A Logistical and Technical Analysis of COVID-19

As humanity battles the distinct strain of coronavirus originating from Wuhan, COVID-19, considering whether it is the result of naturally emergent mutations against the possibility that it may be a bio-engineered strain - directly altered by genetic manipulation, subject to artificially-guided evolutionary selection, or both – that was accidently released into the public has never been more urgent. There is a distinct difference between confronting a wild animal, and being attacked by one that has been genetically altered in a laboratory – those scenarios require profoundly different approaches and precautions. This difference is witnessed by the fact that segments of the population all across the globe have yet to take this outbreak seriously, and are still treating social-distancing like an optional exercise, or something to be done only by the elderly or infirm. The reality is that an accidental release of COVID-19 is a distinct possibility since China's rate of occupational accidents is about ten-times higher than America's, and some twenty-times more than Europe's,[1] the only other regions with high-level virology labs. Bearing this out, one lab in Beijing has leaked SARS-CoV four separate times.[2]

China's only ABSL-4 virology lab, the Wuhan Institute of Virology Disease Engineering Technical Research Center, was already actively looking into the risks posed from bat coronaviruses,[3] and actively researching coronavirus treatments - by definition both of these projects would require live virulent strains of coronavirus. Additionally, Wuhan's Center for Disease Control and Prevention was also proximate to the epicenter of the outbreak, and also housed experimental research animals.[4] Raising the odds that this Wuhan Strain of coronavirus was accidently released, researchers from the Wuhan virology lab were reported to have particularly sloppy field research methods, being both bled and peed on by local bats that host coronaviruses remarkably similar to COVID-19. [5] And they've also been reported to smuggle used research animals out of their labs, selling them for cash on the street.[6] Perhaps unsurprisingly, in mid-February 2020, the Chinese Ministry of Science sent out a directive to all its labs emphasizing the importance of carefully handling bio-infectious agents, alluding to slack oversight and past lapses, and even referring to coronaviruses explicitly.[7]

Mistakes may have been precipitated by the need to finish research that was being rushed for John Hopkins' Event 201 which was held this past October and meant to game-plan the containment of a global pandemic. Research may also have been hurried due to deadlines before the impending Chinese New Year - the timing of these events points to increased human error, not sinister machinations. Especially since a lab in Beijing had four known accidental leaks of the SARS virus in recent years,[8] there is absolutely no reason to assume that this strain of coronavirus from Wuhan didn't accidentally leak out as well. This is unlikely to be a plot twist in one of the novels Tom Clancy wrote after he started mailing it in.

Simply and horribly, this is likely to become another Chernobyl or Fukushima - a catastrophic illustration of mankind's hubris and intransigence clashing with Nature, as fate again reaps a once unimaginably tragic toll.

And exploring the possibility that COVID-19 may have been altered by man is incredibly important because official plans to handle a pandemic outbreak are all designed around metrics derived from natural viruses, and so if COVID-19 in fact has an unnatural origin – plans will need to be upgraded and revised for the unknown. And yet many notable scientists and public officials have gone on the record against the idea that COVID-19 could have been altered in a lab, and downplayed its expected virulence, protesting far too much when a natural origin has

not been conclusively demonstrated. However instead of presenting as a relatively benign cold-like strain of virus, COVID-19 appears to be at least *thirty-four times* more lethal than the seasonal flu.[9] So it is at best wildly irresponsible to speak-out against the possibility that this virus escaped from a lab when a natural origin has not been proven. These protests may be meant largely as a smokescreen for the wanton hubris and greed that transcends national identity and has fueled the gain-of-function research detailed below: As one possibility, coronaviruses have been seen a viable vector for an HIV vaccine for over a decade[10] - a project with hundreds of millions of dollars dangling over it. And so blame shouldn't fall only on the nations and lab personnel involved, but also on those funding dual-use gain-of-function research, and now seeking to suppress its existence – an effort that's been bolstered by the fact most American media outlets are blindly publishing their stories without even a cursory fact-check. And it should be noted that in 2018, the esteemed scientific journal *Nature* – which has published numerous articles speciously claiming this virus is definitely natural - was revealed to have buckled to censorship demands from the Chinese government, killing over 1,000 articles to placate their Chinese partners.[11]

And so, given that this outbreak was said to begin in early winter when most bat species in the region are hibernating[12] and the Chinese horseshoe bat's habitat covers an enormous swath of the region containing scores of cities and hundreds of millions people to begin with, the fact that the Wuhan Strain of coronavirus, denoted as COVID-19 emerged in close proximity to the only ABSL-4 virology lab in China, which in turn was staffed with at least two Chinese research scientists - Zhengli Shi and Xing-Yi Ge - two virologists who had previously worked at an American lab which already bio-engineered an incredibly virulent strain of bat coronavirus: The accidental release of a bio-engineered virus meant for defensive immunotherapy research from Wuhan's virology lab cannot be automatically discounted, especially when the Wuhan Strain's unnatural genomic signals are considered.

Manipulating viral genomes for decades.

