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“Origins of COVID-19: An Examination of Available Evidence”
Senate Committee on Homeland Security and Governmental Affairs

Committee Chair Senator Peters, Ranking Member Senator Paul, Members of the Senate Committee on Homeland Security and Governmental Affairs, invited Senator committee participants, Ladies and Gentlemen.

I am honored to participate in this forum entitled: “Origins of COVID-19: An Examination of Available Evidence.”

I am a physician scientist, a medical doctor and organic chemist, and have a 50-year career that spans academic research in medicine, biotechnology, and scientific fraud investigation.

As a young resident at the Harvard teaching institute, the Massachusetts General Hospital, I was responsible for uncovering a decade of fraudulent cancer research by my professor-advisor. Research that included Nobel Lauret David Baltimore. Nicholas Wade and William Broad, then of the New York Times, wrote a book about my investigative work entitled, *Betrayers of the Truth*. There were hearings in Congress at the time to examine scientific fraud involving NIH-funded research.

In many ways, the last four years of my analysis of the origin of the SARS2 virus has felt like a return to that previous time, including the push back I received at the time from elite academics in the medical schools involved with the fraudulent research and the NIH, until the professor finally confessed to his misconduct.

In academics I have over 390 publications, including thirty-two on the origin of COVID-19, that have been cited over 12,000 times, placing me in the top 1% of scientists worldwide. The paper I wrote for the State Department in late 2020, entitled, “A Bayesian analysis concludes beyond a reasonable doubt that SARS-CoV-2 is not a natural zoonosis but instead is laboratory derived,” has been viewed over 206,000 times. It is included in my background information for this hearing.

In medicine I was part of the Harvard Medical School team that discovered exosomes, virus-sized entities that tumors make to help them defeat the immune system, and with National Academy of Medicine President, Victor Dzau, made fundamental discoveries about the physiology and biochemistry of renin and its role in hypertension. These studies have led to the highly successful anti-VEGF oncology drugs and the renin inhibitors that treat hypertension.

In biotechnology, I have 238 patents and applications in 22 areas of medicine, including inventions in the chemistry of RNA as used in the COVID vaccines, and in therapeutics for coronaviruses and influenza. I have invented seven FDA-approved pharmaceuticals that have helped over 80 million people worldwide.

I am currently the founder and CEO of Atossa Therapeutics, a clinical stage public company advancing my patented drug, Z-endoxifen, for the prevention and treatment of breast cancer and other diseases.

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I am, however, speaking in my capacity as an independent scientist and in no other role.

I would like to acknowledge my many collaborators in the investigation of the origin of COVID-19. These are individuals from all continents, walks of life, and professional skills who have worked tirelessly, and without compensation, to investigate this pandemic. Many of them are part of a loosely affiliated group on the internet known as DRASTIC.

In my opinion, their contribution to uncovering the origin of SARS2 should be formally recognized at some point.

I do not receive any funding from either NIH or NIAID and therefore do not have a conflict of interest with respect to my testimony as it relates to the topics of today’s hearing. Scientists whose livelihood depends on NIH or NIAID funding may have pressure to publicly agree with orthodoxies that, in their private communications with their colleagues, they admit are not correct.

We have seen examples of this very problem in FOIA-revealed private communications between NIH and NIAID leaders and the virologists who are the vanguard of the public facing communication about the origin of SARS2. I believe this is a form of academic fraud that should not go unpunished.

The public deserves to believe that when they send their hard-earned tax dollars to the federal government and their money is used to fund research to solve or prevent serious public health problems, those scientists doing the work can be trusted to be truthful. That those scientists think and speak congruently in their public and private communications and analyses about their fields of expertise.

My prepared remarks will take about seven minutes. A more detailed set of remarks has been provided to be entered into the record.

My approach to the origin of the COVID pandemic that killed 15+ million people worldwide, caused over 20 trillion dollars in economic damage, and changed the world forever is based on six approaches to the data and the events.

Before I start, I want to note something Dr. Garry said privately: “Someone should tell Nature (meaning the British journal) that the fish market probably did not start the outbreak.”

While it would be easy for me to just agree with Dr. Garry and call it a day, I am a scientist and so will provide my independent analysis.

I will describe the six approaches at a high level and then go into each one in detail.

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First, the virus was spreading in Wuhan in the early fall of 2019, two to four months before the first case in the Hunan Seafood Market. This is supported by fourteen observations or evidence. This should be sufficient to dismiss the Hunan Market as the source of the outbreak.

Second, I look at the data from the market, including human infections, animal samples, and environmental specimens. This involves looking at eight observations or evidence. None of these data are consistent with an infected animal passing SARS2 to a human at the market.

Third, documented events at or related to the Wuhan Institute of Virology, or WIV, beginning in March 2019, are consistent with the expected activities of a virology lab in which a laboratory-acquired infection has occurred. I will go through that timeline.

Fourth, the evidence that is found in a natural zoonosis with respect to the animal host, the virus, and the human population in the vicinity of the outbreak is missing for the COVID pandemic. Each of these three components of a zoonosis will be examined separately and each will be found wanting.

Fifth, the genome of SARS-CoV-2 has seven features that would be expected to be found in a virus constructed in a laboratory and which are not found in viruses from nature. The statistical probability of finding each feature in nature can be determined and the combined probability that SARS2 came from nature is less than one in 1.2 billion.

These same features were described in a grant application submitted to DARPA in 2018 by scientists from the WIV, together with US collaborators.

Sixth and final, the earliest genomes of SARS2 were unstable and could not have come from an animal host without the stabilizing mutation, the so-called D614G change, that appeared in human viruses beginning January 1st, 2020. The consequence of this is that I can conclude that the first human infection occurred soon after the insertion of the furin cleavage site in the laboratory and before extensive animal testing. Otherwise, the first human cases would have had this stabilizing mutation.

