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Collapsing buildings of lies: SARS-CoV-2 comes from the laboratory - release intentional or misfortune? [SF survey]

Lotharfinger

2 weeks ago
Bad news for the facts gnome from dpa and for Nils Metzger from ZDF. They all leaned far out of the window and asserted with the conviction that only someone can have who does not even remotely have a clue of the matter they are talking about: SARS-CoV-2 comes from animals, was of zoonotic origin, jumped from bats to humans.

Folks, the scientific consensus that you value so much is changing massively right now. More and more inconsistencies in the story spun by researchers from the Wuhan Institute of Virology are surfacing and annoying scientists, and more and more criticism of the paper “The proximal origin of SARS-CoV-2” by Kristian G. Andersen, Andrew Rambaut, W. Ian Lipkin, Edward C. Holmes and Robert F. Garry, to which everyone refers who claim that SARS-CoV-2 is without a doubt and most certainly of animal origin, cannot be of human origin at all, is loud.

That went stupid for the cheeky fact checkers from dpa and ZDF.

dpa gnome

We have already discussed a number of studies and evidence in the past that raise considerable doubts that SARS-CoV-2 is of animal origin, which, on the contrary, suggest that SARS-CoV-2 came from a laboratory that it is the result of gain of function research carried out at the Wuhan Institute of Virology. We bring further evidence in this post that the official narrative of animal origin, which one wonders why the alleged fact checkers defend it so doggedly, is false. At the end of this text there is a new question that arises: Was the release of SARS-CoV-2 an accident or intent?

Let's start with a story that we haven't told yet.

The story begins with Aksel Fridstrom. Fridstrom is a journalist, one who really deserves the title. Fridstrom has found and made public a number of contradicting information in the chronology and description of the finding and sequencing of SARS-CoV-2. The text by Fristrom,

which came to our table only a few weeks ago, refers to this text that scientists from the Wuhan Institute of Virology published in Springer's “Nature” and in which they presented the discovery of SARS-CoV to the world -2 and its zoonotic origin in bats. Peng Zhou and Zheng-Li Shi, China's flagship virologists, provided the story on which Andersen et al. (see above) and on which the claim that SARS-CoV-2 is of animal origin goes back.

However, it did not take long for scientists to publicize their well-founded opinion that SARS-CoV-2 is not of animal origin. We have reviewed most of the papers in which this has happened on ScienceFiles:

Genetic Engineering: It is now almost certain that SARS-CoV-2 comes from a Chinese laboratory (new study)
Vow of silence: why scientists do not talk about a laboratory origin of SARS-CoV-2
New inconsistencies about the origin of SARS-CoV-2
SARS-CoV-2 is at least seven years old
The virus that came out of the laboratory: the chain of clues is getting longer
And it's not a natural virus
Toxic virus cocktail: China has a tradition of creating deadly viruses.
Designer Virus

The inconsistencies that Fridstrom pointed out concern RaTG13, the closest relative of SARS-CoV-2.

RaTG13 is the gene sequence of a coronavirus that is said to come from bats.

RaTG13 was introduced in the post by Zhou and Shi published in Nature on February 3, 2020 as follows:

"RaTG13 is the closest relative of 2019-nCoV and they form a distinct lineage from other SARSr-CoVs". […] Bat CoV RaTG13 was obtained from R. affinis, found in Yunnan province. ”

The genome of RaTG13 is 96.1% identical to the genome of SARS-CoV-2, according to the text published by 27 Chinese scientists working with Zhengli Shi from the Wuhan Institute of Virology (Zhou et al.). As of this article in Nature, the majority of scientists assume that RaTG13, the closest living relative of SARS-CoV-2, which is of animal origin, is evidence that SARS-CoV-2 is also of animal origin, comes from bats . But there are significant problems with RaTG13:

RaTG13, as a recently published study shows, is not able to infect bats at all. Strange when you consider that RaTG13 is supposed to come from bats, so it must have infected them somehow.

Mou, H. et al. (2020). Mutations from Bat ACE2 Orthologs Markedly Enhance ACE2-Fc Neutralization of SARS-CoV-2.

An analysis of reconstituted RaTG13 that Hron et al. (2018) also shows that RaTG13 is not able to bind to ACE2 receptors in R macrotis bats, the closest relative of R affinis bats, from which RaTG13 is said to be derived.