Back in 2002, Stony Brook first assembled a DNA virus from scratch, building a polio-virus, and providing proof-of-concept for the creation, alteration, and manipulation of entire viral genomes. Two years prior, a separate team had already built a simpler RNA virus from scratch – choosing to create a novel coronavirus, and even swapping out its vital spike-protein genes and confirming they controlled which cell types were infected.[13] And the godfather of this research had emerged a generation earlier, as artificially enhancing selection by intentionally infecting countless series of lab animals with different viruses is understood to have created the H1N1 Swine Flu. Its Franken-genome has a mysterious untraceable genetic parentage and a "clear unnatural origin," appearing to be the product of "sequential passage in an animal reservoir"[14] which was determined by its vast genetic distance from any extant strain of flu, just like COVID-19 which also appears so distant from any related coronavirus that it's been placed in its own clade, an isolated branch way out on its own in the viral family tree - meaning it's the lone example of its kind, and doesn't clump together with all the other known coronaviruses. Although both the H1N1 Swine Flu and COVID-19 sprung into existence spontaneously, and were distant off-shoots of any other known strain of flu - so why is there a scientific consensus that the former leaked out of a lab, while many insist the latter is entirely natural?

Earlier in the 70's prior to the leak, "the swine flu scare… [had] prompted the international community to reexamine their stocks of the latest previously circulating H1N1 strains to attempt

to develop a vaccine," which was seen to have increased the odds that someone, somewhere would make a mistake and leak an altered strain of the flu out of their lab.[15] This increased pace of research mirrors recent times, when scientists have been investigating and trying to understand the supposedly impending threat posed by coronaviruses for years, capturing as many unique strains from the wild as they could, and mixing and matching their genomes in the lab.

And so increased research into the H1N1 Swine Flu back in the 70's eventually increased the odds that a mistake would happen enough that one did, and a leak occurred. Just as our current pandemic was preceded not only by years of research into coronaviruses everywhere from UNC to the Wuhan Institute of Virology's Disease Engineering Technical Research Center, and most recently by a massive international conference meant to study a pandemic caused by a coronavirus, Johns Hopkins' Event 201. Which was meant to model a global pandemic caused by a hyper-virulent strain of coronavirus, and was funded primarily by the World Economic Forum as well as the Bill and Melinda Gates Foundation, and notably occurred in October 2019, just weeks before the start of this outbreak of COVID-19.

If leading up to 1977, the fact that increased research into strains of the flu were seen to increase the odds that an accidental leak would occur, why isn't the same logic being applied to our pandemic today? Why is almost everyone today assuming that the increased pace of research means scientists in fact anticipated this outbreak instead of causing it? Wouldn't an increased pace of research also increase the odds that a leak of a lab-modified coronavirus would occur just like an increased pace of research precipitated the leak of the H1N1 Swine Flu back then?

And so this strain of the H1N1 Swine Flu became the poster-child for a moratorium against *gain-of-function* research - experimentation that seeks to increase a pathogen's virulence, creating a more effective double-edged sword to counter and learn from. In the case of H1N1, it wasn't a question of if it'd escaped from a research laboratory, only whether it'd been designed as part of a weapons system, or been part of a vaccine trial.[16]

Understanding zoonotic jumps.

Nothing about COVID-19's emergence is even the slightest bit natural. When a virus manages to infect a new species of host it's known as a *zoonotic jump*, a process that generally takes months or even years to complete. The first stage is when a virus infects one individual in a new host species, which is typically a dead-end the first time it happens since there's no way for the virus to be adapted to a different species' biology. The second stage of a zoonotic jump is when the virus manages to move from the first new host into more hosts of the new species, which results in some temporary transmission in a localized area – these are known as endemics and generally fizzle out the first few times they happen as the virus adapts to its new host species, and mutations win or lose the survival battle. The final stage, the only time a zoonotic jump is considered complete, is when there's sustained host-to-host transmission in the new species. These zoonotic jumps have some predictable characteristics, the primary one is that adapting to a new host inevitably requires mutations that weren't optimal in the old host. And so the virus gets weakened as its initially attempting to jump into a new host species, which is why the above sequence of steps – one new host, a few new hosts that pass it among themselves temporarily,

and then finally sustained transmission – takes at least several months if not years to play out, since a good bit of time is required for all three steps to occur. Viral trial-and-error is required for the virus to find the right mutations that will allow it to prosper in a new host species, it's never been known to just happen magically all at once.[17]