It also means that the unstable version of SARS2 could not have been circulating in animals, otherwise it would have acquired the stabilizing mutation. If any virologist can find an animal host that can transmit the unstable ancestral SARS2 five or more times without obtaining the stabilizing mutation, they have found a hypothetical candidate for a spillover host. All testing to date of potential hosts has failed this test.

Finally, while not related to the origin, I will end by describing the unpublished dangerous research being done at the WIV on a MERS virus, 30% lethal to humans, and a Nipah virus, >70% lethal to humans.

My preliminary analysis is that any epidemic with a 15% or greater lethal virus will cause a civilization collapse that will last longer than 250 years.

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The time since the Revolutionary War ended.

Let me begin my analysis.

First, the virus was spreading in Wuhan and around the world in the early fall of 2019, two to four months before the first case in the Hunan Seafood Market. This is supported by fourteen observations or evidence.

1. May to August 2019: SARS2 mutates, changes a letter in its genetic sequence, about once every two weeks. This molecular clock, as it is called, allows scientists to use a large collection of virus sequences to determine the ‘time to most recent common ancestor,’ the date when the first human infection occurred. The bigger the data set, the more accurate the date. One dataset of 86,582 genomes, found the date to be August 16, 2019. A larger study, involving 3.14 million genomes, dates the start to between May and October 2019.

Dr. Garry and colleagues used a tiny data set of 787 genomes and came up with the date of November 18, 2019. This data set has three problems: it is way too small to pick up the root of the ancestral virus. It is also way too short an amount of time, having been truncated in mid-February and thus could have had, at most, about eight mutations since the first virus introduction. And it involves only Chinese sequences and not sequences from outside China.

The work of Jesse Bloom to uncover genomes from Chinese patients that had been uploaded to the NIH GeneBank site and then removed was very revealing. He found not only both Lineage A and Lineage B sequences, but sequences containing some of the three mutations that had been predicted to be in more ancestral viruses than Lineage A. This means if you build your phylogenies with Chinese data only, you are using documented sets of genomes that have been curated to remove temporal signals from before December 2019. This is what the Dr. Garry study did.

In his Congressional testimony, Dr. Ralph Baric, arguably the world’s expert on coronavirus genetics, disputes Dr. Garry’s date of November 18, 2019. He said: “If you look at the molecular clock of the virus, it emerged in the middle of October, late October, not the middle or end of November. So people who say that those were the first cases, no chance. There were five or six transmission cycles at least before they would have been infected.”

2. September 3, 2019: COVID antibodies are found in blood samples from the Veneto region of Italy.

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3. September 4, 2019: COVID antibodies found in two blood samples taken from Lombardi, Italy,
4. September 13, 2019: 13 blood samples from Lombardy, Italy, positive for COVID antibodies.
5. September 29 to November 21, 2019: Professor Yu Chuanhua, the Vice President of the Hubei Health Statistics and Information Society and Professor of Epidemiology and Health Statistics at Wuhan University, reported seven cases of CT-diagnosed COVID before November 21, 2019.
6. September and October 2019: Satellite imagery of Wuhan showed a significant uptick in the number of people at six local hospitals surrounding the WIV’s headquarters, coupled with an unusually high number of patients with symptoms similar to COVID-19.
7. September 2019: An African International PhD Student at Wuhan University revealed an outbreak of unusual and severe pneumonia cases starting in September 2019 in Wuhan.
8. October 1 to December, 10, 2019: A Chinese national investigation team “wished to trace early cases of the disease. It asked local authorities for data from all Wuhan medical institutions over the period between Oct. 1 to Dec. 10, 2019.”
 - a. The records obtained by this team showed more than 40 suspected COVID-19 patients across eight hospitals, including 9 deaths due to COVID-19-like conditions at three specific hospitals.
9. Mid-October 2019: Deputy US Consul General in Wuhan, Russell Westergard, said: “By mid-October 2019, we knew that the city had been struck by what was thought to be an unusually vicious flu season. The disease worsened in November. When city officials began to close public schools in mid-December to control the spread of the disease, the team passed the word to Embassy Beijing and continued monitoring.”
10. October 18, 2019: The 7th International Military World Games opened in Wuhan. The games are similar to the Olympic games but consist of military athletes with some added military disciplines. The Games in Wuhan drew 9,308 athletes, representing 109 countries, to compete in 329 events across 27 sports. Twenty-five countries sent delegations of more than 100 athletes, including Russia, Brazil, France, Germany, and Poland.

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- a. Four athletes reported getting sick with COVID-like illnesses during the games. Two of these athletes competed at venues near the WIV, two did not. No venues were near the Hunan Market.
 - b. Blood samples from six out of the 138 Spanish military athletes who travelled to Wuhan Games tested positive for coronavirus antibodies.
 - c. A Canadian athlete stated: “[I got] very sick 12 days after we arrived, with fever, chills, vomiting, insomnia.... On our flight to come home, 60 Canadian athletes on the flight were put in isolation [at the back of the plane] for the 12-hour flight. We were sick with symptoms ranging from coughs to diarrhea and in between.”
 - d. Five countries that sent athletes to the Games reported community cases in November and December 2019.
 - i. A 4-year-old boy in Milan, Italy, on November 21, 2019, had COVID by PCR.
 - ii. Wastewater samples in Brazil were positive on November 27, 2019, for SARS2 by PCR.
 - iii. Wastewater sampling from Milan, Italy, were positive on December 18, 2019.
 - iv. Sweden’s Public Health Agency said individuals in the country were infected with SARS-CoV-2 as early as November 2019.
 - v. A man in France presented to an emergency room on December 27, 2019, with PCR-confirmed COVID.
11. November, 2019: In another Chinese document, nine Covid-19 cases were identified in November 2019 (4 male and 5 female aged between 39 and 79).
12. November, 2019: A California company, Thermogenesis, said “they knew about the outbreak back in November 2019” when speaking about a rapid antibody test they developed. Chris Xu is Chairman and CEO of ThermoGenesis and is also a Professor at Peking University.
13. November 25, 2019: Connor Reed, a Wuhan school teacher originally from Wales, was perhaps the most famous Westerner in Wuhan who got COVID (PCR-confirmed), recovered, underwent the lockdowns, etc. He was a staple figure on British TV giving his on the ground account of what happened. Importantly, despite his high visibility and profile to Western journalists, his case is not contained with “official Chinese records.” This proves Chinese records have been corrupted.
14. December 13, 2019: COVID antibodies found in blood samples from Washington state, Oregon, and California.

