Is Covid-19 a bioweapon?

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Before 2002 human coronaviruses were considered relatively harmless, a common cause of flu. Unlike in many animals, coronaviruses did not cause serious diseases in humans. This is not any more the case. Since 2002 there have appeared three new serious human coronaviruses: SARS-CoV, MERS-CoV and SARS-CoV-2.

Coronaviruses are usually host specific: they attach to hosts with the spike protein and its particular shape normally fits only one host. The shape of the spike protein is determined by the S gene. Therefore, the S gene must have changed if a coronavirus jumps to a new host. This change cannot be a small set of point mutations as different animal species require quite different spike proteins. Consequently we find a larger change in the S gene in each three cases of coronaviruses (SARS-CoV, MERS-CoV and SARS-CoV-2) that have recently jumped from an animal host to humans. There are two possible reasons for this larger change. One is recombination, a natural process, and the other is genetic engineering.

The spike proteine is also the part of the virus that antibodies try to disable. There is one problematic thing concerning coronaviruses in general. Repeat infections are common with coronaviruses. It is because the immune response against these viruses is not complete or it is short lasting. It is possible that the spike protein changes over time so that antibodies do not give complete protection. This phenomenon can be noticed not only with SARS-CoV-2 but with all three. For instance, [3] tells that camels that were given a vaccine expressing the spike protein of MERS showed antibodies and a significant reduction of excreted infectious virus. That is, they still were infectious even after being vaccinated. Clearly, coronaviruses should not be underestimated.

SARS-CoV was found in 2002 in Guadong, China, where the SARS epidemic started. SARS-CoV originates in bats and the intermediate host is likely to be a civet. Himalayan palm civet. CoVs in a live-animal market in Guadong had nearly identical (99.8%) genomes to the human SARS-CoV [1].

Figure 2 in [1] gives the phylogenetic tree for SARS-CoV. The palm civet viruses are slightly different from human viruses. The earliest human viruses are from Guangdong. The immediate ancestor to civet and human viruses is not given in Figure 2 of [1]. Workers who were in contact with the animals, including the infected civets, did test positive for antibodies, but they did not have symptoms of SARS. It is unclear from this data if the civet virus was able to cause an epidemic in humans, or if some mutation was required. Table 3 in [1] shows several point mutations between the human CoVs and the civet CoVs, but the text informs that the animal viruses has an additional sequence which the human viruses lacked. The main differences were in the S gene. These differences show that the animals in this market were not the direct source of the human SARS epidemic. The authors of [1] expect that the true reservoir is somewhere else, but that it is an animal population. According to the preprint [2] SARS-CoV shares recombinant history with at least three different groups of bat CoVs, and for this reason phylogenetic trees for these coronaviruses must be considered with reservation: SARS-CoV did not just arise from a civet CoV. It either was a result of recombination events, as [2] claims, or it was engineered.

MERS-CoV was found in 2012. It is endemic in dromedary camels in East Africa and Middle East [3]. [3] suggests that the original reservoir of MERS-CoV was bats, as bats are the main reservoir for many types of coronaviruses. In 2009-2011 there was a large study of bats which showed that of ten tested bats in Ghana only one, Nycteris bat, had 2c-beta coronavirus (i.e., of the type of MERS-CoV). One third of Nycteris bats had the virus. 14.7% of Pipistrellus bats from four European countries had 2c-beta coronavirus. Both 2c-beta

coronaviruses are close to MERS-CoV. This indicates that the original reservoir is bats, but these bat viruses did not infect humans or domesticated animals. A South African bat was found in 2011 to have a virus that was very close to MERS-CoV. Feces of the Egyptial thomb bat were tested in Saudi Arabia in 2012 and match with MERS-CoV was 100% for a part of the genome. These findings support the claim that originally this virus derives from bats, but it did not directly jump to humans from bats.