Hron, T., Farkašová, H., Gifford, R. J., Benda, P., Hulva, P., Görföl, T.,. . . Elleder, D. (2018). Remnants of an ancient deltaretrovirus in the genomes of horseshoe bats (Rhinolophidae). Viruses, 10 (4),

Now the publication date of the work by Hron et al. erring, because Zhou and Shi et al. have in their article in Nature, which appeared in 2020 and informed the world about RaTG13 as the closest relative of SARS-CoV-2, claimed that the gene sequence of RaTG13 was not deciphered until 2020, even though the name RaTG13 shows that this Coronavirus was already collected in 2013. That is one of the inconsistencies that Fridstrom picked up. The work of Fridstrom and other works that reveal inconsistencies have now resulted in an addendum that was negotiated between the editors of Nature and the authors of the WIV, as the email exchange that we have before us shows. We are in the process of processing the email change and will come back to it shortly.

The now new story is: RaTG13, of which there are no longer any physical remains, was actually collected in 2013 in a mine in Yuannan. Six miners who were tasked with cleaning the mine of bat droppings had previously fallen ill, three of whom died of pneumonia. Although RaTG13 was already collected in 2013 and, as it now turns out, was already discussed in 2016 in a work in which Zhou and Shi were again involved, both of them claimed in 2020 with others from the WIV that the gene sequence of RaTG13 had only just been decoded been. That, it now turns out, happened in 2018. An obvious lie? No, one of those oversights that has been increasing lately. The name of the coronavirus had been changed, so RaTG13 was not recognized as its predecessor, the coronavirus, which was discussed in 2016 and deciphered in 2018.

This is what scientists claim from a laboratory in which BSL (Biohazard Safety Level) 4 research is carried out. Not exactly reassuring. And not likely, as a brand new study just PUBLISHED in BioEssays by Wiley shows. The work of Rossana Segreto and Yuri Deigin, University of Innsbruck and Youthereum Genetics Inc. Toronto, Canada, picks up on the two special features that have distinguished SARS-CoV-2 since its discovery - its optimization for ACE2 receptors in human cells and its ability to react different affecting human organs. Both are properties that were unknown for coronaviruses before SARS-CoV-2. In addition to these properties, there are a number of coincidences that make SARS-CoV-2 a suspect companion:

SARS-CoV-2 shares 100% identity of the E protein with ZC45 and ZXC21. Nerd has Power has already pointed this out: If, according to the statement that Nerd Has Power suggests and pronounces, you take ZC45 and ZXC21 as a starting point in order to create a new coronavirus - for whatever reasons (the Chinese have a tradition of this) and concentrates on optimizing the receptor binding domain, the result would be a virus like SARS-CoV-2. That this is actually the case is shown by Yan et al. (2020). ZC45 and ZXC21 are coronaviruses that were also collected in Yunnan Province. They are both owned by the Chinese military. A 100% identity of the E protein, i.e. the shell that provides protection for the RNA of SARS-CoV-2, with other coronaviruses has a statistical probability that makes winning the lottery a piece of cake.
In order to produce SARS-CoV-2 on the basis of ZC45 and ZXC21, it is necessary to install the SARS-CoV Receptor Binding Domain and to optimize it somewhat. A problem? No:
“In 2008, Dr. Zhengli Shi's group [Wuhan Institute of Virology] swapped a SARS RBM [Receptor Binding Motif] into the Spike proteins of several SARS-like bat coronaviruses after introducing a restriction site into a codon-optimized spike gene. They then validated the binding of the resulted chimeric spike proteins with hACE2 [human ACE2]. Furthermore, in a recent publication, the RBM of SARS-CoV-2 was swapped into the receptor-binding domain (RBD) of SARS-CoV, resulting in a chimeric RBD fully functional in binding hACE2. " In both cases a virus emerged whose receptor binding domain was almost completely identical to that of SARS-CoV-2.

Researchers at the Wuhan Institute of Virology are therefore not only involved in research whose stated aim is to increase the infectiousness of viruses, they are also experts in the field of “swapping”, the replacement of gene sequences in a virus with Gene sequences from another virus.

A special gem of SARS-CoV-2 is the so-called Furin Cleavage Site, a segment of the Receptor Binding Domain of the spike protein of SARS-CoV-2, which is optimized for the human enzyme “Furin”. The result: the ability to infect human cells increases and the likelihood of a serious illness also increases. The Furin Cleavage Site, which SARS-CoV-2 is providing to improve its access to human cells and the ability to use the human cells for reproduction, is unique in its placement and nature, so unique that Yan et al . (2020) summarize the results of their analyzes as follows: “This observation strongly suggests that this furin-cleavage site should be a result of **genetic engineering**” (Yan et al. 2020).

Yan, Li-Meng, Kang, Shu, Guan, Jie & Hu, Sanchang (2020). Unusual Features of the SARS-CoV-2 Genome Suggesting Sophisticated Laboratory Modifications Rather Than Natural Evolution and Delineation of Its Probable Synthetic Role

Before we present the arguments of Segreto and Deigin, an excursion into the world of Chimera:

"The working definition of the term" chimera "is a new hybrid microorganism created by joining nucleic acid fragments from two or more different microorganisms in which each of at least two of the fragments contain essential genes necessary for replication."

