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Structural proteins as the molecular basis of SARS-CoV-2 infection

Las proteínas estructurales como bases moleculares de la infección por SARS-CoV-2

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Mr. editor:

The new coronavirus named SARS-CoV-2, which causes zoonotic COVID-19 disease, emerged in the city of China, Wuhan and spread worldwide with a record of alarming numbers. The World Health Organization declared it an international health emergency in January 2020 and despite countless efforts to contain its rapid spread, it was classified as a pandemic[1,2].

SARS-CoV-2 belongs to a diverse family of single-stranded positive-sense RNA viruses. In humans, four endemic coronaviruses (HCoV-229E, HCoV-OC43, HCoV-NL63 and HCoV-HKU1) are known to cause mild respiratory symptoms in most cases; a picture that is in contrast to the manifestations caused by the new strain, which is characterized by a febrile state, general symptoms, intense cough and dyspnea. Twenty-five percent of cases develop an acute respiratory syndrome due to pneumonia, leading to multiorgan failure[1,3].

Viral genomic sequence analysis demonstrated a typical CoV structure (suffix used to categorize coronaviruses capable of infecting humans). It has a length of 30 kilobases with a single chain and positive polarity. From this study, the new coronavirus was positioned in the group of beta-coronaviruses, which includes its predecessors: SARS-CoV and MERS-CoV[4].

SARS CoV-2 has a total of 11 genes with 11 open reading frames (ORFs): ORF1ab, ORF2 (spike protein S), ORF3a, ORF4 (envelope protein E), ORF5 (membrane protein M), ORF6, ORF7a, ORF7b, ORF8, ORF9 (nucleocapsid protein N) and ORF10. These form the group of structural proteins; they are the basis of the viral infection mechanism and have well-defined functions for the development of this process[5].

The S protein homotrimers form the peaks on the viral surface and are responsible for binding to the host receptors, for which they require the participation of a special enzyme: protease. Each trimeric S protein monomer is approximately 180 kDa; it is divided into two functional units, S1 and S2; S1 facilitates virus infection by binding to host receptors and comprises two domains, the N-terminal domain and the C-terminal receptor binding domain that interacts directly with host receptors. There is only 76,74 % similarity of the S protein structure to predecessor coronaviruses[6,7].

A single region of the spike protein called the receptor-binding domain mediates the interaction with the host cell receptor. After binding the receptor, a nearby host protease cleaves the spike, which releases the fusion peptide facilitating virus entry. Known host receptors for beta-coronaviruses include angiotensin-converting enzyme 2 used by SARS-CoV-2[8,9].

Structural studies of coronaviruses have shown that the receptor-binding domain of the spike protein is capable of folding independently from the rest of the protein. All the structural information for binding to the host receptor is encoded in it[8].

It is the E protein that releases the viral genetic material and induces the development of the pathological traits of the virus; it stimulates NF-κB signaling, leading to pulmonary cytokine signaling and inflammatory cell recruitment. Although SARS-CoV-2 E protein is not required for viral replication, it is important for inhibition of the host cellular stress response, apoptosis and the unfolded protein response[10].

The M protein is responsible for binding to structures called nucleocaspids. This function is provided by its form of 3 transmembrane domains with virons. On the other hand, the N protein determines the binding to the host genome. It is reported that it can bind to the NSP3 protein to help bind the genome and package it in an encapsulated manner in virons and in turn it is an interferon antagonist[4].

Since the report of the first

case of the new coronavirus, the number of infected has been on the rise, however, the number of deaths is not far behind. This has drawn the attention of the world population, especially of the scientific community in search for a cure. In this case, the focus is on the study of viral RNA. Although the S protein has totally different segments, it is clear that the RNA of the new virus has segments that have not yet been deciphered. This emerges as a new beacon for world science, which tirelessly proposes to decipher each segment of the viral genome, taking as a premise the study of the synthesis of protein S, in order to determine the origin, evolution and treatment of the disease caused by SARS-CoV-2.

AUTHORSHIP

Luis Enrique Jiménez-Franco: conceptualization, formal analysis, investigation, methodology, supervision, writing-revision and editing.

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BIBLIOGRAPHIC REFERENCES

1. Stadlbauer D, Amanat F, Chromikova V, Jiang K, Strohmeier S, Asthagiri Arunkumar G, et al. SARS-CoV-2 Seroconversion in Humans: A Detailed Protocol for a Serological Assay, Antigen Production, and Test Setup. CPMicrobiol [Internet]. 2020 [cited 02/28/2021]; 57(1):1-15. Available from: https://doi.org/10.1002/cpmc.100
2. Belasco Silvia AG, Cassiane Dezoti da F. Coronavirus 2020. Rev Bras Enferm [Internet]. 2020 [cited 02/28/2021]; 73(2):1-2. Available from: https://doi.org/10.1590/0034-7167-2020730201
3. Serra Valdés MA. Infección respiratoria aguda por COVID-19: una amenaza evidente. Rev haban cienc méd [Internet]. 2020 [cited 02/28/2021]; 19(1):1-5. Available from: http://www.revhabanera.sld.cu/index.php/rhab/article/view/3171
4. Chen Y, Liu Q, Guo D. Emerging coronaviruses: Genome structure, replication, and pathogenesis. J Med Virol [Internet]. 2020 [cited 02/28/2021]; 20(92): 418-423. Available from: https://doi.org/10.1002/jmv.25681
5. Yoshimoto F. The Proteins of Severe Acute Respiratory Syndrome Coronavirus-2 (SARS CoV-2 or n-COV19), the Cause of COVID-19. The Prot Jurn [Internet]. 2020 [cited 02/29/2021]; 10(4):1-19. Available from: https://doi.org/10.1007/s10930-020-09901-4
6. Xiuyuan O, Yan L, Xiaobo L, Dan M, Ruixuan G, Zhixia M. Characterization of spike glycoprotein of SARS-CoV-2 on virus entry and its immune cross-reactivity with SARS-CoV. Nat Commun [Internet]. 2020 [cited 02/28/2021]; 11(2):1-12. Available from: https://doi.org/10.1038/s41467-020-15562-9
7. Xintian X, Ping C, Jingfang W, Jiannan F, Hui Z, Xuan L. Evolution of the novel coronavirus from the ongoing Wuhan outbreak and modeling of its spike protein for risk of human transmissio. Sci China Life Sci [Internet]. 2020 [cited 02/28/2021]; 63(3):457-460. Available from: https://doi.org/10.1007/s11427-020-1637-5
8. Letko M, Marzi A, Munster V. Functional assessment of cell entry and receptor usage for SARS-CoV-2 and other lineage B betacoronaviruses. Nat Microbiol [Internet]. 2020 [cited 02/28/2021]; 14(2):1-8. Available from: https://doi.org/10.1038/s41564-020-0688-y
9. Andersen K, Rambaut A, Lipkin I, Holmes E, Garry R. The proximal origin of SARS-CoV-2. Nat Med [Internet]. 2020 [cited 02/28/2021]; 15(3):1-3. Available from: https://doi.org/10.1038/s41591-020-0820-9
10. Lisa Gralinski E, Ralph Baric S. Molecular pathology of emerging coronavirus infections. J Pathol. [Internet]. 2015 [cited 02/28/2021]; 235(2):185-195. Available from: https://pubmed.ncbi.mln.nih.gov/25270030/