Introduction

Coronavirus disease 19 (COVID-19) first emerged on 31 December 2019 in Wuhan city, China.COVID-19 is classified as the seventh member of the subfamily Orthocoronavirinae under the family Coronaviridae ,Most members of this family are zoonotic viruses transmitted to humans through contact with infected animals.there is no evidence so far that COVID-19 originated in or was transmitted from a seafood market [1] Comparison of the lipid rafts of coronaviruses has indicated that the new strain COVID-19 has 80% identity with severe acute respiratory syndrome coronavirus (SARS-CoV) These molecules are involved in the entry of viruses into host cells and targeting host lipids is being studied as an antiviral strategy and could have various applications [2],COVID-19 seems to need to bind to the angiotensin-converting enzyme-2 receptor on the membrane host cell to enable it to infect the host cell upon coupled with a reliance of serine protease TMPRSS2. This intracellular protein seems to be a determinant of the virus ability to infect the cell [3].

References

[1]

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[2]

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[3]

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[1]

- In recent years, the emergence and reemergence of infectious diseases like bird flu (influenza A H5N1) in 2003, SARS in 2002/2003, influenza A (H1N1) in 2009, and Zika in 2015 raised numerous questions on the role of epidemiological surveillance. Pandemics have occurred more frequently, and since 2018, the World Health Organization (WHO) has acknowledged the need for preparation in anticipation of the emergence of novel pathogens, including (under the name “disease X”) unknown diseases with potential for international emergence on the priority list for research and development in the emergency setting . The emergence of novel diseases causes impacts far beyond the cases and deaths they generate. It also creates an ideal context that requires national public health systems to validate their surveillance and healthcare systems for timely detection and response in cascade.

[2]

- Background: Viral infectivity depends on interactions between components of the host cell plasma membrane and the virus envelope. Here we review strategies that could help stem the advance of the SARS-COV-2 epidemic. Methods and Results: We focus on the role of lipid structures, such as lipid rafts and cholesterol, involved in the process, mediated by endocytosis, by which viruses attach to and infect cells. Previous studies have shown that many naturally derived substances, such as cyclodextrin and sterols, could reduce the infectivity of many types of viruses, including the coronavirus family, through interference with lipid-dependent attachment to human host cells. Conclusions: Certain molecules prove able to reduce the infectivity of

some coronaviruses, possibly by inhibiting viral lipid-dependent attachment to host cells. More research into these molecules and methods would be worthwhile as it could provide insights the mechanism of transmission of SARS-COV-2 and, into how they could become a basis for new antiviral strategies.

[3]

- SARS-Cov-2 appears to need to bind to the ACE 2 receptor to enable it to infect host cells, coupled with a reliance on the cellular serine protease TMPRSS2 which also seems to be a determinant of the viruses ability to infect cells.3 These authors seem to point to the possibility that the clinically proven serine protease inhibitor camostat mesylate, which is active against TMPRSS2 partially blocked SARS-Cov-2 entry into cells and is thus a potential target as an agent to mitigate the impact of SARS-Cov-2 in individuals affected by COVID-19. They note that at present this agent is licenced for human use in Japan to treat an unrelated condition.