Twitter Thread by Ersa Flavinkins



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This is why they kept these datasets hidden for so long. (notice how there were no original format nor there were any date of original data deposition from the SRP or the Run Selector.) They altered the ACE2 gene on these datasets. it took them a month, and no dataset deposited

before this date have evidence of "RaACE2" 1-85.
this is found in the Jul.01 dataset (altered).

While none of the datasets published before 01/07 (this is published at 01/06 have this "ACE2 1-85". nothce that this study itself does not contain any ACE2 fragments in all it's probe capture studies published before 01/07/2020. the most logical explanation is that they simply

took the fake Shi "clones" and spiked them into the 01/07 datasets. unfortunately, the methodology and the early R.Affinis sneak peak (XGD01) lacked the ACE2 mRNA fragment, which tells us that these datasets have been fraudulently altered ("salted"). that is why it took them

over two months to "publish" the data, and why they have withheld the ability for BLAST analysis on these datasets for over 6 months. They must have worked overtime to alter the ACE2 gene on these datasets to fit Shi's narrative.

This is what that have always been warned about--every single dataset that were published after that Shi paper of "evolutionary arms race" (14/May/2020) should not be trusted. they deliberately withheld the "R.Affinis sequence capture" datasets for this long, because the original

(one R.Affinis used for reference in XGD01, plus the R.Sinicus and R.Pearsonii cluster) datasets and their methodologies does not generate a fragment of ACE2 1-85. so they waited for Shi to Yeast Display up a viable ACE2 (which is still <1/1000 on RaTG13 than CoV2/hACE2) before

incorporating the sequences into a R.Ferrumurquinum Refseq and added these fake "reads" into the datasets. As it's only ~20 reads, the tampering process takes extremely little time to do. (alter a few base calls from R.Sinicus ACE2, keep the quality score.)

Tampering of these datasets were additive--there were traces of ACE2 reads that were identical to R.Ferrumurquinum. likely left in the original library.

There were numerous reads of the R.Ferrumurquinum ACE2 mRNA in these "R.Affinis" "sequence capture" datasets. these are likely the original reads left behind--they added the fake in a few datasets, but have not removed the original.

It seems like that they got cocky with their R.Sinicus (which dont return any ACE2 fragments) and think that their purification is sufficient for datasets that can be witheld and waited for Shi to add in her fake ACE2. unfortunately, some of the originals have slipped through.

We could therefore conclude that ACE2 in R.Affinis is likely identical to R.Ferrumurquinum, as there were reads left from the original database.

Any datasets published after 14/05/2020 should not be trusted. https://t.co/UJXSoZqvMR THere is a reason why it took them over a month to sprinkle in trace of fake "RaACE2" in these datasets. (the Evolution article itself published all the ACE2-free bat datasets in 09/2019.

Annd the 06/01 datasets don't contain any ACE2 1-85, indicating that the methodology as in the study does not target and will exclude any traces of ACE2 1-85. Or any part of the VBM sequences. therefore, the sudden addition of these alleged "RaACE2" was the direct result of

the publication of the Shi article, which included a sheet containing "alignment of R.Affinis ACE2". they took the data and decided to alter a few Sinicus reads and add them to these witheld datasets.

they took the reads from Rf, altered the base call to fit "Ra", then added them to these "datasets".

Another possibility is that real ACE2 reads have been censored from the datasets that were published before the Shi article, probably to ensure that their fake RaTG13 don't fail on an early disclosure of a real RaACE2.

so that they can be consistent when they finally alters the trace reads in these "new" datasets. they witheld the old dataset and cleared away any ACE2 reads so that none of the early (Rf) reads have been left. (these Rf remains are "same across Rf and Ra").

There were also trace reads with a non-error-derived frameshift (Changing 2AA) within these datasets, which, should an alignment-then-alter system being used, will escape detection and ended up in the "01/07 datasets". if they Yeast displayed beginning from a real Ra clone,

the presence of this kind of frameshift within the original DNA library (found in both F and R reads) would indicate a very different RaACE2 that won't be able to bind RaTG13. Which, according to the protein translation, it appears to be the case for the frameshifted segment.

if they change the base call, a frameshift will likely not be registered.

As mammalian genomes are highly homogenous across the species, the fact that this altered ACE2 exist in R.Affinis data, with whatever mechanism, would exclude the Shi's "clones" as being accurate in any way. these changes will abolish binding to RBD, and will likely truncate it

in an actual bat, leading to solubilized ACE2 fragments and the pervalence of non-ACE2-utilizing SL-CoVs. (which is observed in R.Affinis.)

SMRT suggest that Ra loves to mis-splice ACE2 and majority of their ACE2 mRNA is a short isoform. coupled with this non-functional isoform, no wonder why there were so little Ra-infecting Sarbecoviruses, and why the vast majority of these were SL-CoVs. (potentially CD46 is used

by certain SARS-CoVs and also CD147..... could explain LyRa11. if this frameshifted ACE2 existed as a full-length isoform, it could explain LyRa11 but not RaTG13 (which would fail to bind).

As LyRa11 have the same configuration of RBM residues as CoV-2, the R24 version won't be able to bind LyRa11. this support the possibility that the post-14/05 R.Affinis sequencing have been withheld deliberately so that the ACE2 gene could be altered, as none of the previous

datasets (including two large datasets containing RaCytb) contained a fragment of nucleotide 1-85.

https://t.co/j8nBNMNBVs

Also the Ra SMRT data suggest a sinilar frameshift inactivation of the long isoform.

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