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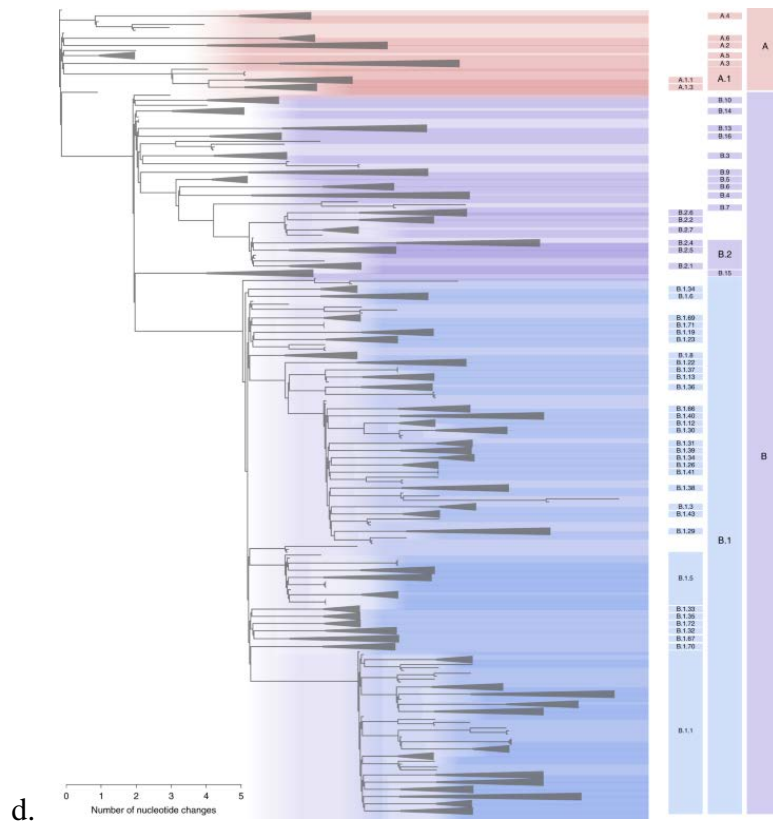
These fourteen observations provide evidence the virus was spreading in Wuhan and outside Wuhan in the early fall of 2019, two to four months before the first case in the Huanan Seafood Market. All market cases have onset in December 2019, and thus are well after the outbreak began. This establishes the market is not the origin.

Second, the data from the market, including human infections, animal samples, and environmental specimens, do not support a zoonosis having happened at the market. This involves looking at eight observations or evidence.

1. The first patient identified in the WHO report was infected on December 8th and has no relationship to the Huanan Market (none of the patient, family members and other contacts had history of exposure to Huanan Market).
 - a. The market he visited was the RT-Mart supermarket in Jiangxia District, which is more than 20 km away from the Huanan Market. This supermarket is a modern western-style market which does not sell live animals.
 - b. In a report entitled, “Early appearance of two distinct genomic lineages of SARS-CoV-2 in different Wuhan wildlife markets suggests SARS-CoV-2 has a natural origin,” Dr. Garry has incorrectly suggested this patient is evidence that multiple markets were involved with the outbreak. The market he visited did not have wildlife.
2. No animal in the market or in the market supply chain was infected.
 - a. 457 specimens from eighteen species of animals were collected at the Huanan Market, related warehouses, and other markets, and all were negative for SARS2 by PCR.
 - b. 616 specimens from market suppliers were tested and were all negative for SARS2 by PCR.
 - c. With SARS1 or MERS, >90% of the civet cats or camels in the markets were infected. The supply chain to the markets for these epidemics also documented infections.
3. All human infections in the market were the non-ancestral virus, Lineage B. They should have been Lineage A if the market was the origin.
 - a. One of my frustrations with the COVID origin question is watching established scientists using an outcome-based process to do science around the origin. What do I mean by that?
 - b. The process of finding the root virus in an outbreak has been established over decades of work with multiple viruses and multiple outbreaks.
 - c. When Rambaut and Holmes and colleagues studied 27,767 complete SARS-CoV-2 genomes through May 18, 2020, they found a solid lineage tree rooted with Lineage A before Lineage B. Here is what it looks like:

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- e. The pink Lineage A at the top has multiple genomes with earlier sequences compared to Lineage B.
- f. This is what you would call settled science. Everyone agreed Lineage A came before Lineage B.
- g. But then a problem arose. The market didn't have any Lineage A viruses in patients and only one Lineage A environmental specimen that has signs of contamination. All of the patients and 72 out of 73 environmental specimens had a version of the virus that was not the first human virus but arose from the first virus.
- h. This would negate the market as the origin.
- i. Furthermore, there became a pattern of patients who had no connection to the market having Lineage A infections. This is what you would expect from an epidemic that began with Lineage A.
- j. All of the data was showing that the pandemic began with Lineage A outside of the market and it had evolved to Lineage B and only then appeared in the market.**
- k. So, what do the virologists do? They stand on their head, create new computer software, and announce that their software has found that Lineage B came before Lineage A and then that there were two spillovers of both viruses.