This is what the United States Department of Agriculture writes. If a new virus, an optimized virus, is created from several sequences of different viruses, then this is such a hybrid, such a chimera. The Wuhan Institute of Virology is identified in the creation of Chimera. The Wuhan Institute of Virology creates new hybrid microorganisms as part of gain-of-function research. Harmless viruses are optimized so that they are no longer harmless:

- In 2007, a research group led by Zheng-Li Shi at the Wuhan Institute of Virology (WIV) produced chimeras from coronavirus spike proteins. The aim was to find out how coronaviruses manage to spread from animals to humans.

- In 2008, a group of researchers at the University of North Carolina took the work of the WIV one step further and created the chimera of a complete coronavirus.

- In 2015, both groups, the group from Wuhan and the group from the University of North Carolina, pooled their efforts and created another coronavirus chimera. Infecting cells of other organisms determined to be exchanged from a coronavirus from mice with the RBD from a coronavirus from bats.

- In 2017, Shi's group described the creation of eight new coronavirus chimeras in an article. In each case, the RBD was optimized so that the ability of the respective coronavirus chimera to attack the cells of other organisms was improved.

The prerequisites for creating SARS-CoV-2 in the laboratory, in the Wuhan Institute of Virology, are therefore in place. And against this background, the following sequence from the contribution by Segreto and Deigin cannot be read otherwise than as an indication, if there is no evidence that SARS-CoV-2 is not of animal origin but was created in the laboratory:

"*Before the SARS-CoV-2 outbreak, pangolins were the only mammals other than bats documented to carry and be infected by SARS-CoV-2 related CoV. [12] Recombination events between the RBD of CoV from pangolins and RaTG13 - like backbone could have produced SARS - CoV - 2 as chimeric strain. For such recombination to occur naturally, the two viruses must have infected the same cell in the same organism simultaneously, a rather improbable event considering the low population density of pangolins and the scarce presence of CoVs in their natural populations. X Moreover, receptor binding studies of reconstituted RaTG13 showed that it does not bind to pangolin ACE2.*[xi] "

In other words, it is as good as impossible that - on the basis of what is currently known about the genetic relationship of SARS-CoV-2 - SARS-CoV-2 developed as a mutation in nature, because this would be necessary that a coronavirus identified in pangolins (pangolins) and RaTG13 meet and infect THE SAME cell in order to mutate into SARS-CoV-2 together. The

likelihood of such a meeting is so small that one does not have to speculate about whether it could have happened that way. Especially not when you know that research has been carried out in the WIV for decades that combines exactly this type of Chimera, a coronavirus Chimera that uses a virus as a basis, RaTG13 and it with another virus (Pangolin CoV) to improve the hybrid in its ability, e.g. in its ability to infect people.

Segreto and Deigin therefore come to the following conclusion:

"***On the basis of our analysis, an artificial origin of SARS-CoV-2 is not a baseless conspiracy theory that is to be condemned[66] and researchers have the responsibility to consider all possible causes for SARS-CoV-2 emergence***. *The insertion of human-adapted pangolin CoV RBD obtained by cell/animal serial passage and furin cleavage site could arise from site-directed mutagenesis experiments, in a context of evolutionary studies or development of pan-CoV vaccines or drugs.*"

According to all the evidence that Segreto and Deigin cite in the course of their work, this is a very cautious assessment. For us there is hardly any doubt that SARS-CoV-2 comes from the WIV. The only question that remains for us is: Was the release of SARS-CoV-2 an accident or an intention?

We propose to the fact checkers of the dpa, who live the Dunning Kruger effect like hardly anyone else, to refute the work of Segreto and Deigin. Perhaps by referring to a press release from the Communist Party of China, which clearly states that China is not responsible for SARS-CoV-2.

What do our readers suspect: Does SARS-CoV-2 come from the laboratory and if so, the release was intentional or an accident. How did SARS-CoV-2 become a pandemic virus? Jumped from bats to humans Deliberately released from the laboratory Accidentally escaped from the laboratory How did SARS-CoV-2 become a pandemic virus? Jumped from bats to humans 15 (0.96%) Deliberately released from the laboratory 1029 (66.05%) Accidentally escaped from the laboratory 514 (32.99%) How did SARS-CoV-2 become a pandemic virus? Jumped from bats to humans 15 (0.96%) Deliberately released from the laboratory 1029 (66.05%) Accidentally escaped from the laboratory 514 (32.99%)

We have been discussing studies on SARS-CoV-2 since the end of January. This makes us one of the few who have followed the new coronavirus since it first appeared and have followed the effects it has found in scientific articles. The following texts document this activity in a way that makes us proud, as a private blog that can in no way compete with the financial resources of public institutions.

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