Assuming that COVID-19 suddenly emerged naturally in a matter of weeks in the middle of a massive urban metropolis the size of New York City, when the host population of bats was hibernating anyways and an additional species vector is needed but hasn't been found anyways, requires completely ignoring everything we know about how viruses transfer between species. Not only was Wuhan's population not interacting with bats since they hardly interact with humans in urban situations to begin with, but any possible host bats were sleeping in their caves anyways – unable to interact with humans or any other species. Along these lines, it should be noted that statistical modeling indicates that this virus did not exist in humans for much time before November 2019.[18] Plus beyond that, a study published in 2018 found that only *two-point-seven* percent of villagers living about a kilometer from local bat-caves carried any evidence of past bat coronavirus infections. That study happened to examine people living in Wuhan as well, and found absolutely zero evidence of previous bat coronavirus infection at all there, making it all-but-impossible that zoonotic jumping occurred since earlier less-lethal variants of the virus would have left a wide signature in its new host population.[19] Additionally, there's the fact that the version of COVID-19 found in the first few dozen hosts was exactly the same - there aren't any variants whatsoever, just one version. This is not what would be found after the genomic trial-and-error required for a full zoonotic jump, which requires sustained human-to-human transmission as different variants of the virus try and fail to adapt to human biology. Here, only one variant was found in all the initial infected humans, instead of the multiple variants that would be expected. But finding just one variant in all the initial hosts does fit what would happen if a virus that already had high affinity to the ACE2 receptor, which is the same in human and ferrets, leaked out of a lab.

And so not only would the circumstances of this transfer require rewriting the textbooks on zoonotic jumps if it occurred it all, but beyond that: supposedly not only did a zoonotic jump happen almost instantly without the necessary steps, but when it hit humans it was extraordinarily virulent from the start, something that's supposed to take an extensive amount of time to ever happen as mutations go through selective trial-and-error. This trial-and-error takes time and is why viruses have never made a zoonotic jump and been instantly virulent in the new host species like COVID-19 has been in humans. Nothing we know about how viruses naturally make zoonotic jumps points to that happening here, a virus hitting a population and spreading as quickly and lethally and prolifically as COVID-19 has points to it already being tailored to the human genome.

Gain-of-function fundamentals: Serial passage through an animal host.

Tinkering with viral genomes is not anything new, however is not something that has ever been fully embraced by the scientific community at large. About a decade ago, two separate research teams successful tweaked the genome of the H5N1 Bird Flu in just two spots and then passed it through ferrets until it became both airborne and pathogenic to mammals, creating a virus that "could make the deadly 1918 pandemic look like a pesky cold."[20] This involved selecting for a mutation that allowed the virus to access a receptor that's found in ferret lungs, and was

alarming enough that the research was urged to be published without revealing the specific methods involved and data collected – however it appears that only the most technical details were left out, and most of the research is freely available.[21] And studies examining SARS-CoV-2's infectivity in ferrets found that it spreads readily among them, and also appears airborne in that animal model,[22] lending support to the idea that ferrets were used for serial passage. It should also be noted that several years prior to tinkering directly with bat coronavirus spike-proteins, research was done that involved isolating a coronavirus from civets and then passing it through mammalian ACE2 receptor cells that were grown in the lab from kidney and brain samples – serial passage through host cell lines instead of entire hosts, which imparted a strong affinity for ACE2, and presumably created an airborne strain of coronavirus. And if cells derived from kidneys and brains were used for the serial passage development of COVID-19, that might help explain its affinity for attacking the kidneys and brains of its human hosts.

But whether or not the current COVID-19 outbreak is determined to have originated in a lab or not, the possible specter of a global pandemic caused by pathogens whose virulence has been accentuated in a lab should remind us that this dual-use gain-of-function research was banned for reasons that have not changed, and in fact have only grown stronger – as the technology that allows us to tinker with viral genomes has advanced so have the risks. This is research that has been ongoing for several years.

Back in 2015, conducting research that was met with an enormous amount of concern, scientists at UNC had successfully created a "chimeric, SARS-like virus"[23] by altering the viral genome of a Chinese bat coronavirus's *spike-protein genes* - sequences that code for the spikes that poke out from surface of viruses and allow them to unlock entry into host cells, in this case making the bio-engineered coronavirus incredibly contagious. This experimentation raised eyebrows since it was clearly gain-of-function research, a practice banned in America from 2014 until December 2017 when NIH lifted the ban, specifically to allow research on coronaviruses. Looking at UNC's gain-of-function research on coronavirus spike-proteins, which received its funding just before the ban was implemented and was only allowed to go forward following a special review, a virologist with the Louis Pasteur Institute of Paris warned: *"If the [new] virus escaped, nobody could predict the trajectory."*

Scientists have expressed concern about China's ability to safely monitor this ABSL-4 lab in Wuhan since it opened in 2017: "*an open culture is important to keeping ABSL-4 labs safe, and he questions how easy this will be in China, where society emphasizes hierarchy. 'Diversity of viewpoint, flat structures where everyone feels free to speak up and openness of information are important.'*"[24] This lab is at most 20 miles from the wild-meat wet market where the virus had been originally assumed to have jumped from animal to human, an assumption that's since been largely debunked: The first three known cases from early December had no contact with that market,[25] and roughly one-third of the initial exposed cohort had no direct ties to Wuhan's wet-market,[26] the original presumptive source of the virus. And in mid-February, reporting indicated that COVID-19's patient zero in fact had no connection at all with the wet-market.[27] This is reinforced by the fact Chinese research has further concluded that COVID-19 "may have begun human-to-human transmission in late November from a place other than the Huanan seafood market in Wuhan."[28]

