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].

Abstraction

- Coronavirus disease 2019 SARS-CoV-2 (COVID-19) is a zoonotic virus causing a variety of severe respiratory diseases,. SARS-CoV-2 is closest to SARS-CoV and MERS-CoV in structure ,by evaluating 11 complete genome sequences of different coronaviruses using BAST and MAFFT software, they conclude that COVID-19 might produce new mutations, specifically in glycoproteins, so caution and complete preparation by health authorities is required.

References

[1]

R. Lana, F.C. Coellaho, M.F. Gomes, O.G. Cruz, L.S. Bastos, D.A. Villela, *et al.***The novel coronavirus (SARS-CoV-2) emergency and the role of timely and effective national health surveillance**Rep Public Health (2020)Epub ahead of print

http://doi:10.1590/0102-311X00019620

Google Scholar

[2]

M. Baglivo, M. Baronio, G. Natalini, T. Beccari, P.C. Fuulcheri, P. Petralia, *et al.***Natural small molecules as inhibitors of coronavirus lipid-dependent attachment to host cells: a possible strategy for reducing SARS-COV-2 infectivity?**Acta Biomed, 91 (2020), pp. 161-164

https://www.scopus.com/record/display.uri?eid=2-s2.0-85082038349&origin=inward&txGid=ae34ba2897cc992e3b383181ec39e549

[3]

G. Thomso**COVID-19: social distancing, ACE 2 receptors, protease inhibitors and beyond?**Int J Clin Prac (2020), 10.1111/ijcp.13503Epub ahead of print

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7228364/

[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.

[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.

proposed methodology

- COVID-19 is related to the beta-coronavirus that infects humans and probably developed from bat coronaviruses. Structural analysis shows that COVID-19 probably derives from a bat SARS-like coronavirus, which has mutated in the spike glycoprotein (protein S) and nucleocapsid N protein The positive-sense RNA genomes of COVID-19 differ from SARS-CoV and MERS-CoV, In addition, the spherical external spike protein displays a characteristic crown shape with electron microscopy [9]. In the current study, we have compared the novel COVID-19 complete genome with other related coronaviruses to identify mutations and gaps. We selected data from NCBI and we performed the FASTA and BLAST. The comparison between genomes with alignment used MAAFT-7 software. COVID-19 (GenBank MT188341.1) and COVID-19 (MT066175.1), bat-SL-CoVZC45 (MG772933.1) and SARS-CoV BJ182b (EU371561.1) showed alignment identities of 99%, 89% and 82%, respectively. Fig. 1 shows the differences between the four complete genomes.

```
GGTATGAGCTATTATTGTAAATCACATAAACCGCCCATTAGTTTTCCATTGTGTGCTAAT  16440
................................A.............................  16494
GGACTACCAACTCAAACTGTTGATTCATCACAGGGCTCAGAATGTGACTATGTCATATTC  17820
.............................................A................  17874
GGACTTTTTAAAGATTGTAGTAAGGTAATCACTGGGTTACATCCTACACAGGCACCTACA  18060
.....C.....................................................  18114
```

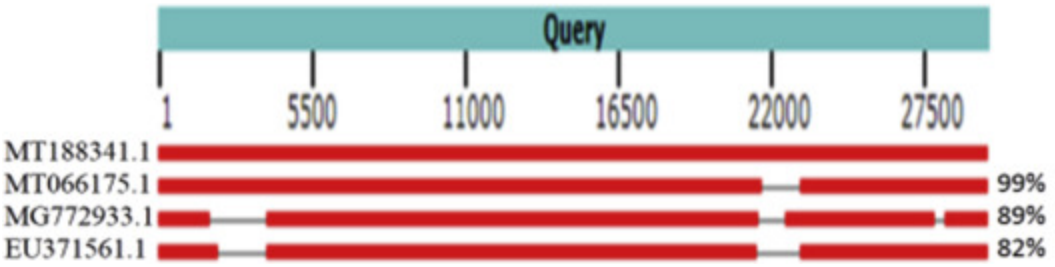
MT188341.1 + MT066175.1 similarity 99% partial seq.

```
TCATCAAACGTTCGGATGCTCGAACTGCACCTCATGGTCATGTTATGGTTGAGCTGGTAG  479
............T.....C.............C..C..........C..AT.A....  532
CAGAACTCGAAGGCATTCAGTACGGTCGTAGTGGTGAGACACTTGGTGTCCTTGTCCCTC  539
..........T..........T........................T..........  592
ATGTGGGCGAAATACCAGTGGCTTACCGCAAGGTTCTTCTTCGTAAGAACGGTAATAAAG
....A..A..GG.......T........T..A.............................  652
```

