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M. Soledad Celej, Irene Mangialavori

Diseño de Tapa y Logo

Comité organizador

Asistencia Técnica

Juan Pablo Acierno

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A region of the SARS-CoV-2 spike protein functionally interacts with the human $\alpha 7$ nicotinic receptor

Chrestia JF^a, Oliveira AS^b, Mulholland AJ^b, Gallagher T^b, Bermúdez I^c, Bouzat C^a

a - Instituto de Investigaciones Bioquímicas de Bahía Blanca (INIBIBB). Departamento de Biología, Bioquímica y Farmacia, Universidad Nacional del Sur (UNS- CONICET), Bahía Blanca, Argentina

b - School of Chemistry, University of Bristol, Bristol, United Kingdom

c - 4Department of Biological and Medical Sciences, Oxford Brookes University, Oxford OX3 0BP, United Kingdom.

Coronavirus disease 2019 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The binding of the viral spike protein (S) to angiotensin-converting enzyme 2 in host cells is crucial for infection. The S protein has been suggested to interact with nicotinic acetylcholine receptors (nAChRs), and their contribution to the COVID-19 inflammatory pathophysiology has been proposed. $\alpha 7$ is an interesting candidate target because it is present in neuronal and non-neuronal cells, and it has neuroprotective and anti-inflammatory actions. By whole-cell and single-channel recordings we revealed that the Y674-R685 region of the S protein shows a direct functional interaction with human $\alpha 7$ nAChR. The S fragment exerts a dual effect, acting as a low-efficacy agonist and a non-competitive antagonist. In agreement with molecular dynamics simulations showing stable binding of this region to the ACh binding pocket, the S fragment activates $\alpha 7$, but only in the presence of a potentiator, supporting its action as a very low-efficacy agonist. In addition, it allosterically inhibits $\alpha 7$ responses elicited by ACh, which may result in the predominant effect. This study provides unequivocal evidence supporting a functional $\alpha 7$ -S protein interaction, which may play a role in infectivity and/or disease progression and may be explored for new therapeutic opportunities.