The idea that a Chinese lab could have a viral sample escape is well-documented - as mentioned, one lab in Beijing has had four separate incidents of the SARS virus leaking out accidentally. And since its discovery, scientists have been unable to fully determine the zoological origins of COVID-19 - it was initially thought to have passed through snakes, but now all that's agreed upon is that it's mostly bat in origin. This inability to derive an exact zoological source is exactly

what would be expected if the virus had been artificially guided, and although it doesn't prove an artificial nature, it is consistent with one. And although there has been speculation that pangolins may have been the missing vector, the only data about the pangolin virome wasn't entered into NCBI's system until late January,[29] and couldn't possibly have been collected any earlier than late September 2019,[30] and doesn't fully answer the vector question anyways since "no pangolin [coronavirus] has yet been identified that is sufficiently similar to [COVID-19] across its entire genome to support direct human infection."[31] And further research examining the regions of the genome that best show genetic heritage indicated it was "very unlikely" that similarities between their spike-protein genomes of COVID-19 and pangolins, where they share the most similarities, was due to the virus passing through pangolins at all.[32] And notably this specific type of spike-protein cleavage site "has also been observed after repeated passage in cell culture or through animals,"[33] specifically by serial passage through an animal host in the lab, which would explain everything else about COVID-19's unusual genome as well.

As explained in *Nature,* COVID-19's Franken-genome combines a cornucopia of distinct genetic markers from each of the three other distinct branches of the coronavirus family tree, but is distinct enough from all of them that it in fact forms its own clade.[34] Along those same lines, a full-genome evolutionary analysis of COVID-19 published in *The Lancet* concluded that "recombination is probably not the reason for emergence of this virus"[35] since it seems that the Wuhan Strain isn't a mosaic of previously known coronaviruses, but instead draws from distant, discrete parts of the coronavirus family tree - not how these viruses naturally evolve.[36] Because even mixing and matching coronavirus genomes from every known zoological virus, scientists have been unable to find any possible combination that would explain those distinct regions of the Wuhan Strain's genome. This paper muses that a mysterious animal host could still be out there, however since they've already searched through every known possibility and been unable to find a match, another reasonable explanation is that serial-passage bio-engineering accounts for the inexplicable nucleotide signature of the Wuhan Strain's genome

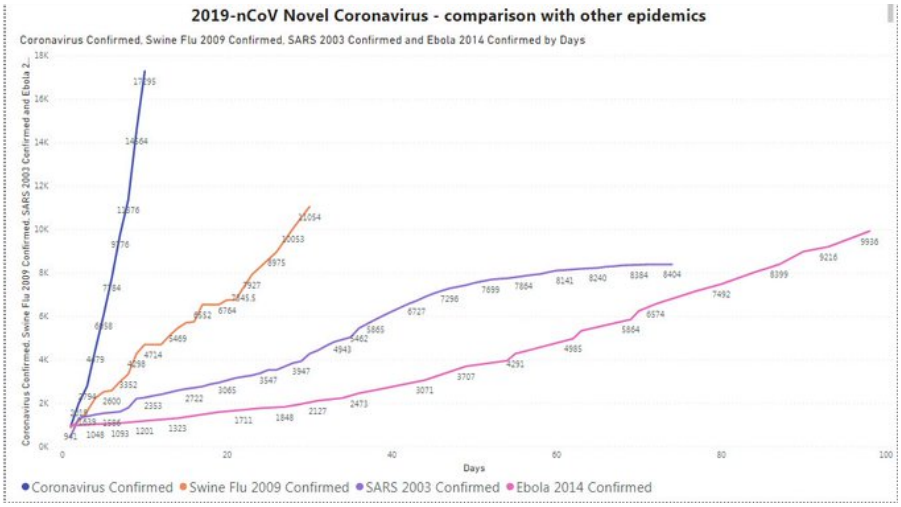
Early research found that COVID-19 targets the ACE2 receptor, which seems to be distributed in roughly equal proportions across global populations, indicating that the Wuhan Strain was likely developed as part of a defensive gain-of-function project possibly linked to immunotherapy or vaccinations - never meant to leave the lab, but meant to serve as a Red Team to fight back against, not as an offensive weapon targeting one specific global population. But counterintuitively, researchers have pointed out that the most critical sections of the COVID-19's protein-spike genome don't match the previously reported pattern that would be expected for optimal binding to the specific ACE2 receptors found only in humans and ferrets[37], which indicates that these particular segments wouldn't have been directly genetically engineered to increase virulence. However, this is exactly what researchers looking to design something for a "safe" vaccine candidate to target *would* engineer, and doesn't rule out a scenario where the virus was passed through a series of ferrets so that accelerated natural selection could occur. The research team in fact notes that its spike "appears to be the result of selection on human or human-like ACE2 permitting another optimal binding solution to arise,"[38] failing to directly mention that the only other human-like receptors are found in ferrets - which have frequently been used for years in vaccine trials for viruses with this sort of protein-spike,[39] and is exactly how the H5N1 Bird Flu virus was altered to make it airborne. And so the Wuhan Strain's unique affinity for the human ACE2 receptor, which a pre-print reports to be 10 to 20 *times* greater than SARS,[40] may be the exact type of vaccine-related accident that led to the moratorium on gain-of-function research in the first place and caused scientists to unsuccessfully call for the research around H5N1 to be protected.

