Extensive lung damage induces consequent cytopathic effect which triggers the inflammation. The induction of cytokines such as Interleukins, Tumor Necrosis Factor- $\alpha$ , C-Reactive Protein (CRP), leptin, ferritin etc are the main regulators of inflammatory response during COVID-19 infection. Incidentally similar inflammatory response have also been observed during obstructive sleep apnea, one of the most prevailing sleep-related respiratory disorder [2].

It can be hypothesized that COVID-19 exposure in the patients who already have OSA put them at heightened risk as they both involve and affect respiratory system. Thus special assistance and, the new therapeutic approach should be incorporated for the prevention and control of COVID-19 vulnerability in OSA patients [3]. A strong relationship exist between angiotensin-converting enzyme receptor II (ACE II) as well as hypertension in OSA [4]. In addition, the transmembrane protease serine 2 (TMPRSS2) mediated cleavage of S protein with ACE-2 receptor elucidates their entry and regulates hypertension in mild to moderate early diagnosed OSA patients [5].

The cited approach can be considered for OSA patients to attenuate the risk of COVID-19 infection. However, due to limited clinical data, it remains a challenge to identify the pathogenesis that may lead to improved outcomes of using ACE-2 inhibitors during COVID-19 infection in OSA patients. The new P4 medicine approach that involve personalization, prediction, prevention, and patient participation for OSA need to be considered during treatment of patients at high risk for COVID-19. OSA management requires a new molecular-based personal medicine approach, as they improve patient care and enhance our understanding of this complex disease. There is a need to enhance the awareness among the patients, health care professionals and organizations for effective therapeutic approach and control measures among these patients.

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None.

### Conflict of interest

None.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <https://doi.org/10.1016/j.sleep.2021.02.022>.

### References

- [1] South AM, Diz DI, Chappell MC. COVID-19, ACE2, and the cardiovascular consequences. *Am J Physiol Heart Circ Physiol* 2020;318(5):H1084–90.
- [2] Tay MZ, Poh CM, Renia L, et al. The trinity of COVID-19: immunity, inflammation and intervention. *Nat Rev Immunol* 2020;20(6):363–74.
- [3] Bonsignore MR, Baiamonte P, Mazza E, et al. Obstructive sleep apnea and comorbidities: a dangerous liaison. *Multidisciplinary respiratory medicine* 2019;14:8.
- [4] Koyama RG, Drager LF, Lorenzi-Filho G, et al. Reciprocal interactions of obstructive sleep apnea and hypertension associated with ACE I/D polymorphism in males. *Sleep Med* 2009;10(10):1107–11.
- [5] Heurich A, Hofmann-Winkler H, Gierer S, et al. TMPRSS2 and ADAM17 cleave ACE2 differentially and only proteolysis by TMPRSS2 augments entry driven by the severe acute respiratory syndrome coronavirus spike protein. *J Virol* 2014;88(2):1293–307.

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