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- l. Without getting into the weeds, this work has been seriously challenged, including the authors having to make changes in the results that were published.
- m. How serious is this issue? If all of the patients in the market had a Lineage A virus that would be good, solid evidence it came from this market. But the opposite is also true.
4. Only one environmental specimen contains a Lineage A SARS2 sequence. The specimen is an outlier according to the PCR and NGS data and has mutations that were not seen anywhere in the world until spring 2020. This is most consistent with the specimen being a post-collection, laboratory contamination during the processing of the specimen in the spring of 2020.
5. Only 10 of the 678 stalls in the market sold wildlife, overseen by 10 vendors. None of these 10 vendors had COVID. With SARS1 three out of the first five human cases found on premises relevant to the wildlife trade were directly engaged with the trade, two civet butchers, and one driver shipping wild animals into Guangdong. Here the wildlife vendors were not sick.
6. I have been asked well, maybe the vendors lied about not being sick because of the consequences of selling wildlife in the market. Well possibly, but if you are going to go down that road you must allow for symmetrical lying. That is, when Dr. Shi says she did not have SARS2 in the lab she might be lying and saying that because of the consequences of having contributed to the virus that caused the pandemic. You can't use a lying hypothesis to support the market spillover and deny that hypothesis to the lab origin scenario.
7. There is complete agreement that the closest viruses to SARS2 are coronaviruses found only in bats from southern China or across the southern border in Laos. This is 1500 km from Wuhan. The distance from Washington DC to the Florida Everglades.
 - a. Imagine you are having dinner at a restaurant in North Bethesda next to NIAID labs. You get sick and are told that the virus you caught is only found in bats from the Everglades, but it is also being studied at those laboratories you see out the restaurant window.
 - b. That's what the market origin people are asking you to believe.
8. One wildlife vendor from the market had animals from Yunan Province, where the bats that harbor SARS-like viruses reside. He marketed bamboo rats. However, neither that vendor nor the bamboo rats from the market tested positive for SARS2 by PCR.
9. **In SARS1, Guangdong in southern China had eleven spillovers in eleven different markets in nine different cities, creating three distinct lineages and three outbreaks differing by up to 30 letters of diversity, about one year of diversity.**
10. Environmental specimens near the toilets and the stairs to the lunchbreak room were positive for SARS2 and are most consistent with a human SARS2 infection.
 - a. Only 21 of the 176 environmental samples had more than 10 reads of SARS2 in samples with over 100,000,000 total reads. Careful scientists would never draw

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definitive conclusions from such tiny genetic reads, as the probability they arose as contamination from one of a number of the laboratory processing steps used to prepare these samples is high.

- b. None of the samples with double-digit numbers of SARS-CoV-2 reads have a substantial fraction of their host DNA from any non-human species.
- c. Only one of the fourteen samples with at least 20% of the DNA from raccoon dogs contains any SARS-CoV-2 reads, and that sample had only 1 read out of ~210,000,000 reads mapping to SARS-CoV-2. 13 of 14 of the specimens with raccoon dog DNA had no SARS2. With SARS1, 100% of the market animals were infected.
 - i. **I frankly think it is shameful for scientists to mislead journalists and the public, saying these specimens are evidence that racoon dogs were infected with SARS2, without giving these details as context.**
 - ii. **This is why trust in science is broken.**
- d. Instead, SARS-CoV-2 reads are most correlated with reads mapping to various fish, such as catfish and largemouth bass. These animals cannot be infected with SARS2.
- e. None of the six samples with at least 20 per cent of their DNA from bamboo rats contained any SARS-CoV-2 reads. This is consistent with the lack of SARS2 in either bamboo rat whole animal specimens and the lack of infection of the bamboo rat vendors.
- f. Overall, SARS2 was most associated with only one mammalian species, and that is human.

- 11. Human cases in the market for the first two weeks were >20 meters distant from the wildlife stalls. One interesting hypothesis suggests that the market vendors were infected in a lunch break room on the second floor of the market accessed by stairs near the wildlife stalls. The room had poor ventilation and a daily Mahjong game provided a perfect COVID incubator.

These eight evidence sets are not what would be found if the market was the origin of SARS2.

Now let's go through the timeline of events related to the WIV and look at them through the lens of a possible laboratory-acquired infection.

March 31, 2019: A shipment of deadly pathogens from Canada's National Microbiology Lab was sent to the Wuhan Institute of Virology. The shipment was routed from Winnipeg to Toronto and then to Beijing on a commercial Air Canada flight.

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The scientists who shipped the pathogens, Dr. Xiangguo Qiu, her husband Keding Cheng and her students from China were removed from Canada’s only level-4 lab over what was described as a possible "policy breach."

The list of shipped viruses included two vials each of 15 strains of virus: seven varieties of Ebola virus, the Hendra virus, and two strains of Nipah virus, Malaysia and Bangladesh. These are the top three most deadly human pathogens on the planet.

Shipped on a commercial airline full of unsuspecting passengers.

The Canadian government has not, as of this date, provided sufficient details about this remarkable incident.

July 4, 2019: The PRC’s Ministry of Science and Technology orders a review of several grants, including grant no. 2013FY113500. This is the grant which funded the collection of hundreds of coronaviruses and bat samples from the cave in Yunnan province.

July 16, 2019: The WIV publishes a tender requesting bids to conduct renovation on the hazardous waste treatment system at the Wuhan National Biosafety Lab (WNBL). The closing date was July 31st.

August 2019: Eddie Holmes said privately to the Proximal Origin authors: “I meet this guy who said his mate at the Wuhan Institute of Virology had human ‘SARS-like sample’ from August 2019.”

September 12, 2019: At 12:00am local time, the Wuhan University issues a statement announcing lab inspections.