MERS-Cov in camels is even more similar to human MERS-CoV than any bat virus. Archived serum samples from camels show that the virus was already common in camels in the early 1980s in Sudan and Somalia. No older serum samples are mentioned, probably there are no older samples. We can conclude that the virus was wide spread in camels at least for decades, maybe longer, but apparently it did not spillover to humans before the present decade. Dromedary camels in Australia and dromedary and bactrian camels Central Asia have tested negative to MERS-CoV. It follows that in camels the virus distribution is limited to Africa and the Middle East and dromedary camels cannot have had the virus at the time when camel caravans were still used for transport, that is before cars and trains took over the role of camels in transport.

There is good evidence that today camels act as a reservoir of the MERS-CoV virus and cause spillover to humans. A very high percentage of dromedary camels in some areas in the Middle East and Africa had antibodies for MERS according to [3]. Some camels had multiple strains of the virus, which is a necessary condition for natural recombination events. Multiple strains can be explained by the flow of new camels from different origins to the same physical area. Only 4.1% of camels in the Canary Island were seropositive to MERS-CoV and the seropositive camels were imported from Africa 20 or more years ago. Had these strains been infectious to humans, there would have been cases of MERS in tourists to the Canary Islands. It seems that the strain of MERS-CoV that can infect humans is much newer. Again we are in the same situation: the original camel virus could not infect humans. Very recently the S gene was modified. We have two possibilities, recombination or genetic engineering.

The preprint [2] confirms this by stating that all three new human CoVs (SARS, MERS and Covid-19) are a result of recombination of CoVs. The preprint does not explain what the recombination is in the case of MERS-CoV, it only says that it involved the S gene.
The preprint [2] sees the situation as evidence of the major role of recombination in the evolution of coronaviruses.

SARS-CoV-2 was found January 2020 in Wuhan, China, but it seems to have been born in November 2019 or a bit earlier. It is genetically most similar (96.3%) to RaTG13 CoV sampled from a bat in Yunnan, China, in 2013, but there is also similarity (91.2%) with Malaya pangolin CoV viruses from Guadong, China. The preprint 2] suggests that Covid-19 is a cross-species recombination between the bat and the Malaya pangolin CoVs. The preprint suggests that a bat CoV virus obtained the ability to infect humans from a pangolin CoV via cross-species recombination in ORF1a and S genes. Pangolins in China are originally from Malaya, but they seem to have been infected by CoV in China. This is shown by pangolins in two different districts of China having different strains of CoV. Indeed, if pangolins got CoV form bats, it could only be from Chinese bats. According to [2] the S gene in pangolin CoV closely resembles the S gene from SARS-CoV-2, though in other respect SARS-CoV-2 resembles bat CoV. Then a question is where from pangolins, recently taken to China, got the S gene. Obviously, recombination cannot answer this problem, unless another animal is found that originally had the S gene. This S gene of SARS-CoV-2 resembles the S gene in SARS-CoV, thus the answer may be that it is from SARS-CoV, but SARS died out and the gene is not completely identical. Genetic engineering as an answer does not have this problem.

Finding a virus nearly identical to SARS from a live-animal market in Guadong makes a good case for the origin of Covid-19 also being one of these markets. The SARS epidemic started from Guandong and the Wuhan live-animal market was an important early epicenter for Covid-19, but tracking different strains of SARS-CoV-2 does not support the idea that the Covid-19 virus originated in the Wuhan live-animal market.

There is another preprint, [4]. It studies the S gene of SARS-CoV-2 and finds insertations in it. These insertations [4] notices to resemble the HIV virus. The preprint [5] argues that this is not the case, but the insertions remain unexplained.

Coronaviruses are RNA viruses, as is the Ebola virus, found in 1976. The phylogenetic tree in [6] is drawn as a tree, so recombination is not a major behavior of this virus, but there has been a case of recombination in Zaire Ebola virus, described in [7]. A recombinant event between two lineages dated between 1996 and 2001 was found. This modified virus caused a series of Ebola outbreaks 2001-2003. The time of this recombination event is relatively recent, in the time frame when genetic engineering was possible.

