The Smoking Gun Proving SARS-CoV-2 Is an Engineered Virus

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STORY AT-A-GLANCE

- The National Institutes of Health have in recent years funded dangerous gain-of-function research on bat coronaviruses at the biosafety level 4 (BSL4) laboratory in Wuhan, China
- Gain of function research refers to research in which pathogenicity or transmissibility of pathogens is enhanced to make a pathogen more dangerous to humans
- To gain entry into a cell, the virus must first bind to an ACE2 or CD147 receptor. Next, the S2 spike protein subunit must be proteolytically cleaved. Without this protein cleavage, the virus would be unable to enter
- There are several enzymes that cleave spike proteins, including plasmin, which also degrades fibrin. When a blood clot is dissolved, a byproduct called D-dimer is created, and many patients with serious COVID-19 infection have elevated D-dimer, which is indicative of blood clots
- Another protein cleaver is furin, and the presence of a furin cleavage site on SARS-CoV-2 is "the smoking gun" that proves SARS-CoV-2 was lab-created

Since the breakout of COVID-19, a number of scientists have spoken out saying the virus does not appear to have evolved naturally, and those suspicions are only getting stronger.

As reported[1] by Newsweek April 28, 2020, the National Institutes of Health (NIH) has in recent years funded dangerous gain-of-function research on bat coronaviruses at the biosafety level 4 (BSL4) laboratory in Wuhan, China.

This research was backed by the **National Institute for Allergy and Infectious Diseases (NIAID), led by Dr. Anthony Fauci**, who is now heading up the White House pandemic response team. According to Newsweek:[2]

> *"In 2019, with the backing of NIAID, the National Institutes of Health committed $3.7 million over six years for research that included some gain-of-function work. The program followed another $3.7 million, 5-year project for collecting and studying bat coronaviruses, which ended in 2019, bringing the total to $7.4 million.*
>
> *Many scientists have criticized gain of function research, which involves manipulating viruses in the lab to explore their potential for infecting humans, because it creates a risk of starting a pandemic from accidental release."*

As noted by GM Watch,[3] "Bolstering the lab escape hypothesis in the eyes of the media is the news that the U.S. Defense Intelligence Agency (DIA) has updated its assessment of the **origin of the COVID-19 virus SARS-CoV-2** to reflect that it may have been accidentally released from a lab in Wuhan due to 'unsafe laboratory practices.'"

Unfortunately, mainstream media journalists are by and large ignoring the long history of accidental releases of dangerous pathogens from BSL3 and 4 laboratories. Journalist Sam Husseini discusses this history in a May 5, 2020 article in Independent Science News.[4]

Mainstream media journalists clearly are also not asking enough questions, or the right questions, about the origins of SARS-CoV-2. In his May 4, 2020, video update (above), Chris Martenson,[5] who has a Ph.D. in pathology, carefully details the science behind his assertion that SARS-CoV-2 must have undergone laboratory manipulation. The evidence he lays out is close to conclusive, and really would be front-page news if unbiased journalism still existed.

What Is Gain of Function?

As explained by Martenson, gain of function research refers to research in which the pathogenicity or transmissibility of pathogens is enhanced. In other words, pathogens are manipulated in various ways to make them deadlier, and/or allow them to infect humans with greater ease. They also take viruses that are harmless to humans and conduct experiments to make them transmissible to humans.

As noted by Martenson, while this kind of research is justified by saying we need to know how viruses adapt and mutate so we can more easily figure out how to combat them should they gain these functions naturally, there's not a shred of evidence suggesting we've learned anything about how to combat SARS-CoV-2. If we're not actually learning how to treat illnesses through gain-of-function research, then why are we doing it?

How Viruses Enter Your Cells

Martenson goes on to explain the two-stage process viruses use to gain entry into your cells. This is important, as viruses can only replicate by entering into and infecting a cell.

To gain entry, the virus must first bind to an ACE2 or CD147 receptor on the cell. Next, the S2 spike protein subunit must be proteolytically cleaved (cut). Without this protein cleavage, the virus would simply attach to the receptor and not get any further.

There are several enzymes that can do this job, including plasmin and furin. Plasmin, which is present in your blood, also degrades fibrin — plasma protein that can cause blood clots. When a blood clot is dissolved, a byproduct called D-dimer is created.

As explained in "**Might Enzymes Help Blood Clotting Associated With COVID-19?**" many patients with serious COVID-19 infection have elevated D-dimer, which is indicative of blood clots.

Martenson also cites the review paper[6] "Elevated Plasmin(ogen) as a Common Risk Factor for COVID-19 Susceptibility," which found that COVID-19 patients who have comorbidities that increase their susceptibility for the illness (i.e., those with high blood pressure, diabetes, coronary heart disease, cerebrovascular illness, chronic

obstructive pulmonary disease and kidney dysfunction), tend to have elevated levels of plasmin.