Between 2:00am and 3:00am, the WIV’s viral sequence and sample database is taken offline.

At 7:09pm, the WIV publishes a tender requesting bids to provide security services at the WIV to include gatekeepers, guards, video surveillance, security patrols, and people to handle the “registration and reception of foreign personnel.” The budget provided was in excess of \$1.2 million US.

The databases taken offline contained 15,000 samples from bats (amongst the 22,000 records of samples in the DB) collected by the WIV. The DB contained over 1,400 strains of viruses across all species (animal, insect), including around 1,000 coronaviruses, with at least 500 recently discovered bat coronaviruses, and at least 50 of these close to SARS.

From January 2015 to March 2019 there were almost no downloads from the database. Beginning in April 2019 to September 2019, there were 603,793 pages downloaded, with 99% downloaded in June 2019. 99% of these page downloads were from Beijing computers.

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Three times Dr. Shi, in writing or orally, said the database was taken offline because of cyberattacks “during the COVID pandemic.” It has occurred to me that this might be Dr. Shi’s “Jeremiah Denton” moment. Denton was the Vietnam War prisoner who sent messages of help by blinking Morse Code with his eyes during TV interviews.

Maybe Dr. Shi is trying to communicate with the western world in an obtuse way, to prevent retribution from the government.

November 15, 2019: The WIV filed a patent for a device to treat accidental injuries sustained while working with pathogenic viruses in a biosafety lab. The device is a tourniquet for wrapping around the fingers of someone who starts bleeding in a virology lab accident.

It is the only patent of the 365 patents filed by the WIV relating to responding to an injury sustained while working with pathogenic viruses.

The inventors include a bat coronavirus researcher and colleague of those who have researched the suspected SARS outbreak at the Mojiang mineshaft, where the closest match to SARS-CoV-2 was identified by the WIV. Others include those responsible for biosafety when working with infected animals, biosafety laboratory management, and Communist Party discipline at the WIV. Both Tang Hao and Wu Jia have also filed both of the WIV's patents relating to breeding bats.

As Plato said, paraphrasing: “Necessity is the mother of invention.”

December, 2019: The installation of a People’s Liberation Army’s bioweapons expert as the head of the WIV’s Biosafety Level 4 lab (BSL-4).

December 30, 2019: Dr. Shi, head coronavirus researcher at the WIV and collaborator with Baric and Daszak, said in an interview that when she heard there was a coronavirus outbreak in Wuhan, her first thought was: “Could they have come from our lab?”

December 30, 2019: Dr. Shi makes universal changes to another database, Batvirus.whiov.ac.cn, scrubbing all references to sample collection from wildlife animals. Dr. Shi must have been very busy with sequencing patient samples, etc. that day. Why would she personally stop and make changes to a database?

End of December, 2019: The WIV laboratory where the first seven clinical specimens were sent was highly contaminated when the specimens arrived the end of December 2019.

How do I know that?

Because my colleagues and I examined the raw sequencing data for things that shouldn’t be in a clinical specimen and found a cornucopia of “things” that human lung specimens do not have.

These include:

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- The detection of the Nipah virus in an infectious clone format, a BSL4-level pathogen and CDC-designated Bioterrorism Agent, in raw RNA-Seq sequencing reads deposited by the Wuhan Institute of Virology (WIV).
- Research involving Nipah infectious clones has never been reported to have occurred at the WIV.
- The format of Hepatitis D virus ribozyme and T7 terminator downstream of the 5-prime end of the NiV sequence is consistent with truncation required at the end of the genome for a full-length infectious clone.
- This indicates that research at WIV was being conducted on an assembled NiV infectious clone.
- Contamination of patient sequencing reads by an infectious NiV clone of the highly pathogenic Bangladesh strain could indicate a significant breach of BSL-4 protocols.
- **This is the same strain of Nipah virus shipped from the Canadian BSL-4 lab on a commercial plane in March 2019.**

2020: Repeated actions by the Chinese Communist Party and scientists working at or affiliated with the WIV to hide or coverup the type of research being conducted at there.

Fourth, the evidence that is found in a natural zoonosis with respect to the animal host, the virus, and the human population in the vicinity of the outbreak is missing for the COVID pandemic. Each of these three components of a zoonosis will be examined separately and each will be found wanting.

The animal: In a natural zoonosis, the animals that hosts the virus are usually universally infected. In both SARS1 and MERS, >90% of the civet cats and camels, respectively, in markets with human infections were infected. Here it is different:

China conducted the largest wildlife sampling in the history of the world. Specifically, they tested:

457 specimens from 18 species of animals were collected at the Hunan Market, related warehouses, and other markets, and all were negative for SARS2 by PCR.

616 specimens from market suppliers were tested and were all negative for SARS2 by PCR.

1287 specimens from 27 species of animals from three southern provinces, Yunnan, Guangdong and Guangxi, were all negative for SARS2 by PCR. This included bats and pangolin, an on-again, off-again candidate for virus host.

11,708 stored animal blood specimens from 2019 and 2020 from domestic animals were tested for antibodies to SARS2 and were negative.

12,092 stored samples from domestic animals were all negative for SARS2 by PCR.

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26,807 stored samples from all over China were tested by PCR with a broad coronavirus probe. 1711 were positive for animal coronaviruses. None were positive for SARS2. The animal viruses were 54% identical to SARS2 and could not be the precursor of SARS2.

3643 samples from 74 species of captive wild animals from Hubei Province and 27,000 samples from 208 species across all of China were tested for SARS2 by PCR. None were positive.

2328 samples from 69 species of non-captive wild animals from Hubei Province was all negative for SARS2.

1000 bats from Hubei Province tested negative for SARS2 by PCR.

13,064 bats from 703 hot spots in China were sampled for sarbecoviruses. 146 new viruses were found but all were SARS1 related, none were SARS2 related.