Phylogenetic trees of traditional DNA viruses, like variola (smallpox)[8] and the measles virus [9] seem to be trees. The way variola jumped to humans is not known but [8] gives a convincing scenario. The human variola virus was born around 2000 BC in the Horn of Africa. At that time were created also a camel virus and a gerber virus. The dating and timing is fixed by the arrival of domestic camels to Africa and by the fact that the rodent lives only in a part of Africa. It may be so that the original ancestor to variola is a horse or camel. Both were domesticated around this time. It is interesting to speculate that the Bronze Age collapse in the Near East, thought to be caused by invasions of peoples with horses and iron weapons, may have been assisted by an epidemic of smallpox. That would explain why in the Bible God tells Israelites to burn all loot. Measles is a much newer disease. It developed from the rinderpest virus and jumped to humans between the 11th and 12th centuries [9]. No major role for recombination can be seen in these old infectious diseases.

As a conclusion, both possible explanations seem insufficient. The natural recombination explanation does not work with the pangolin CoV: in the recombination explanation a pangolin would have been infected with two CoV viruses, one from a bat with a S gene that does not infect humans, and the other from some other animal that has a S virus that can infect humans. Then the RNA of these viruses would recombine. The problem is that there is no such other virus and assuming that there is such a virus only moves the problem further. Finally all CoVs must go back to bat CoVs and the problem is not solved. The genetic engineering explanation has a problem with camel MERS-CoV, the one before the present one, i.e., the one in the Canary Islands where camels do not infect people. This virus was born before 1980s. Though some people, like the Oldmicrobiologist [10], claim that the US military was able to create the HIV and Ebola viruses, thus had this capability in the 1970s, most think that genetic engineering is relatively new. It cannot explain the camel MERS-CoV.

I think the best solution is that there is some mechanism that we do not know. It is the same problem that there always is with the birth of a new species: in order to get a new species some gene(s) need to change much. Point mutations cannot do it because if there are a bit more mutations the gene does not work. Then it becomes a pseudogene and there has to be a copy of the original gene in the genome to do the tasks this gene was supposed to do. As a pseudogene the gene is not working and natural selection cannot act on it. Thus, it can only mutate randomly until it by a lucky chance turns into a gene that can again do something useful and will be turned on. Random mutations seldom lead to anything that works. This problem there is, though Darwin believed that he solved it with natural selection, but selection works only with active genes, not inactive pseudogenes. The problem appears in CoVs in the S gene. In order to jump from one species to another, the S gene needs larger changes, some four insertations or recombination taking a whole piece. But where do you take the new piece

from in the first species jumping case? Nevertheless, viruses do jump from one species to another. This requires a new mechanism, or if you like, you can shift it to God's punishment. To the coming end-of-the-times and so on. Surely, it is coming, doubt it not.

With viruses random mutations just might produce enough changes to create a significantly different S gene because a virus population can be very large, but even genetics do not dare to suggest this possibility. Thus, [2] prefers to suggest recombination with a pangolin CoV and [4] recombination or genetic engineering with HIV. It is simply so that a claim that a successful step is a sequence of many random mutations without any guiding principle of selection is just too unbelievable, not only for mathematicians but also for genetics.

It is suspicious that three new deadly coronaviruses appeared in such a short time. It is also true that phylogenetic trees of SARS-CoV-2 (each author of the tree writes it a bit differently) show that many early strains of the virus were found in the USA. It may be that SARS-CoV-2 was man-made. But for sure the DNA virus variola was not genetically engineered when it jumped to humans around 2000 BC in the Horn of Africa. Later variola was human specific. There had to be a significant change in the genome of the virus for it to do the jump to humans. Viruses do these jumps. The real problem is not whether SARS-CoV-2 was man-made or a result of recombination. The real problem is to find a mathematically sound explanation how species specific viruses can jump from one species to another. It requires a larger change in the genome and it is unclear how this can happen in a finite time. There is only a mathematically unsound explanation that it is by evolution, natural selection and random mutations. But as was the case in theoretical physics when Max Planck gave a mathematically unsound derivation for blackbody radiation, unsound explanations are not acceptable. In Planck's case a sound derivation was later found, in the evolution theory there seems to be no effort to look for any sound explanations. Thus, we have suggestions that Covid-19 was genetically engineered by one of the possible culprits.

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