In other words, it's this elevated plasmin that — at least in part — puts these people at a higher risk for serious COVID-19 infection. In his May 6, 2020, update below, Martenson discusses this clotting problem encountered in many COVID-19 patients. As he points out, COVID-19 is "really more of a blood disorder, a clotting disorder," than a normal lung infection.

Furin Cleavage Site Is the 'Smoking Gun'

As mentioned, furin can also cut or cleave the S2 spike protein subunit. Furin is a protein coding gene that activates certain proteins by snipping off specific sections. As explained by Martenson, contrary to other protein-cutting enzymes, furin is very specific about the locations it cuts. What's more, when arginine is present in the second or third place of the protein sequence, then the efficiency of the cleavage is magnified.

This, he says, is "the smoking gun" that proves SARS-CoV-2 was created in a lab. An excellent, well-written article[7] in Medium also addresses this finding and explains why furin cleavage sites are so important for determining whether SARS-CoV-2 is natural or not.

In "Furin, a Potential Therapeutic Target for COVID-19,"[8,9] Chinese researchers report that CoV-2 is the only coronavirus with a furin cleavage site. Not even distant relatives of CoV-2 have it, and the coronaviruses that do have it share only 40% of CoV-2's genome. As reported in this paper:[10]

> *"It was found that all Spike with a SARS-CoV-2 Spike sequence homology greater than 40% did not have a furin cleavage site … including Bat-CoVRaTG13 and SARS-CoV (with sequence identity as 97.4% and 78.6%, respectively).*
>
> *The furin cleavage site 'RRAR' in SARS-CoV-2 is unique in its family, rendering by its unique insert of 'PRRA.' The furin cleavage site of SARS-CoV-2 is unlikely to have evolved from MERS, HCoV-HKU1, and so on.*

> *From the currently available sequences in databases, it is difficult for us to find the source. Perhaps there are still many evolutionary intermediate sequences waiting to be discovered*."

Mutation Cannot Explain Furin Site in SARS-CoV-2

According to these researchers, the furin cleavage site present in SARS-CoV-2 "is unique in its family" and "is unlikely to have evolved." In other words, the virus must have been modified somewhere along the way to give it a furin cleavage site, as there's no apparent source for this virus.

Put another way, there's no coronavirus out there that is similar enough that SARS-CoV-2 might have evolved or mutated from it.

Martenson does an excellent job of explaining this in his video, so I strongly recommend watching it. Yuri Deigin also does this in his Medium article,[11] so if you prefer reading, you can review much of the same data there.

Importantly, both reveal how virologists claiming SARS-CoV-2 is a natural bat coronavirus that jumped to pangolin and then to humans are simply wrong, and the genetic sequence proves it. The furin cleavage site PRRA found in SARS-CoV-2 is NOT found in either bats or pangolins, so it could not have mutated through these animals.

The fact that this furin cleavage site is present in SARS-CoV-2 is evidence that it has been inserted (opposed to mutated), and Martenson provides an easy to understand illustration of the difference between a mutation and an insert in his video. It is extremely unlikely that 12 new nucleotide base pairs would all of a sudden emerge from where there was nothing before.

What About the Studies Saying It's Natural?

Two studies heavily cited by mainstream media as evidence SARS-CoV-2 is a natural mutation that jumped from animal to human include a February 3, 2020, Nature paper,[12] which claims SARS-CoV-2 is a coronavirus of bat origin that then jumped species. However, one of the authors of this paper, Shi Zhengli, was involved in the

weaponization of the SARS virus, and therefore has reason to try to cover up any link to such research.

A second paper,[13] published in Nature Medicine, March 17, 2020, offers "a perspective on the notable features of the SARS-CoV-2 genome," and discusses "scenarios by which they could have arisen." According to this paper, "Our analyses clearly show that SARS-CoV-2 is not a laboratory construct or a purposefully manipulated virus."

However, even though they acknowledge SARS-CoV-2 has a polybasic cleavage site (PRRA) that does not exist elsewhere, they fail to explain how these 12 base pairs could have magically been inserted naturally. As noted by Martenson, "whole inserts are not part of the mutation pathway."

Scientific Community Has Reason to Hide Origin

He goes on to cite several studies showing how scientists around the world have been working on inserting cleavage sites to make coronaviruses more virulent. Clearly, we have the capability to create SARS-CoV-2, and scientists around the world have engaged in such research for many years.

Martenson calls out leading virologist Michael Osterholm who, in a March 10, 2020, interview with Joe Rogan, stated that "we could not have crafted a virus like this to do what it's doing; I mean we don't have the creative imagination or the skill set."