The purpose of this recitation of sampling work in China is to make clear that the largest effort to find a virus host in the history of the world came up empty.

If someone says, well we don't know the definitive host of Ebola 40 years after it first appeared, please tell them that after 3.052 bat samples for infection or 3% of the SARS2 effort, the reservoir host has been narrowed down to one of nine species of African bats.

96,359 samples later, no animal has ever been found to be infected with SARS2.

The human: Seroconversion is the finding of evidence of an infection, that is antibodies, in stored blood samples from before the pandemic. It is a hallmark of a zoonosis. Before a new virus can spread human-to-human it has to practice jumping into people. They may not even know they were sick but their blood shows evidence of an infection.

In both the SARS-CoV-1 and MERS epidemics, around 0.6 percent of people in the region had antibodies in their blood before the outbreaks actually occurred. That number was much higher among supply chain workers, sometimes reaching up to 19.9 percent.

43,586 blood samples from blood donors taken between September and December 2019 were tested for SARS2. If the virus had been silently in the community like with SARS1 or MERS, you would have expected about 260 positive specimens. In fact, there were none. Because these were blood donors, an active infection would be screened out during the processing of the individual.

This is not consistent with a natural spillover.

The virus: The natural spillovers of SARS1 and MERS were multiple jumps from animals to humans and the natural genetic diversity of the viruses in their animal hosts would be noted in the early cases. In SARS1 there was an approximate 30 letter difference in the early cases, reflecting it had been present in the civet cat populations for well over six months. With MERS

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this ‘posterior diversity’ as it is called, indicated it had been circulating in camels for several years.

SARS2 was different. There was no posterior diversity.

Whether you look at the animals, the viruses, or the populations near the outbreak, SARS2 and COVID did not obey any of the rules.

Fifth, the genome of SARS-CoV-2 has seven features that would be expected to be found in a virus constructed in a laboratory and which are not found in viruses from nature. The statistical probability of finding each feature in nature can be determined and the combined probability that SARS2 came from nature is less than one in a billion. These eight features are:

1. **The backbone.** Coronaviruses from Hubei province, where Wuhan is, have two deletions the prevent ACE2 binding in humans. Dr. Shi from the WIV helped establish this observation. As Dr. Shi said: “I had never expected this kind of thing to happen in Wuhan, in central China.” Here, the backbone is only found in SARS-related viruses from southern China or northern Laos, both places that EcoHealth Alliance and the WIV had repeatedly taken samples in the years before the pandemic.
2. **The receptor binding domain**, a 200 amino acid section of the spike protein, was optimized for binding to the human ACE2 receptor. An analysis of over 3800 possible substitutions of amino acids in a 200 amino acid receptor binding region shows that SARS2 is 99.5% optimized for binding to the ACE-2 receptor. This near perfect binding has never been seen before in a recent interspecies transmission jump. SARS1 had only 15% of the optimized amino acids in the first cases in humans. It needed to change 85% of the amino acids to become an epidemic virus. The first SARS1 viruses bound better to civet cat ACE2 than to human ACE2. The best binding for SARS2 was to human ACE2. In fact, SARS2 is so adapted to humans it can no longer infect bat cells.
3. **The furin cleavage site** has never been seen before in SARS-related viruses from nature. But scientists, including at the WIV, have put furin cleavage sites in SARS1 and MERS viruses. Critics of a lab origin point out three things: it has an unusual amino acid, a proline, at its start; it is a non-standard furin cleavage site; and computer algorithms predict it is not an optimally efficient site.
 - a. First, the leading proline is in the MERS furin cleavage site. Baric and Shi took a bat MERS-like virus that could not infect human cells, HKU4, and added a furin cleavage site to see what would happen. It became human cell adapted.
 - b. Second, the non-standard furin site is found in an alpha coronavirus that infects and kills cats. Ralph Baric confirmed that DEFUSE proposed inserting novel furin cleavage sites into live viruses, inspired by feline coronaviruses.

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- c. The furin cleavage site of SARS2 is identical over 8 amino acids to the human sodium channel in the lung which is a classic human furin site. It is very efficiently cleaved.
- d. Privately Dr. Garry said: “...the question whether or not the market was the type of environment where you could have had the intense selective pressure required to generate an optimal furin cleavage site.” To which his collaborator, Dr. Eddie Holmes said: “No way the selection could happen in the market. Too low a density of mammals.”
- e. Dr. Garry also said the following about the origin of the furin cleavage site:

From Bob [Garry]:

Before I left the office for the ball, I aligned nCoV with the 96% bat CoV sequenced at WIV. Except for the RBD the S proteins are essentially identical at the amino acid level – well all but the perfect insertion of 12 nucleotides that adds the furin site. S2 is over its whole length essentially identical. I really can't think of a plausible natural scenario where you get from the bat virus or one very similar to it to nCoV where you insert exactly 4 amino acids 12 nucleotide that all have to be added at the exact same time to gain this function – that and you don't change any other amino acid in S2? I just can't figure out how this gets accomplished in nature. Do the alignment of the spikes at the amino acid level – its stunning. Of course, in the lab it would be easy to generate the perfect 12 base insert that you wanted. Another scenario is that the progenitor of nCoV was a bat virus with the perfect furin cleavage site generated over

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evolutionary times. In this scenario RaTG13 the WIV virus was generated by a perfect deletion of 12 nucleotides while essentially not changing any other S2 amino acid. Even more implausible IMO.

That is the big if.

You were doing gain of function research you would NOT use an existing close of SARS or MERSv. These viruses are already human pathogens. What you would do is close a bat virus th[at] had not yet emerged. Maybe then pass it in human cells for a while to lock in the RBS, then you reclone and put in the mutations you are interested – one of the first a polybasic cleavage site.