A notable clinical presentation and notable personalities.

Nothing about COVID-19'S clinical presentation is typical, including the fact that one of the first signs of infection seems to be losing your senses of smell and taste without any other symptoms, something no other virus on earth is known to do to otherwise asymptomatic patients. Additionally, an unnaturally juiced-up ability to use ADE would also explain what our front-line medical workers are observing in their patients: *"I'm seeing people who look relatively healthy with a minimal health history, and they are completely wiped out, like they've been hit by a truck. This is knocking out what should be perfectly fit, healthy people. Patients will be on minimal support, on a little bit of oxygen, and then all of a sudden, they go into complete respiratory arrest, shut down and can't breathe at all… That seems to be what happens to a lot of these patients: They suddenly become unresponsive or go into respiratory failure."*[41]

This sort of sudden precipitous decline is exactly what would be expected if COVID-19's ability to use ADE had been accentuated in the lab, and would also explain the clinical observations that *"this severity of [acute respiratory distress] is usually more typical of someone who has a near drowning experience — they have a bunch of dirty water in their lungs — or people who inhale caustic gas. Especially for it to have such an acute onset like that. I've never seen a microorganism or an infectious process cause such acute damage to the lungs so rapidly. That was what really shocked me."* And also the following horrific account: *"Holy shit, this is not the flu. Watching this relatively young guy, gasping for air, pink frothy secretions coming out of his tube and out of his mouth. The ventilator should have been doing the work of breathing but he was still gasping for air, moving his mouth, moving his body, struggling. We had to restrain him. With all the coronavirus patients, we've had to restrain them. They really hyperventilate, really struggle to breathe. When you're in that mindstate of struggling to breathe and delirious with fever, you don't know when someone is trying to help you, so you'll try to rip the breathing tube out because you feel it is choking you, but you are drowning."*[42]

In another unusual turn, the Wuhan Strain of coronavirus, COVID-19, appears to be transmissible even before its host shows any symptoms at all, making temperature-scanning at airports ineffective since hosts appear to be contagious for about a week before any symptoms emerge. This is in stark contrast with SARS, whose hosts weren't contagious until they were symptomatic, allowing for its relatively quick containment. This chart captures the drastically different comparative rates of infections between recent outbreaks:



Unsurprisingly, A recent pre-print now gives COVID-19 an infectivity rating between R4.7 and R6.7 – meaning each host will infect about five new ones.[43]

Engineering a coronavirus' protein-spike has already been done before: The successful end results of the aforementioned bat coronavirus bio-engineering research at UNC that was critiqued for being too risky in 2015, which was published the following year and described the successful bio-engineering of a highly-virulent coronavirus derived from bats was achieved by tinkering with its spike-protein genes. [44] In this paper, researcher #8 is listed as one "Zheng-li Shi" who's listed as being attached to the *"Key Laboratory of Special Pathogens and Biosafety, Wuhan Institute of Virology, Chinese Academy of Sciences,* ***Wuhan, China****."*

Zhengli Shi seems to have returned to Wuhan at some point since 2016, specifically to the Wuhan Institute of Virology's Disease Engineering Technical Research Center, since she then appears in an eerily salient September 2019 paper on the human behaviors most likely to lead to bat-borne coronavirus exposure in southern China,[45] which could either be seen as enormously prescient or as a very intentional attempt at publishing plausible deniability. Zhengli also appears in the paper claiming that this coronavirus was bat in origin,[46] which was rather peculiarly submitted for publication in direct coordination with the official Chinese announcement of the outbreak.[47] This is just a small sample of the dozens of coronavirus-related papers she's published over a three decade career.

And not only does Zhengli Shi provide a direct chain of expertise tying the already successful bio-engineering of a virulent bat-based coronavirus at UNC directly to the ABSL-4 virology lab in Wuhan, but back in January 2014 she'd received a $665,000 grant from NIH for a study titled *The Ecology of Bat Coronaviruses and the Risk of Future Coronavirus Emergence (NIAID R01 AI1 10964)* as well as $559,500 more from USAID for a study titled *Emerging Pandemic Threats PREDICT_2China* (Project No. AID-OAA-A-14-00102). Beyond this American funding specifically into viral diseases zoonotically transferring from animals to humans which slipped in just before the ban, over the years she's also received around $3 million in grants to study these zoonotic viruses from China and other countries, and has served on the editorial board of several virological research magazines. More of her research into the intersection of coronaviruses like the Wuhan Strain and their epidemic potential was funded by the U.S. Department of Defense, the U.S. Threat Reduction Agency, and U.S. Biological Defense Research Directorate of the Naval Medical Research Center.

