Perspective
Volume 7:8, 2022

Journal of Infectious Diseases and Medicine

ISSN: 2576-1420 Open Access

The Role of Human Cell Organelles in SARS-CoV-2 Infection: A Current Review

Bartosz Ustianowska*

Department of Clinical and Molecular Biochemistry, Pomeranian Medical University, 70-204 Szczecin, Poland

Introduction

In the final weeks of 2019, Wuhan, in the Chinese province of Hubei, had a series of severe atypical viral respiratory infections that spread quickly by droplets and direct contact. A novel enclosed spherical virus has now caused havoc over the entire world. SARS-CoV-2 was discovered to be related to the coronavirus that caused the SARS outbreak. In terms of RNA sequence, these viruses are 80% identical. The World Health Organization (WHO) named the novel infection Coronavirus Disease 2019 (COVID-19) a worldwide pandemic in March 2020, making it a threat to global health. More than 500 million confirmed COVID-19 cases and more than 6 million fatalities have been documented by the WHO to date (as of 1 May 2022) Due to day and night study undertaken in laboratories, the first half of 2019 and the first half of 2020 offered us fresh information about this dangerous virus.

The Coronaviridae family of positive-sense, single-strained RNA coronaviruses, which mostly infect birds and mammals as pathogens, includes SARS-CoV-2. This new coronavirus resembles another virus that has been identified recently and causes MERS, the Middle East Respiratory Syndrome Coronavirus (MERS-CoV). Furthermore, Angiotensin II (AngII) receptors are used by SARS-CoV, SARS-CoV-2, and MERS-CoV to enter cells. Random mutations are prone to occur in its genome. 20 proteins are present in SARS-CoV-2, of which 4 are structural and the remaining 13 are crucial for replication and transcription. The genetic diversity of SARS-CoV-2 influences its virulence, transmissibility, and severity of clinical presentation, resulting in breakouts of many varieties of concern (VOC) have increased worldwide hospitalisation rates, recorded cases, and fatalities. The mechanism of viral entrance and the participation of host cell organelles alter with the generation of fresh variations [1-5].

Description

The viral Spike (S) glycoprotein is necessary for SARS-CoV-2 entrance into the host cells. The S protein is a homotrimer composed of the S1 and S2 subunits. While the second links the S protein to the membrane surface and is essential for the fusion of membranes, the first is in charge of attaching to a receptor on the surface of the host cells. In electron micrographs, the protein is evenly distributed across the virion's surface, giving the impression of a crown. The N-terminal (NTD), receptor-binding (RBD), and two C-terminal domains (CTDs) make up the S1 subunit's structure. It is still unknown how NTD affects cell entrance. NTDs in animal coronaviruses are understood to increase the either bind to a receptor or serve as stabilising agents. A receptor-binding motif found in the RBD domain is in charge of the direct contact with the receptor. The link depends on RBD having the correct conformation, which is made feasible by conformation changes in the other domains. The S protein structural modifications required for proper interaction with the receptor and membrane fusion are stabilised by the CTDs, which are important domains. The novel CoV invades host cells using the angiotensin converting enzyme 2(ACE2) receptor, just as the SARS-CoV.

The RAAS's (renin-angiotensin-aldosterone) ACE2 is a physiological component that plays a role in controlling electrolyte levels and blood pressure. Angiotensin II is converted into angiotensin (1-7) by ACE2 to exert its vasodilatory and anti-inflammatory properties. Comparable to target cells for SARS-CoV-2, human tissues express ACE2 in similar ways. A high amount of ACE2 expression is present in the upper respiratory tract, which is often where viruses replicate in the early stages of infection. However, the intestinal, colon, kidney, or heart muscle exhibited the highest levels of ACE2 expression, which explains the organ problems of COVID-19. Despite COVID-19 The expression of ACE2 in the lung is restricted to type II alveolar cells, highlighting the additional parameters influencing viral invasion, which might result in respiratory failure. After interacting with ACE2 on the host cell membrane, two distinct paths of host cell invasion may take place. SARS-CoV-2 prefers to enter cells through the cell surface, but it needs the transmembrane serine protease 2 (TMPRSS2) enzyme to do so The enzyme's physiological function is still a mystery. The invasion process of other viruses, such as influenza viruses, MERS-CoV, or SARS-CoV, depends on the changes of viral proteins by TMPRSS2. The mucosal surface of the gastrointestinal, urogenital, and respiratory tracts are where the TMPRSS2 is primarily found.

