

## Lipid Metabolism Modifications in SARS-CoV-2

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### ABSTRACT

SARS-CoV-2 is the reason of the COVID-19 pandemic that has infected over a hundred million people globally. There have been more than two million deaths recorded worldwide, with no end in sight until a widespread vaccination will be attained. Current research has centred on different aspects of the virus interaction with cell surface receptors, but more needs to be done to further comprehend its mechanism of action in order to develop a targeted therapy and a method to control the spread of the virus. Lipids production a crucial role throughout the viral life cycle, and viruses are known to exploit lipid signalling and synthesis to move host cell lipids. Emerging studies using untargeted metabolomic and lipidomic approaches are providing new insight into the host response to COVID-19 infection. Indeed, metabolomic and lipidomic methods have identified numerous circulating lipids that directly correlate to the severity of the disease, making lipid metabolism a potential therapeutic target. Circulating lipids play a key function in the pathogenesis of the virus and exert an inflammatory response. A better knowledge of lipid metabolism in the host-pathogen interaction will provide valuable insights into viral pathogenesis and to the development of novel therapeutic targets.

**Keywords:** SARS-CoV-2; Lipids; Fatty acids

### INTRODUCTION

The year 2020 will be certainly remembered as the year of the coronavirus disease 19 (COVID-19) pandemic. Towards the end of 2019, a novel coronavirus communicable disease generated by the plain acute syndrome coronavirus 2 (SARS-CoV-2) made its appearance in Wuhan, China [1]. As it was the case with the other two recent coronavirus spreads, 2003 Severe Acute Respiratory Syndrome (SARS-CoV) and 2012 Middle East respiratory syndrome (MERS-CoV), and many other so-called “bird flu” worldwide, it is believed to be a zoonotic virus, originated in a live market from bats and then transferred to human hosts [2]. Therefore, directing membrane sphingolipids and interfering with the virus lipid metabolism could represent a path to follow towards the development of COVID-19 treatments. Lipids play a key role in the initial stages of the interaction of viruses with host membranes. The first step of this complex process is the virus adhesion to the cell membrane this involves unspecific binding with negatively charged sugars of glycoproteins and glycolipids that represents a first dock before the engagement with a specific receptor. Heparan sulphate proteoglycans,

expressed by the majority of eukaryotic cells, have been shown to play a role in the adhesion of many viruses to the cell external membrane, including SARS-CoV-2 [3]. Similarly, the discovery of LA-binding pockets could be exploited for scheming specific inhibitors able to obstruct virus-host interactions by blocking the spike protein in the closed conformation. The subsequent step involves the virus entry into cell cytosol using either a direct fusion mechanism or the misuse of the endocytic pathways of the host cells. In both cases, this is a critical energy-consuming stage connecting viral membrane fusion [4]. However, strategies pointing at the inhibition of PI-kinases should be taken with caution considering the potential side-effect that can be caused. In particular, PIKfyve inhibition has been shown to generate large vacuoles in cells and lead to apoptosis *in vitro* and *in vivo*.

### CONCLUSION

Viruses are known to feat the host resource and machinery, particularly lipid metabolism pathways, to invade cells and replicate. Therefore, therapeutic approaches targeting conserved cellular mechanism that virus manipulates to enter host cells and

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replicate are particularly attractive. Strategies to specifically target lipid metabolism have been strained, such as the use of cholesterol depleting agents or autophagy inducers. One way of responding in a short time frame and avoiding severe adverse effects is to look, to drugs that have received endorsement to treat other diseases. Unfortunately, given the frequency of viral epidemics that we have witnessed in the last decade, it is likely that we will see the appearance of a new pandemic in the near future. Consequently, a therapeutically method to COVID-19 that can be adapted and exploited in the event of future outbreaks is urgently needed.

## REFERENCES

1. Morens DM, Daszak P, Taubenberger JK. Escaping Pandora's box another novel coronavirus. *New England J Med.* 2020;382(14): 1293-1295.
2. Weiss P, Murdoch DR. Clinical course and mortality risk of severe COVID-19. *The Lancet.* 2020;395 (1001): 1014-1015.
3. Liu J, Zhang S, Dong X, Li Z, Xu Q, Feng H et al. Corticosteroid treatment in severe COVID-19 patients with acute respiratory distress syndrome. *J Clin Inv.* 2020;130 (1545): 6417-6428.
4. Keller MJ, Kitsis EA, Arora S, Chen JT, Agarwal S, Ross MJ et al. Effect of Systemic Glucocorticoids on Mortality or Mechanical Ventilation in Patients With COVID-19. *J Hosp Med.* 2020;15(8): 489-493.