And so a scientist who's been prolifically involved with studying the molecular interaction of coronaviruses and humanity, spending decades and millions of dollars, and having even helped build a hyper-virulent coronavirus from scratch at UNC - just so happens to be working at the only ABSL-4 virology lab in China that also just so happens to be at the epicenter of an outbreak involved a coronavirus that's escaping zoological classification, and has other unnatural characteristics that will be discussed below.

Additionally, another Chinese virologist, Xing-Yi Ge, appears as an author on the 2016 UNC paper in question and is also attached to the lab in Wuhan. Previously in 2013, he'd been the very first scientist to successfully isolated a SARS-like coronavirus from bats which targets the ACE2 receptor,[48] just like our present virus, the Wuhan Coronavirus COVID-19 uses and which may be amplified through passage through series of ill-fated ferrets. And it turns out that sections of the Wuhan Strain's ACE2 receptor's binding genes are unique: they're almost identical to SARS's spike-protein genes - despite the fact that almost none of the two coronavirus's genomes are similar anywhere else at all.[49] Beyond that, although the Wuhan Strain's spike-protein genome differs from SARS in four out of the five most important genomic

spots that determine binding to the ACE2 receptor,[50] they surprisingly don't affect the protein-spike's eventual shape. And in an even bigger coincidence, these four spots also code for the outside region of the spike that allows entry into cells, and do not affect it either - allowing the Wuhan Strain to still use the ACE2 receptor to unlock cells while possibly gaining additional capabilities. The odds that this concordance was bio-engineered into the virus are several orders of magnitude more likely than for this to randomly have evolved in nature.

The specter of antibody-dependent enhancement.

And some of the dystopian carnage that crept and may still be seeping across China may be due to the fact that much of China's population may have already been exposed to coronavirus infection via SARS or other less notorious strains, which would allow COVID-19 to use antibody-dependent enhancement to much more efficiently enter into cells,[51] and then become much more virulent since this enhancement hijacks the body's preexisting immune response to coronavirus infections and allows easier entry, and allows it to enter its hosts' nervous systems. However whether or not people have been exposed to a coronavirus infection before, once it's been circulating in a population for long enough the Wuhan Strain may be able to re-infect its own past hosts and use this molecular hijacking on antibodies left from its own previous infection to become far more virulent, regardless of whether or not someone has been exposed to other coronaviruses before COVID-19. And early reporting from Chinese doctors in fact indicates that re-infections of the Wuhan Strain are far more lethal than the first.[52] More evidence that ADE is occurring is its drastically higher affinity for the ACE2 receptor viral loads that are up to 1,000 higher than what was seen in SARS patients – both of these may be due at least in part to ADE allowing COVID-19 to much more efficiently bind to and enter cells. And another hint that the Wuhan Strain may be using ADE to more effectively attack its host is the fact that it seems to be targeting its host's neurological systems, which is explored in depth below. A final clue is the fact that children seem to be far less effected by the Wuhan Strain COVID-19, a phenomenon that's found in Dengue Fever, which is one of the classic examples of a virus that uses ADE. And curiously Zhengli Shi, of UNC and Wuhan fame, co-authored a 2019 paper which used inert viral shells to figure out exactly how SARS, with its affinity to the ACE2 receptor just like COVID-19, was able to harness ADE to hijack white blood cells for enhanced cell entry. A gain-of-function extension of this research would be exactly the kind of experiment that could've given birth to COVID-19, especially considering that 2019 paper managed to fine-tune exactly the concentration of antibodies that would best allow ADE to occur.

Much of COVID-19's unique genome is difficult to interpret, since although a retracted pre-print noted several very short genomic sequences in COVID-19's spike-protein gene that look far more similar to sequences found in HIV than to other coronaviruses - critics quickly pointed out that the shared homology didn't reach statistical significance. However a closer look at the data reveals that there were a few small shared genomic segments that, despite being physically separated from each other along each strand of DNA, all worked together to code for the Wuhan Strain's protein-spike's crucial receptor binding site. Something that is highly unlikely to have happened by chance. And despite most of its protein-spike being shared with SARS, these substituted segments weren't shared at all - nor were they found in any other coronavirus. One possible reason for these HIV-like segments is that they were meant to be *epitopes*, or molecular flags meant to mark intruders for a vaccine to target – indicating COVID-19 may have been designed to have built-in red-flags for a presumptive vaccine to target. It is mathematically possible for this to happen in nature - but only in a *ten-thousand bats chained to ten-thousand Petri dishes and given until infinity* sense. Alternatively to this mathematical unlikelihood,

these patterns could also be produced by infecting a room full of ferrets with a bespoke coronavirus vaccine and sifting through the wreckage for your genomic needle.