MT188341.1 & MG772933.1 similarity 89% partial seq.

```
GAAAACTTGTTACT-TTATAT--TGACATTAATGGCAATCTTCATCCA-GATTCTGCCAC  4017
A.T..---......C..G.T.GC...T..C.....T..G...T.-...T......-.AGA  3997
TCTTG-TT--A-GTGACATTGACATCACTTTCTTAAAGAAAGATGCTCCATATATAGTGG  4073
A.A..C..AG.G....-.--..T..GT.....C.TG....G.....A..T..C..G..A.  4054
GTGATGTTGTTCAAGAG-GGTGTTTTAACTGCTGTGGTTATACCTACTAAAAAGGCTGGT  4132
.........-A...CT..T....A.A.C...TG...T..A.....CT.C............  4113
```

MT188341.1 & EU371561.1 similarity 82% partial seq.



Conclusion & future look

- COVID-19 is a great biological hazard and is a worldwide threat. COVID-19 is highly contagious during the latency period. It is may be necessary to adopt and invest in more modern technologies both to facilitate notification, and to allow speedier data dissemination and analysis
- We suggest that close contact with an infected person is the major factor in disease transmission. Health-care workers must also follow CDC guidelines and should not attempt to perform any virus isolation or characterization. The effect of mass gathering cancellations on reducing the spread of COVID-19 needs to be determined. Any mutation occurring will be especially important. There is no evidence that part of COVID-19 is synthetic.

References

[1]

R. Lana, F.C. Coellaho, M.F. Gomes, O.G. Cruz, L.S. Bastos, D.A. Villela, *et al*.**The novel coronavirus (SARS-CoV-2) emergency and the role of timely and effective national health surveillance**Rep Public Health (2020)Epub ahead of print

http://doi:10.1590/0102-311X00019620

Google Scholar

[2]

M. Baglivo, M. Baronio, G. Natalini, T. Beccari, P.C. Fuulcheri, P. Petralia, *et al*.**Natural small molecules as inhibitors of coronavirus lipid-dependent attachment to host cells: a possible strategy for reducing SARS-COV-2 infectivity?**Acta Biomed, 91 (2020), pp. 161-164

https://www.scopus.com/record/display.uri?eid=2-s2.0-85082038349&origin=inward&txGid=ae34ba2897cc992e3b383181ec39e549

[3]

G. Thomso**COVID-19: social distancing, ACE 2 receptors, protease inhibitors and beyond?**Int J Clin Prac (2020), 10.1111/ijcp.13503Epub ahead of print

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7228364/

[6]

A. Wu, Y. Peng, B. Huang, X. Ding, X. Wang, P. Niu, *et al.***Genome composition and divergence of the novel coronavirus (2019-nCoV) originating in China**Cell Host Microbe (2020), 10.1016/j.chom.2020.02.001Epub ahead of printGoogle Scholar

[7]

A. Cheepsattayakorn, R. Cheepsattayakorn**Proximal origin and phylogenetic analysis of COVID-19 (2019-nCoV or SARS-CoV-2)**EC Microbiol (2020)Epub ahead of printGoogle Scholar

[8]

Y.-R. Guo, Q.-D. Cao, Z.-S. Hong, Y.-Y. Tan, S.-D. Chen, H.-J. Jin, *et al.***The origin, transmission and clinical therapies on coronavirus disease 2019 (COVID-19) outbreak—an update on the status**Milit Med Res, 7 (2020), p. 11CrossRefView Record in ScopusGoogle Scholar

[9]

J.-M. Kim, Y.-S. Chung, H.J. Jo, N.-J. Lee, M.S. Kim, S.H. Woo, *et al.***Identification of coronavirus isolated from a patient in Korea with COVID-19**Osped Pub Health Res Per, 11 (2020), pp. 3-7CrossRefView Record in ScopusGoogle Scholar