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4. **The furin cleavage site genetic sequence** contains the rare two codons for the arginine-arginine amino acid pair that are CGG-CGG. There are 64 codons in coronaviruses and the CGG is the 62nd least used codon. Based on what is found in nature you would expect this to be found in 1 out of 400 arginine dimers. There are 13 conserved such dimers in SARS-related viruses and this CGG-CGG dimer has never been found in nature. In the lab CGG is the most common synthetic arginine codon used.
5. **SARS2 has the number and pattern of synthetic genetic assembly sequences** that would allow the use of Ralph Baric's No See Em technology for assemble. The pattern seen in SARS2 has never been seen in nature.
 - a. Synthetic biology of coronaviruses is done with two restriction enzymes that cut the SARS2 RNA wherever there are two different and specific six letter sequences. Assuming these two sites are random, there should be 11.4 of these sites in a 30,000-letter coronavirus genetic code. A broadly diverse set of SARS-related coronaviruses from nature have on average 11.8 of these sites and no virus from nature has fewer than seven of these sites. SARS2 has five sites, which have a likelihood of having come from nature of one in 1100. The twelve sites in natural SARS-related viruses are conserved as to location in nature.
 - b. For SARS2, the seven sites that are extinguished are all done so as to not change the amino acid it codes for. The likelihood this happened by accident is one in 71,000.
 - c. Looking from left to right, the twelve sites in natural viruses are randomly ordered. But to do the No See Em technique in the lab, the sites need to be symmetrically distributed and segregated left to right. No natural virus has that pattern but SARS2 does. The five sites are A-A-A-B-B, where A and B are the two different enzymes. The likelihood this could happen in nature is one in 60.
 - d. When you combine the total number of only five sites, the method of silencing the seven, and the pattern of the five, the chance these occurred together in a natural virus is one in 1.2 billion.
 - e. I predicted how this virus would be constructed based on the five sites it has. Baric and his colleagues used exactly this predicted method when they made an infectious clone of SARS2.

In 2018, Baric and Daszak from the US, Shi from the WIV, and Linfa Wang from Singapore wrote a grant to DARPA called DEFUSE. In this grant they proposed to hunt for viruses from the caves in Yunnan that RaTG13, the closest virus to SARS2, came from. They were looking for viruses that were about 25% different from SARS1. They would adapt these viruses to infect and kill human cells by growing the new viruses in human cells in a petri dish and in transgenic animals with human lung ACE2. They proposed using the same restriction sites in the analysis I

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just conducted to move pieces of the genetic material around. They also proposed inserting a “human specific” furin cleavage site, like the one found in SARS2 at an exact spot in the gene called the S1/S2 junction.

SARS2 has the proposed backbone from the proposed region of China, has the proposed adaption for human killing, has the proposed 22% diversity from SARS1, has the proposed No See Em cleavage site profile, and the proposed human furin cleavage site at the proposed S1/S2 junction.

The grant from the WIV and US colleagues predicts seven features found in SARS2!

Interestingly, in the grant proposal they said potentially dangerous experiments of growing the synthetic viruses would be done in North Carolina in Baric’s lab. But a FOIA request generated a draft grant proposal that told a different story.

An early draft of DEFUSE acknowledged that the engineering and testing of novel coronaviruses would occur at BSL-2. The proposal advertised this approach to DARPA grant makers as “highly cost-effective.”

But “BSL-2” was edited to “BSL-3.”

Daszak also said in marginal comments to the draft that they would downplay the work being done in China but after the grant was approved they could move the work to the WIV for speed and efficiency.

In a comment on the this point in the document, Baric acknowledged that U.S. researchers would “freak out” if they knew the novel coronavirus engineering and testing work would be conducted in a BSL-2 lab.

6. **The ORF8 gene** in SARS2 is unique and has properties forbidden to be worked on with gain of function experiments. Those gain-of-function features relate to asymptomatic spread and immune system evasion. Since these two features would greatly increase the pandemic potential of a virus, governments and academic scientists have agreed not to conduct gain-of-function research in these areas.
 - a. Where does SARS2 come in?
 - b. SARS2 contains a protein called ORF8, so named because it is the eighth protein in the SARS2 genome. It is one of the only proteins that is not part of the finished virus or is involved in taking over the cellular machinery that makes new viruses.
 - c. ORF8 is diabolical. It is made early after an infection before other viral proteins begin to be synthesized. At this point the cell is largely unaware it is infected and hasn’t mounted any defenses. ORF8 enters the blood stream and interacts with the immune system, doing two things.
 - d. First, it blocks the production of interferon. Interferon has two important functions: First, it is a blunt weapon against infections that is used early by the

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body to slow down an infection, allowing time for antibodies to be produced and T-cells to respond. And second, it produces the familiar symptoms of an infection: fever, chills, sweating, red skin. The symptoms of an infection are not directly from the microbe itself but are from the body’s response to the presence of a microbe. Take interferon away and you have asymptomatic spread.

- e. I am aware of no other new respiratory virus that is asymptomatic when it first entered the human population.
- f. If you remember back to the early days of COVID, no one thought we could be missing infections because of lack of symptoms. We now know that 40 to 50% of COVID from the beginning was asymptomatic.
- g. The other property of ORF8 is that it interferes with the immune system’s process of making antibodies and teaching T-cells about the virus. This so-called MHC antigen presentation system is important for fighting infections. The AIDS virus is the poster child of viruses that become chronic infections because, among other things, it inhibits the normal immune system response.
- h. No one knew about ORF8 and these features when the vaccine target was being selected and so immunity from vaccination does not include inhibiting ORF8. Interestingly, in a natural infection your body recognizes ORF8 as a highly foreign protein and actually makes more antibodies against it than any other protein, including the spike protein.
- i. **What does this have to do with gain-of-function research at the WIV?**
- j. Prior to 2019, the WIV had conducted extensive research on optimizing the ORF8 gene and its function and on creating a synthetic biology cloning pathway for manipulating this protein and putting it in viruses in the laboratory. This work was found in two master theses from students at the WIV that were never translated from Mandarin nor did they ever lead to publications. This secret work on ORF8 is a classic “dual use” research project.