Despite the fact critics have brushed off the Wuhan Strain's shared homology with HIV as statistically insignificant, clinical reporting indicates that the Wuhan Strain may be using this shared HIV homology to attack immune cells just like HIV does, as an unusually high percentage of patients are showing low white blood cell counts,[53] especially in the most serious cases. This pathogenicity may well be due to the unique HIV-like genomics of the Wuhan Strain, as one white-paper by LSU's professor emeritus of Microbiology, Immunology, and Parasitology who's also a Harvard-educated virologist with a PhD in Microbiology and Molecular Genetics notes: "This is the first description of a possible immunosuppressive domain in coronaviruses... The three key [mutations] common to the known immunosuppressive domains are also in common with the sequence from [the spike-protein]. While coronaviruses are not known for general immunosuppression of the style shown by HIV-1, this does not rule out immunosuppression at the site of active infection in the lung, which would prolong and potentially worsen infection at that site."[54] And early research has indicated that this unique region may make COVID-19 up to 1,000 times more likely to bind to human cells than SARS, which could also indicate that ADE is occurring.[55]

Even more troubling, a published paper noted that one particular part of the Wuhan Strain's spike-protein genome also wasn't found in any of its relatives, "and may provide a gain-of-function to [COVID-19] for efficient spreading in the human population."[56] And according to that paper, this particular type of cleavage site makes similar viruses both more pathogenic and more neurotoxic. Additionally, this particular type of cleavage site is a hallmark of being passed through a series of animal hosts in a lab.[57] Evidence for the Wuhan Strain's neurotoxicity arrived in late February, in a published paper which notes that "the most characteristic symptom of COVID□19 patients is respiratory distress, and most of the patients admitted to the intensive care could not breathe spontaneously." Combined with the observation that "some COVID□19 patients also showed neurologic signs such as headache, nausea and vomiting," this paper asserts that since SARS was found heavily concentrated in the brainstems of its autopsied victims, COVID-19 is also probably crossing the blood-brain barrier and killing its victims not just via pneumonia, but also by causing neurological respiratory failure[58] - indicating that ADE may be occurring, but at a much faster rate than nature allows since reinfections of Dengue Fever that use ADE typically have years pass between infections.

Additionally, it should be noted that SARS - much ballyhooed as a close relative to the Wuhan Strain - didn't notable effect white blood cell counts.[59] Additionally, clinical treatment guides published online in late January by established Chinese medical sources[60] note the progressive reduction of white blood cells, as well as the importance of monitoring this decline. And reporting from Thailand indicates that adding a cocktail of two different anti-HIV drugs to the typical flu treatment regime seemed to effectively knock back the Wuhan Strain's clinical presentation. Additionally, one of the only autopsies performed outside of China to date found that the deceased had a severely depleted white blood cell count.[61] These lowered counts may come from this shared similarity with HIV, or it could also be the result of antibody-dependent enhancement as well, since this phenomenon primarily targets white blood cells for its hijackings and may help explain why consecutive infections are so lethal.

And in a highly concerning turn, scientists have noted that the Wuhan Strain can have a "striking" short term rate of mutation[62] which doesn't indicate an artificial origin but captures the unique threat posed by this coronavirus regardless of its providence, since a faster mutation rates makes it more likely this virus can dodge testing and neutralize vaccines. Something there

is already early evidence for, as several COVID-19 patients have been re-infected after being discharged from the hospital, and many others test negative multiple times before popping a positive test. And although the full implications of this are not yet known - none of them are good, since re-infections may be far more severe due to antibody-dependent enhancement.

One of the worst possible scenarios for COVID-19's mutation rate would be if it falls into the Goldilocks range that would allow it to form mutant viral swarms: too many mutations will cause a virus to eventually implode, not enough allows host immune systems to catch-up, but if things are just right mutant swarms can form and spread across host populations, burrowing into host nervous systems and causing permanent neurological damage.[63] Mutant swarms form when a virus produces mutationally-damaged copies of itself inside a host, some of which aren't infectious but find their way into the nervous system where they burrow in causing damage, and others that combine with complimentary broken copies inside host cells to produce working infectious copies of the virus. So a host can not only become crippled with neurological issues, but also still be producing infectious copies of the virus. And it seems as if COVID-19 has many characteristics that indicate the potential to form mutant swarms: the "striking" mutation rate mentioned above and the fact a second widespread mutated strain seems to have already emerged in Washington State with many other isolated strains reported elsewhere, crossing between species is another factor and a dog in Hong Kong appears to have tested positive, the fact that the Wuhan Strain can infect not only the respiratory tract but feces as well – multi-organ involvement is an important contributor to viral swarms,[64] and finally the markedly viral load rate of COVID-19 compared to SARS – SARS produced a viral load orders of magnitude lower which decreased over time, while COVID-19 produces a "very high" viral load[65] that appears to increase over time and can peak several orders of magnitude higher than SARS was measured to reach.[66] And alarming evidence that this phenomenon is occurring emerged from a Chinese pre-print which noted that over one-third of the roughly 200 patients studied has some neurological symptoms, with nearly half of the most severe patients exhibiting neurological degradation.[67] And further evidence for the possibility of both mutant swarms and ADE is witnessed by a study published in *Lancet,* which notes that the case fatality rate in Wuhan could actually be as high as 20%[68] - the outbreak's epicenter would be expected to have the highest rates of both phenomena as different variants of the Wuhan Strain infected and reinfected overlapping hosts.

