Wuhan Corona Virus is Manmade.
COVID-19: What China's Hiding Is Really Frightening by Shah Gilani

If the coronavirus that causes COVID-19 isn't nearly as contagious as influenza viruses are, why did China shut down Wuhan, a huge manufacturing city, and quarantine 46 million people?

What don't we know about the virus?

What are world governments really frightened about?

This latest coronavirus report answers those questions to try and break down what's really going on…

The Origin of the Virus
At this point in the brief history of the coronavirus, the only thing scientists seem to agree on is that it originated in Wuhan, Hubei province, China, based on where the first recognized infections were diagnosed.

Researchers haven't been able to definitively determine the origin of the new virus.

Or have they?

The virus that causes COVID-19 appears, for the most part, as a zoonotic coronavirus, which is a virus that's transmuted from infecting animals to infecting humans.

The SARS (Severe Acute Respiratory Syndrome) betacoronavirus, which emerged in Guangdong province, China, in 2002, is a zoonotic coronavirus that originated in bats, then likely infected civet cats and transmuted to infect humans.

Based on its genomic structure, COVID-19, also a betacoronavirus, is a single-stranded RNA virus of zoonotic origin that comes from bats.

However, scientists discovered disturbing anomalies in the long strand of the new virus's RNA.

Dr. Yuhong Dong, who holds a doctorate in infectious diseases from Beijing University, wrote in The Epoch Times, "Based on recently published scientific papers, this new coronavirus has unprecedented virologic features that suggest genetic engineering may have been involved in its creation." He added, "The virus presents with severe clinical features; thus, it poses a huge threat to humans."

Studying the virus's phylogenetic tree, its full genome sequence, Dr. James Lyons-Weiler, founder and CEO of the Institute for Pure and Applied Knowledge (IPAK), identified a long unique sequence of 1,378 nucleotide base pairs not found in other coronaviruses. According to Dr. Lyons-Weiler, the "novel sequence" lacks homology (similarity due to shared ancestry between a pair of structures, or genes) between similar bat coronaviruses. He says the inserted sequences should not be there.

Dr. Lyons-Weiler points to the presence of a SARS-binding protein sequence in the new coronavirus that allows it to easily infect human cells.

He explains, "Despite considerable genetics distance between the Wuhan CoV and the human-infecting SARS-CoV, and the overall low homology of the Wuhan CoV S-protein to that of SARS-CoV, the Wuhan CoV S-protein had several patches of sequences in the receptor binding (RBD) domain with a high homology to that of SARS-CoV. The residues at positions 442, 472, 479, 487, and 491 in SARS-CoV S-protein were reported to be at receptor complex interface and considered critical for cross-species and human-to-human transmission of SARS-CoV.

So, to our surprise, despite replacing four out of five important interface amino acid residues, the Wuhan CoV S-protein was found to have a significant binding affinity to human ACE2. The Wuhan CoV S-protein and SARS-CoV S-protein shared an almost identical 3-D structure in the RBD domain, thus maintaining similar van der Waals and electrostatic properties in the interaction interface. Thus, the Wuhan CoV is still able to pose a significant public health risk for human transmission via the S protein-ACE2 binding pathway."

Dr. Yuhong Dong of Beijing University asks, "How could this novel virus be so intelligent as to mutate precisely at selected sites while preserving its binding affinity to the human ACE2 receptor? How did the virus change just four amino acids of the S-protein? Did the virus know how to use Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) to make sure this would happen?"

A study by researchers at the University of Texas at Austin also found the novel coronavirus and SARS share the same functional host-cell receptor, called angiotensin-converting enzyme 2 (ACE2). They warn, "The deadly new coronavirus is up to 20 times more likely to bind to human cell receptors and cause infection than severe acute respiratory syndrome."

The report, published on the website bioRxiv.org (pronounced "bio-archive"), a free online archive and distribution service for unpublished preprints in the life sciences, operated by Cold Spring Harbor Laboratory, a not-for-profit research and educational institution, said the new coronavirus "had around 10- to 20-fold higher affinity – the degree to which a substance tends to combine with another – for human ACE2 compared with SARS." The researchers added that further studies were needed to explore the human host-cell receptor's role in helping the new virus to spread from person to person.

