Twitter Thread by Rossana Segreto

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I must say it, @K_G_Andersen never ends to surprise me with his compelling arguments... If you want to figure out why Wade is wrong you can Google it ■■■

Here he downplays the FCS of SARS2. These fragments do not really come and go all the time. I am not aware of experiments on CoV where the FCS arose by cell or animal passage. More on the FCS here:
https://t.co/oyPKBVVG4f

> The FCS is unique in the whole Sarbecovirus group to which SARS2 belongs. Here other reasons why it should be taken seriously. The codon used for arginine is very rare in CoVs but it translates very well in humans.https://t.co/s7q6Ynh7NYhttps://t.co/lHcshQp2Uq pic.twitter.com/kXKKb8FIk7
>
> — Rossana Segreto (@Rossana38510044) May 9, 2021

Here he refers to RmYN02, which has no insertion at the S1/S2 cleavage site
https://t.co/0Goapx6cXj
https://t.co/zEALrUUj8F
And to Gallaher's paper, with the fanciful theory of recombination on a train to Wuhan
Recombination is rare outside its group +
https://t.co/DBs7RMeq4X

As also my co-author @ydeigin says
https://t.co/qrOG0nlIzR
the FCS could be not optimal because part of a vaccine attenuation strategy
https://t.co/tHWG1Igf7Q
Andersen admitted to be wrong with his prediction of the O-linked glycans

> Could the uncharacteristic furin cleavage site in SARS2 have been a part of a vaccine attenuation strategy? You know, the pan-coronavirus vaccine that Fauci, Daszak and other have dreamed about? https://t.co/EpSXwzfBD6
>
> — Yuri Deigin (@ydeigin) May 9, 2021

The FCS might be similar at the aa level to some FCoV sequences, but less at the nt level.
https://t.co/Hvi3azygu6

Andersen does not mention that the FCS binds extremely well to heparan sulphate as possible result of cell passage https://t.co/s7q6Ynh7NY

> indeed. at the nucleotide level the FCS differ in the wobble bases. I initially thought of cold-adapted live-attenuated feline vaccines, which can be administered intranasally (e.g. Felocell FIP IN). pic.twitter.com/bcwdAEphlA
>
> — Chris de Z (@CZilcho) April 30, 2021

That the "P" is mutating towards residues creating more optimal furin sites could be reversion, which is not rare in live attenuated viruses, mostly if not completely attenuated because accidentally leaked.

"the exact (P)RRAR can be found in other coronaviruses." is true only at the amino acid level, not as nucleotides.

Andersen admits that the codon CGG is rare in CoV because it stimulates an immune reaction. It is only found in 3 % of arginine in SARS2. But he forgets to mention that CGG is double in the FCS, making it very special.
https://t.co/dgrP8nwPRz
Nothing unusual here.

> 22/ The FCS in SARS2 has highly CpG-rich insertion (CGG-CGG) which is extremely rare as double instance in CoVs and deoptimizes the codon for replication https://t.co/D4Z2AdS1AX pic.twitter.com/E0Ffr4vKXk
>
> — Rossana Segreto (@Rossana38510044) October 3, 2020

He forgets also to mention that by chance CGG is the best codon for arginine in humans, and CGA, used as second codon in FCoV is not that good (0.11 CGA vs 0.21 CGG)
https://t.co/KQpNwt6QLq

And the fact that the FCS is remarkably stable and necessary for human-to-human transmission in a virus which had only few months to adapt to a new host might hint to previous cell passage in human airways cells, where it is stable or humanized mice.

So all the points from Andersen to disprove Baltimore's observation on the FCS are false/misleading.

https://t.co/otPnkrF7KS
Actually our @Daoyu15 commented on this, but his replies have been hidden by @Merz. This is the way virologists welcome dissenting opinions.
https://t.co/X8DgaKljk0
https://t.co/THbWA1HhUz

> Re. the furin site present in SARS-CoV-2. A stem loop immediately follows it, immediately suggesting a mechanism for replicase stalling followed by strand slippage or template switching.
>
> I've not seen anyone else comment on this. https://t.co/27vhhvbrAQ
>
> — Alex Merz (@Merz) May 9, 2021

Andersen does not take into account also the other special feature of SARS2, which is its special RBD adapted from the first isolate for very efficient human infection. He tried to justify it with its presence in the pangolin CoV MP789, which we can't trust.

https://t.co/U2qvG3tXBG

And beside the special FCS and RBD we have a mountain of circumstantial evidence for a lab leak, well explained by Wade, which comprises deception of data and blocking a fair investigation.