Nothing about this appears natural.

Another exceptional trait of the Wuhan Strain COVID-19 is that not only does it form its own clade, it's calculated to have diverged from SARS and its other sister coronaviruses some 260 years ago.[69] And yet in all that time, while it looks like every other branch of the coronavirus tree was busy branching-off into countless variants,[70] if it emerged naturally, COVID-19 somehow spent a quarter of a millennium as the lone known example of its clade, somehow not mutating into related lineages in all that time. Another simpler explanation is that this apparent hereditary distance and uniqueness is just the result of being altered in a lab. And although two distinct strains of COVID-19 have been identified, there's no reason to believe this mutational differentiation happened before contact with humans in December of 2019. Additionally, when neutral sites, the specific points in the genome which most reliably show evolutionary change, were examined: COVID-19 looks even more evolutionarily distant from any of its possible relatives.[71]

Giving further credence to the idea that the Wuhan Strain was bio-engineered is the existence of a patent application from 2005 that looks to modulate a coronavirus' spike-protein genes[72] - the precise region altered by Zhengli Shi at UNC to make a hyper-virulent strain of coronavirus, and whose alteration and adaptation would explain the Wuhan Strain's unusual behavior as discussed above. And notably, this patent was filed by a researcher whose only affiliation is with the University of Wuhan.

Adding to the intrigue was the posting of a pre-print on ResearchGate funded by the Chinese National Natural Science Foundation that concluded: “somebody was entangled with the evolution of 2019-nCoV coronavirus. In addition to origins of natural recombination and intermediate host, the killer coronavirus probably originated from a laboratory in Wuhan.”[73] However the paper was quickly retracted and both scientists responsible for it deleted their profiles off ResearchGate entirely. And furthering the perception of a cover-up, back January 1st the lab was ordered to destroy all of the samples of COVID-19 that it’d collected, and then on January 2nd, the Wuhan Institute of Virology's director sent out a memo forbidding discussion of an "unknown pneumonia in Wuhan,"[74] making it abundantly clear that the Chinese government knew about this outbreak long before they took any steps to contain it, or made any public announcement.

Given the above facts, either a coronavirus spontaneously mutated and jumped to humans at a wet market or deep in some random bat cave which just so happened to be 20 miles from China's only ABSL-4 virology lab, a virus with an unusually slippery never-before-seen genome that's evading zoological classification, that may be as much as twenty-times more contagious than SARS and whose spike-protein region which allows it to enter host cells holds an unique HIV-like signature with the concomitant clinical response, that somehow managed to infect its patient zero who had no connection to this market, and then be so fined-tuned to humans that it's gone on to create the single greatest public health crisis in Chinese history with approaching 100 million citizens locked-down or quarantined - also causing Mongolia to close its border with its largest trading partner for the first time in modern history and Russia to ban Chinese citizens from entry into their country.

Or, Chinese scientists failed to follow correct sanitation protocols possibly while in a rush leading up to an international virological conference and during their boisterous holiday season, something that had been anticipated since the opening of the ABSL-4 lab and has happened at least four times previously, and accidentally released this bio-engineered Wuhan Strain - likely created by scientists researching immunotherapy regimes against bat coronaviruses, who've already demonstrated the ability to perform every step necessary to bio-engineer the Wuhan Strain COVID-19 - into their population, and now the world. As would be expected, this virus appears to have been bio-engineered at the spike-protein genes which was already done at UNC to make an extraordinarily virulent coronavirus.

No facts point against this conclusion.

The Chinese government has been trying to prevent the full story about what's going on from getting out, probably since it wants the realpolitikal scales to be even, since they're now facing a severe pandemic and depopulation event. However governments acting in their own self-interest is nothing new, nor or is it at the root of our current crisis – political regimes have always and will always put their own self-preservation first, often while making calculations regarding social harmony that outsiders might weigh differently. And China is certainly not the only nation pursuing this gain-of-function research, and so demonizing an entire nation for what may well be one individual’s mistake does nothing to get to the root of the issue – scientists

who play God and then shirk all accountability when their human fallibility comes to light. Whether or not the Wuhan Strain COVID-19 is ever conclusively demonstrated to have been subject to human augmentation, it is only a matter of time until a virus that's been subject to gain-of-function of research escapes, and a prospective Golden Goose is turned into a virological Pandora's Box that humanity may never be able to close.

An immediate international moratorium on all dual-use gain-of-function research must be instated and all existing experimentation must be autoclaved, only greed and hubris have ever been served by attempting this type of genetic manipulation. Humanity does not need a vaccine against HIV derived from a coronavirus, nor do we need to be tinkering with viruses that holds the potential to wipe a significant percentage of us off the face of the Earth.

Failure to embrace such a ban may effectively become a death sentence for our species, assuming we aren't already on our last mile.

Sign the position calling for an end for dual-use gain-of-function research here

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