One theory surrounding the novel coronavirus's extraordinary ability to bind to host receptors is that it was engineered to do just that.

Chinese scientists have been working on a SARS vaccine. It's possible they engineered the RNA of their vaccine to make it more receptive to humans.

The engineered "binding" sequence is similar to the pShuttle-Sn recombination vector abs INS1378 patented by the Chinese, using it to develop an immunogenic vaccine against SARS.

According to the patent filing (translated from Chinese), "The present invention belongs to the field of genetic engineering, particularly relates to adenoviral vector SARS vaccines, their preparation and coronavirus S genes in SARS (SARS) on vaccines for the prophylaxis. By means of biological engineering, the coronavirus S gene in combination with deficient recombinant adenovirus, the protective immunogen protein or polypeptide expressed therein, through expansion culture, purification, and formulation to prepare a mucosal immunogenicity can cause the gene vaccine, respiratory mucosal immune response induced by the body, to produce antibodies against the virus infection. Specific conditions of the present invention,

compared with conventional inactivated virus particle vaccine, safe, easy to use, without limitation intramuscular, have broad clinical applications."

If the new coronavirus is an experimental SARS vaccine that escaped a research lab, the Chinese should tell the world.

But that's not the whole story.

An HIV Connection?
On Jan. 27, 2020, researchers Prashant Pradhan, Ashutosh Kumar Pandey, Akhilesh Mishra, Parul Gupta, Praveen Kumar Tripathi, Manoj Balakrishnan Menon, James Gomes, Perumal Vivekanandan, and Professor Bishwajit Kundu, from the Indian Institute of Technology, published a paper on bioRxiv.org.
The authors of the paper, titled, "Uncanny similarity of unique inserts in the 2019-nCoV spike protein to HIV-1 gp120 and Gag,"claim they found four insertions in the spike glycoprotein (S), which are unique to the 2019-nCoV and are not present in other coronaviruses.

"Importantly, amino acid residues in all 4 inserts have identity or similarity to those of HIV-1 gp120 or HIV-1 Gag," the team reported. They wrote, "Interestingly, despite the inserts being discontinuous on the primary amino acid sequence, 3D-modelling of the 2019-nCoV suggests that they converge to constitute the receptor binding site. The finding of 4 unique inserts in the 2019-nCoV, all of which have identity/similarity to amino acid residues in key structural proteins of HIV-1 is unlikely to be fortuitous in nature."

Pradhan added, "To our surprise, these sequence insertions were not only absent in S-protein of SARS but were also not observed in any other member of the Coronaviridae family. This is startling as it is quite unlikely for a virus to have acquired such unique insertions naturally in a short duration of time."

The paper states, "Unexpectedly, all the insertions got aligned with Human immunodeficiency Virus-1 (HIV-1). Further analysis revealed that aligned sequences of HIV-1 with 2019-nCoV were derived from surface glycoprotein gp120 (amino acid sequence positions: 404-409, 462-467, 136-150) and from Gag protein (366-384 amino acid). Gag protein of HIV is involved in host membrane binding, packaging of the virus and for the formation of virus-like particles. Gp120 plays a crucial role in recognizing the host cell by binding to the primary receptor CD4. This binding induces structural rearrangements in GP120, creating a high-affinity binding site for a chemokine co-receptor like CXCR4 and/or CCR5."

It is well known that CD4 cells are essential to human immunity and are the direct targets of the Human Immunodeficiency Virus, or HIV. HIV attaches to CD4 cells, enters, and infects them. The virus then turns each infected CD4 cell into a factory creating more HIV virus until eventually all CD4 cells are destroyed.

Other researchers chimed in, reporting, "If we take a closer look at the 4 insertions of the S-protein… they are all located on the binding surface of the protein, seemly designed to be able to bind to target cell receptor sites. Natural accidental mutation would be randomly distributed across the whole length of the S-protein. It is highly unlikely that all of these insertions would coincidentally be manifested on the binding site of the S-protein."