Really? Published research shows we clearly have the technology, know-how and "creative imagination" to create SARS-CoV-2, and Osterholm simply cannot be ignorant of that fact.

Another source you may want to look over is the Project Evidence webpage,[14] which lists more information pointing toward a lab-created SARS-CoV-2 than I could possibly cover here. A summary of the evidence can be found toward the bottom of the page under "Conclusion."

Naturally, there must be people in the scientific community who would now want to cover up any link to such research. Would you want to be responsible for creating, funding or having any association whatsoever with a virus responsible for a

pandemic that has killed people, destroyed the world economy and put people out of work around the globe?

Would you want to be found guilty of violating the Biological Weapons Anti-Terrorism Act of 1989, the punishment for which goes up to and includes life in prison? The Biological Weapons Anti-Terrorism Act of 1989 states:[15]

> *"Whoever knowingly develops, produces, stockpiles, transfers, acquires, retains, or possesses any biological agent, toxin, or delivery system for use as a weapon, or knowingly assists a foreign state or any organization to do so, shall be fined under this title or imprisoned for life or any term of years, or both. There is extraterritorial Federal jurisdiction over an offense under this section committed by or against a national of the United States."*

Other Experts Challenge Natural Evolution Claims

Martenson is far from alone in his belief that SARS-CoV-2 was genetically manipulated. An April 27, 2020, GM Watch article[16] features professor Stuart Newman, who also believes "genetic engineering may have been involved at some point in the virus' history."

According to Newman, a professor of cell biology and anatomy at New York Medical College and editor-in-chief of the journal Biological Theory, the argument used to deny that SARS-CoV-2 is a laboratory construct in the March 17, 2020, Nature Medicine paper mentioned earlier (which stated "Our analyses clearly show that SARS-CoV-2 is not a laboratory construct or a purposefully manipulated virus") actually points to the exact opposite. GM Watch writes:[17]

> *"As Adam Lauring, an associate professor of microbiology, immunology and infectious diseases at the University of Michigan Medical School, has noted,[18] Andersen's paper argues that, 'the SARS-CoV-2 virus has some key differences in specific genes relative to previously identified coronaviruses — the ones a laboratory would be working with. This constellation of changes makes it unlikely that it is the result of a laboratory 'escape."*

But Professor Newman says[19] that this is totally unconvincing because "The 'key differences' were in regions of the coronavirus spike protein that were the subject of genetic engineering experiments in labs around the world (mainly in the U.S. and China) for two decades' …

In an email interview with GMWatch, Newman … amplified this speculation by noting, 'The Nature Medicine paper points to variations in two sites of the spike protein of the new coronavirus that the authors claim must have arisen by natural selection in the wild.

However, genetic engineering of one of these sites, the ACE2 receptor binding domain, has been proposed since 2005 in order to help generate vaccines against these viruses (see this paper[20]). It is puzzling that the authors of the Nature Medicine commentary did not cite this paper, which appeared in the prominent journal Science …

The second site that Andersen et al. assert arose by natural means, a target of enzyme cleavage not usually found in this class of viruses, was in fact introduced by genetic engineering in a similar coronavirus in a paper[21] they do cite. This was done to explore mechanisms of pathogenicity.'

Newman said that he does not believe that these changes were deliberately introduced to increase the pathogenicity of any single strain, but that SARS-CoV-2 may have had genetically engineered components in its history before being inadvertently introduced into the human population."

There Are Many Ways to Manipulate Pathogens

Those who claim the lack of "fingerprints" in the genetic code of SARS-CoV-2 is evidence of natural evolution also fail to take into account methods that do not leave clearly identifiable traces. As noted by Dr. Meryl Nass (my interview with her will be posted May 24):[22]

"Prior to genetic engineering techniques being developed (1973) and widely used (since late 1970s), more 'primitive' means of causing mutations, with the intention of developing biological weapons, were employed …

They resulted in biological weapons that were tested, well-described, and in some cases, used … These methods can result in biowarfare agents that lack the identifiable signature of a microbial agent constructed in a lab from known RNA or DNA sequences.

In fact, it would be desirable to produce such agents, since it would be difficult to prove they were deliberately constructed in a lab. Here are just a few possibilities for how one might create new, virulent mutants:

1. *Exposing microorganisms to chemical or radiological agents that cause high mutation rates and selecting for desired characteristics*
2. *Passaging virus through a number of lab animals or tissue cultures*
3. *Mixing viruses together and seeking recombinants with a new mix of virulence factors"*

In my opinion, the strongest pieces of evidence so far all point toward SARS-CoV-2 being a laboratory creation. As Martenson asserts, the presence of furin cleavage sites[23] makes a clear case for this, as this section of genetic code wouldn't just emerge by itself by way of natural mutation. How it got released, however, is anyone's guess.