If China had been forthcoming we would have known from the beginning that SARS2 had these three genetic features, that is, an optimized receptor binding domain, the effects of the furin cleavage site on transmissibility and multi-organ affinity, and the properties of ORF8, it would have significantly helped in reducing the pandemics impact for three reasons:

- Human-to-human spread was accruing from the beginning and did not have to be acquired slowly, like with SARS1. The world lost almost a month of response time while public health officials made pronouncements about lack of human-to-human spread;
- Rapid spread within the body because of the humanized furin cleavage site, beginning in the lungs but leading often to multi-organ attack, could have guided treatment to better outcomes;

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- And finally, knowing that 40% of cases were asymptomatic and that vaccines might be improved by including immunizing against ORF8 could have been easily done and might have improved vaccine efficacy, as well as not missing early asymptomatic cases.

In closing, let's do a thought experiment. Let's imagine it is 2018 and you are asked: do you think a market spillover of a coronavirus could occur in Wuhan, China?

Dr. Daszak and Dr. Shi, scientists who have studied coronaviruses for almost two decades, certainly did not. How do I know? Because they used residents of Wuhan as the control group in a study looking for antibodies to coronaviruses in people living near bat caves in southern China. Sure enough, the rural residents near the caves had a 3% incidence of antibodies in their blood to coronaviruses and no one in Wuhan did.

Let's do a second thought experiment, again imagining it is 2018.

Do you think a laboratory-acquired infection could begin in Wuhan, a city with the world's most dedicated laboratory to collecting coronaviruses from nature, doing synthetic biology on coronaviruses, doing petri dish and animal research on coronaviruses, and that had written a proposal to make a coronavirus that had seven unique properties that ended up being found in the coronavirus that caused an outbreak in Wuhan?

I'll let you answer that question for yourself.

What reforms should be considered in order to assure that such research is conducted in a safe and transparent manner?

While I find no actual benefit of gain-of-function research, I believe efforts to ban it, given the vested interests of literally the entire virology community, and maybe others, is a hill too steep to climb. I do have a number of proposals I would like to offer for your consideration:

One, a proposal that I believe is achievable is the placement of all select agent research within an institutional review board structure similar to what is used for human clinical trials. I believe this effort would put guardrails around the most dangerous aspects of this research and has the added benefit of potential international acceptance, even including within China.

My second reform would be to separate the governmental oversight of this research from the funding agency. We have now documented the failure of the internal NIH system, the so-called P3CO, to provide adequate oversight. The model is atomic energy research, which is largely funded by the Department of Defense, but which is overseen by the Atomic Energy Commission.

My third suggestion is to place western biotechnology equipment under export controls and monitoring. There are ways to build into these systems a forensic and law enforcement capability that

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could, for example, with probable cause and a court-issued search warrant, allow the work of any laboratory in the world to be scrutinized remotely. I have written a detailed proposal for such a system that I would be willing to share with Congress.

My fourth recommendation is simple. Don't put dangerous infectious laboratories near subways, like Line 2, where every major city in the world is accessible within the incubation period of an infection. And consider a “quarantine dormitory” for select agent researchers to cool off after a set of experiments. This works when WHO doctors return from the latest hotspot.

My work on the origin began with my identification of the Line 2 ‘COVID Conduit’ as I called it, in mid-2020.

The earliest cluster of hospitalized patients with both the Lineage A and Lineage B virus was at the People's Liberation Army Hospital in Wuhan. This hospital is about 3 km from the WIV and on Line 2 of the Wuhan subway system. All early cases of COVID in Wuhan are found in hospitals adjacent to Line 2 and the probability of this being a chance occurrence is one in 68,000.

The Line 2 COVID Conduit, as I call it, includes the PLA Hospital, the WIV, the market, and the international airport. You can literally walk down into the subway system from the WIV in China and next go outside again until you exit into the world in London, Paris, Milan, Dubai, or New York City, all before having any symptoms. **Modelling by others suggested the pandemic could not have occurred without the spreading impact of Line 2.**

Finally, include what I call “gain of opportunity” research in new oversight efforts. Going into caves where humans are seldom found, taking a bat fecal sample containing thousands of viruses, bringing those viruses back to a laboratory, and culturing the specimens, where a virus that might be controlled in a diverse natural environment but is now able to grow unrestricted in pure culture provides an immense increase in opportunity for potential pandemic risk, even without genetic engineering.

This is the goal of the Global Virome Project, a Gates Foundation funded, EcoHealth Alliance associated effort. Their stated goal: collect the estimated 500,000 unknown viruses that are capable of infecting humans and bring them back to a laboratory near you.

What could go wrong?

What happens if we have these hearings, and nothing changes?

As I said earlier, in December 2019 we found synthetic biology experiments with the Nipah virus at the Wuhan Institute of Virology. They had created a cloning vector with a virus the US CDC defines as a “Bioterrorism Agent.” The Nipah virus is one of the deadliest viruses on the planet, with a >75% lethality. This is 60-times deadlier than SARS2. Why were they conducting synthetic biology research at the WIV in December 2019 on the Nipah virus?

Opening Statement of Steven Quay, MD, PhD

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I cannot speculate.

But a laboratory-acquired infection with a modified, for example, airborne Nipah virus would make the COVID19 pandemic look like a walk in the park.

The work of this committee is critical to protecting the American people as well as the people of all countries, from future pandemics, manmade and natural. If we now fail to act with the knowledge we have, history, if it can still be recorded, will judge us poorly.

Thank you for the opportunity to speak before this committee.