The article by Pradhan et. al., a preprint made available through bioRxiv, has not to-date been peer-reviewed and has been "withdrawn."

According to bioRxiv, "This paper has been withdrawn by its authors. They intend to revise it in response to comments received from the research community on their technical approach and their interpretation of the results. If you have any questions, please contact the corresponding author."

BioRxiv's website says, by posting preprints on bioRxiv.org, "authors are able to make their findings immediately available to the scientific community and receive feedback on draft articles that are not peer-reviewed, edited, or typeset before being posted online. However, all articles undergo a basic screening process for offensive and/or non-scientific content and for material that might pose a health or biosecurity risk and are checked for plagiarism. No endorsement of an article's methods, assumptions, conclusions, or scientific quality by Cold Spring Harbor Laboratory is implied by its appearance in bioRxiv. An article may be posted prior to, or concurrently with, submission to a journal, but should not be posted if it has already been accepted for publication by a journal."

If the new coronavirus has an HIV-like mutation, its ability to bind with human cells could be up to 1,000 times as strong as the SARS virus, according to scientists in China and Europe.

When looking at the genome sequence of the new coronavirus, Professor Ruan Jishou of Nankai University in Tianjin found a section of mutated genes that did not exist in SARS but were similar to those found in HIV and Ebola.

"This finding suggests that 2019-nCoV [the new coronavirus] may be significantly different from the SARS coronavirus in the infection pathway," the scientist said in a paper published on Chinaxiv.org, a platform used by the Chinese Academy of Sciences to release scientific research papers before they have been peer-reviewed.

"This virus may use the packing mechanisms of other viruses such as HIV."

According to the study, "The mutation can generate a structure known as a cleavage site in the new coronavirus's spike protein. The virus uses the outreaching spike protein to hook on to the host cell, but normally this protein is inactive. The cleavage site structure's job is to trick the human furin protein, so it will cut and activate the spike protein and cause a "direct fusion" of the viral and cellular membranes."

Compared to the SARS way of entry, this binding method is "100 to 1,000 times" as efficient, according to the study.

In a follow-up study, a research team led by Professor Li Hua from Huazhong University of Science and Technology in Wuhan, Hubei province, confirmed Ruan's findings.

"The mutation could not be found in SARS, MERS, or Bat-CoVRaTG13, a bat coronavirus that was considered the original source of the new coronavirus with 96 percent similarity in genes," it said.

This could be "the reason why SARS-CoV-2 is more infectious than other coronaviruses," Li wrote in a paper released on Chinaxiv.

"Compared with SARS-CoV, 2019-nCoV [which is another medical term for the virus that causes COVID-19, same as with SARS-CoV-2] appears to be more readily transmitted from

human to human," the report of the study said. "The high affinity of 2019-nCoV S for human ACE2 may contribute to the apparent ease with which 2019-nCoV can spread from human to human."

A good reason to panic.

Like almost everything related to COVID-19, we don't know for sure what the origin of the virus is, if the virus was engineered as a SARS vaccine, or if it was engineered with HIV elements.

We do know, however, doctors are using Kaletra – the brand name for a combination of ritonavir and lopinavir, two antiretroviral medications used to fight HIV – to fight COVID-19.

We also know that this novel coronavirus isn't supposed to be as dangerous as the flu, but it's got governments around the world trying not to concern their citizenry.

Maybe they should.

Whatever governments know, they should tell us. And based on the extreme measures taken in China, now Italy, and elsewhere, they know a lot more than they're letting on.

Obviously, governments don't want to create panic. But maybe they should.

If COVID-19 is a far greater threat than we're being told because it's an out-of-control engineered vaccine or a bioweapon, maybe controlled panic – which is already evident – is justified. And maybe it's what's necessary to get global governments, scientists, doctors, and the public immediately working together to eradicate this potentially dangerous threat.

Dr. Yuhong Dong, from Beijing University, says, "It is imperative for scientists, physicians, and people all over the world, including governments and public health authorities, to make every effort to investigate this mysterious and suspicious virus in order to elucidate its origin and to protect the ultimate future of the human race."