Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Letter to Editors

Pulmonary lipid modulation: A possible therapeutic target for SARS-CoV-2 infection

Dear Editor,

We read with much interest the article by doctor Takano [1] who hypothesized a possible effect of the pulmonary surfactant treatment for COVID-19 and other viral pneumonia. The purpose of our Letter is to draw further attention upon the relevance of the lipid pattern regulation of the respiratory tract both in respiratory inflammation and in cellular viral entry.

In 2010, Ray et al. [2] described high levels of cardiolipin (CL), a potent phospholipidic surfactant inhibitor, in lung fluid of patients affected by severe pneumonia. Using experimental models, they demonstrated that CL transport was related to ATP8B1 flippase activity, a known crucial regulator of membrane fluidity involved also in type 1 progressive familial intrahepatic cholestasis, in which patients have also an increased risk of respiratory distress [3]. The administration of the recombinant CL binding domain peptide to ATP8B1 knock-down mutant mice lowered CL levels in lung fluid, reduced TNF-α/IL-β levels and normalized lung function. Because ATP8B1 is expressed in type II pneumocytes, it has been suggested that enhanced ATP8B1 flippase activity in the uninfected areas of the lung could reduce excess CL, and also the need of mechanical ventilation during acute respiratory distress syndrome and pneumonia. Others [4] demonstrated that CL maintains a condition of non-resolving lung inflammation by suppressing production of anti-inflammatory IL-10. In addition, mitochondrial CL is also known to induce NLRP3 inflammasome scaffold activation in both viral and bacterial pneumonia. Moreover, lipid flippases have been recently involved in the innate immunity which mitigates the inflammatory response, via Toll-like receptor 4 [5]. Said that, it is not surprising that the specific lower airway lipid composition might be associated with different intensities of host inflammatory responses, and that some respiratory viral infections cause disruption of wild type lipidomic patterns [6]. Lastly, some viruses such as SARS-CoV bind to the cytosolic membrane in a lipid-dependent manner, in the presence of the negatively charged lipid on membrane surface [7].

The above observations support doctor Takano hypothesis on the relevance of the lipid pattern regulation of the respiratory tract as a possible target of both treatment and prophylaxis against COVID-19 and other enveloped virus pneumonia [1].

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Claudia Mandato[a,*,1], Pietro Vajro[b,1]

[a] *Department of Pediatrics, AORN Santobono-Pausilipon, via Mario Fiore 6, 80129 Naples, Italy*

[b] *Department of Medicine, Surgery and Dentistry, "Scuola Medica Salernitana" University of Salerno, Baronissi, SA, Italy*

[*] Corresponding author at: Department of Pediatrics, Santobono-Pausilipon Children's Hospital, via Mario Fiore 6, 80129 Naples, Italy.
E-mail addresses: cla.mandato@gmail.com, c.mandato@santobonopausilipon.it (C. Mandato).

[1] Contributed equally.

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