The bronchial epithelium and colon were reported to co-express TMPRSS2 and ACE2, emphasising the target cells of SARS-CoV-2. Endosome development is connected to the second pathway of cell entrance. This method might operate when there is not enough TMPRSS2 expression on the membrane surface. In this situation, clathrin-mediated endocytosis takes place, which results in the internalisation of the virus associated with ACE2. The second cleavage of the S protein may occur in the endosome, which is mediated by cathepsins. The breakdown of proteins in lysosomes and endosomes depends heavily on this class of non-specific proteases. Cathepsins are frequently used by viruses as they enter the host cells. The roles of cathepsin B in Ebola virus entry and cathepsin L in SARS-CoV and SARS-CoV-2 entry are the best recognised and SARS-CoV-2 was thought to have a low dependence on the endosomal entry pathway before the introduction of the now-dominant Omicron VOC, indicating the restricted effectiveness of endosomal enzyme inhibitors in the treatment of COVID-19. Recent research on Omicron VOC, however, revealed that the virus has a greater capacity to penetrate the host cell without the assistance of TMPRSS2. Additionally, the Omicron VOC can target more respiratory tract cells thanks to the endosomal entry pathway, which raises transmissibility. The fusing of the membranes is sparked by the alteration of the S2 subunit of the S protein, which is catalysed by both TMPRSS2 and cathepsins. Following the second cleavage, the S2 subunit's shape undergoes a considerable shift, and the S1 subunit separates. These processes cause the membranes to come together and produce a fusion pore, which allows viruses to enter the body [5-10].

Conclusion

Worldwide researchers have been studying coronaviral infections since

***Address for Correspondence:** Bartosz Ustianowska, Department of Clinical and Molecular Biochemistry, Pomeranian Medical University, 70-204 Szczecin, Poland; E-mail: Bartoszustianowska24@gmail.com*

Date of submission: 01 August, 2022, Manuscript No. jidm-22-77639; **Editor Assigned:** 03 August, 2022, PreQC No. P-77639; **Reviewed:** 17 August, 2022, QC No. Q-77639; **Revised:** 23 August, 2022, Manuscript No. R-77639; **Published:** 29 August, 2022, DOI: 10.37421/2576-1420.2022.7.249.

the turn of the century in 2019 and 2020. Multiple cellular mechanisms that SARS-CoV-2 altered during the course of the pandemic's more than two years have been found. We summarised research publications on the function of cellular organelles and discussed potential therapeutic targets in this review. In order to slow the progression of infection, various clinical trials identified the mechanisms of cell invasion and subsequently targeted those mechanisms. Furthermore, SARS-CoV-2 replication may be significantly influenced by mitochondria. During COVID-19, mitophagy and autophagy are also disturbed, which causes a buildup of dysfunctional organelles. Our examination of cellular dysfunction and host cell organelles during COVID-19 has come to a close, but there are still many areas that need additional study to fully comprehend this intricate process. It is still too early to say whether future months will see the emergence of affordable agents with a good safety profile.

References

1. Xiong, Jiaqi, Orly Lipsitz, Flora Nasri and Leanna MW Lui, et al. "Impact of COVID-19 pandemic on mental health in the general population: A systematic review." *J Affect Disord* 277 (2020): 55-64.
2. Yu, Ping, Jiang Zhu, Zhengdong Zhang and Yingjun Han. "A familial cluster of infection associated with the 2019 novel coronavirus indicating possible person-to-person transmission during the incubation period." *J Infect Dis* 221 (2020): 1757-1761.
3. Sheam, Md Moinuddin, S. Syed, S. Barman and Md Rockybul Hasan, et al. "COVID-19: the catastrophe of our time." *J Adv Biotechnol Exp Ther* 3 (2020): 1-13.
4. Nitin, P., R. Nandhakumar, B. Vidhya and S. Rajesh, et al. "COVID-19: Invasion, pathogenesis and possible cure–A review." *J Virol Methods* (2021): 114434.
5. Ksiazek, Thomas G., Dean Erdman, Cynthia S. Goldsmith and Sherif R. Zaki, et al. "A novel coronavirus associated with severe acute respiratory syndrome." *N Engl J Med* 348 (2003): 1953-1966.
6. Drożdżal, Sylwester, Jakub Rosik, Kacper Lechowicz and Filip Machaj, et al. "COVID-19: Pain management in patients with SARS-CoV-2 infection—molecular mechanisms, challenges, and perspectives." *Brain Sci* 10 (2020): 465.
7. Giovanetti, Marta, Francesca Benedetti, Giovanni Campisi and Alessandra Ciccozzi, et al. "Evolution patterns of SARS-CoV-2: Snapshot on its genome variants." *Biochem Biophys Res Commun* 538 (2021): 88-91.
8. Angeletti, Silvia, Domenico Benvenuto, Martina Bianchi and Marta Giovanetti, et al. "COVID-2019: the role of the nsp2 and nsp3 in its pathogenesis." *J Med Virol* 92 (2020): 584-588.
9. Kotfis, Katarzyna and Karolina Skonieczna-Żydecka. "COVID-19: gastrointestinal symptoms and potential sources of SARS-CoV-2 transmission." *Anaesthesiol. Intensive Ther* 52 (2020): 171-172.
10. Lechowicz, Kacper, Sylwester Drożdżal, Filip Machaj and Jakub Rosik, et al. "COVID-19: the potential treatment of pulmonary fibrosis associated with SARS-CoV-2 infection." *J Clin Med* 9 (2020): 1917.

How to cite this article: Ustianowska, Bartosz. "The Role of Human Cell Organelles in SARS-CoV-2 Infection: A Current Review." J Infect Dis Med 7 (2022): 249.