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Weaponized Pathogens and the SARS-CoV-2 Pandemic

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ABSTRACT

This review zeros in on the aspect of vaccine theory, practice, and research that is the most dangerous, the most controversial, and that is at the epicenter of the alleged SARS-CoV-2 "pandemic". Regardless whether the "pandemic" itself is real or an illusion manufactured out of fear by vested interests, it is central to ethics and policy discussions seeking to understand bioweapons research in general. The official involvement of the USA in civilian bioweapons research dates at least from World War II under President Franklin Delano Roosevelt. The historical records, cloaked in secrecy until after the Anthrax mailing of 2001, reveal an intimate connection to vaccine research and development, its governmental protection from public scrutiny, and from citizen initiated lawsuits. It is an industry that has released dangerous weaponized pathogens by accident and by sinister designs supposedly compensated in the peace-loving nations by unrealistic hopes in non-existent counter-measures for outbreaks, including epidemiological tracking after the fact, vaccines being researched to counter the weaponization of pathogens being studied, immunity enhancing drugs, and downstream hoped for blood sera containing antibodies. Critical questions concern the ratio of real-risks to hoped-for-benefits, the "mitigating" measures "governments" (especially in the USA) have supposedly established to prevent pandemic outbreaks from bioweapons research, and how all that has played out in the instance of SARS-CoV-2.

Keywords: *avian influenza (HPAI), bioweapons research, coronaviruses, COVID-19 pandemic*, *chronic noncommunicable diseases, dual use research, flu vaccines, gain of function*, *H1N1, H5N1, H7N9, Middle East respiratory syndrome (MERS), severe acute respiratory syndrome (SARS), SARS-CoV-2*, *weaponized pathogens*

The Path to the Present Pandemic

In 1942, the War Research Service — the USA's official bioweapons research program — was established as a "civilian" agency in Franklin D. Roosevelt's "New Deal". It was headed by George W. Merck (see **Error! Reference source not found.** 2018), the second President of Merck Pharmaceuticals in the USA, an off-branch of the oldest pharmaceutical company in the world. It is still largely owned by the Merck family dating from 1668 in Darmstadt, Germany (Anon 2020d; Staff

at Manager Magazine 2006). Merck remains one of the world's leading manufacturers of vaccines and one of the 10 largest pharmaceutical money-makers in the world (BizVibe 2020).

Bioweapons Research and Vaccines

Figure 1. George Merck in 1952. Public domain.

Bioweapons and vaccine research may seem somewhat like the opposite sides of the same coin. On the bioweapons side the goal is to make certain disease agents more harmful, and on the vaccine side the objective is to make potential infections less harmful. But no matter how we turn the coin, the two sides are natural cognates — the two faces of a single industry. Developing remedies for microbial pathogens requires knowledge about their genetic construction which is also necessary for increasing the power of selected pathogens to do harm.

From its inception, bioweapons research has been regarded as the evil twin lurking in the shadows while vaccine research has basked in the sunshine of public approval. Meanwhile, fear generated by the threat of offensive bioweapons has motivated a wall of protection around the world's vaccine industry (College of Physicians of Philadelphia 2018). Legal barriers protecting manufacturers of vaccines are especially strong in the USA (Rovner 2005a; Hensley 2011b; USLegal 2016) — the world's largest importer of pharmaceuticals at $99.7 billion (BizVibe 2020) — and in Germany (Picheta 2019), the world's largest exporter of them at $84.7 billion (BizVibe 2020). However, the titanium bubble protecting the vaccine producers racing toward one or more SARS-CoV-2 vaccines (Andrews 2020) is becoming a little more transparent, certainly to much of the whole world through open access academic (peer-reviewed) journals enabling critical discussion of theory, practice, and experimental research.

Fort Detrick, Maryland

The US bioweapons program was originally housed at Fort Detrick, Maryland and in 1944 its oversight was shifted to the US Army's Chemical Warfare Service created during World War I. During that conflict, it was discovered that the Germans were using *Bacillus anthracis* (anthrax) and *Burkholderia mallei* (the bacterium that produced glanders disease) to infect animals being shipped to the enemies of Germany by certain neutral countries (Wheelis 1998). When the US entered the war in 1917 the American counterpart of the Merck company was "expropriated" — meaning its German ownership was terminated and it was taken over by the US government. In 1919, it was, however, re-acquired by George Merck, and now a hundred and one years later both the US company and its smaller German counterpart still remain largely under the ownership and control of the Merck family. In 1957, George W. Merck died at the age of 63 (Anon 2020d). He towered over others in his generation and from his 6 feet and 5 inches in height he uttered the lofty claim that medicines were created "for people not for profits". Coincidentally, the SARS-CoV-2 pandemic, persisting at least in theory throughout 2020 — according to measures grounded largely in tests

known to produce invalid positives in about 99% of the so-called "cases" (so mild as to be completely asymptomatic) of SARS-CoV-2 (Kirkham and Yeadon 2020; Yeadon 2020; also Lyons-Weiler in this issue) — marks the 63rd anniversary of Merck's passing. The public face on the involvement of the bioweapons industry of the USA was all about vaccines while secretly Merck and collaborators were engaged in work that would have given any sensible person the chills.

"Gain of Function" Research and "Dual Use Research of Concern"

Bioweapons research over the last two decades has variously been referred to as "dual use" and more recently "gain-of function" research. These innocent sounding phrases were preferred over the straight-forward "bioweapons" moniker. Later the words "of concern" would be added keeping a poker face in place while acknowledging that bioweapons research is a serious danger to everyone on the planet. After the 2001 mailout of anthrax spores from a source believed to have originated at Fort Detrick, Maryland (Small, Klusaritz, and Muller 2002; Lindler, Lebeda, and Korch 2004, pp. x, 37) — because as one expert, Dr. Quinlisk, testifying before a Congressional Subcommittee put it, "you can't do smallpox or weaponized anthrax in your garage" (Lindler et al. 2004:36) — early in the 21st century the reality of bioweapons loomed in the public imagination and onto the world stage.

After the mailout of weaponized Anthrax spores, what began as whispers behind closed doors about "dual use research of concern" (DURC) and "gain of function research of concern" (GOFROC) were destined to be shouted from rooftops about the ongoing "pandemic" that seems now quite certainly to have been caused by one of the pathogens under study in a multitude of poorly regulated, well-funded, and notoriously insecure bioweapons laboratories of the world (Madjid et al. 2003; Zapanta and Ghorab 2014; Duprex et al. 2015; Evans, Lipsitch, and Levinson 2015; Silver 2015; N. G. Evans 2018; Bhadelia et al. 2019; Latham and Wilson 2020a, 2020b). In plain English, bioweapons are "potential pandemic pathogens" (PPPs) in development, or in plainer language, they are *half-baked and poorly understood deliberately intensified disease causing agents*. They may be poisons produced by disease agents, the active "pathogens" themselves, or they may be the self-same pathogens deliberately "enhanced" like a sharpened sword or a more powerful bomb enabling greater lethality.

Are Communicable Diseases the Greatest Threat to World Health?

Kankeu et al. (2013) asserted that by 2010 it was no longer infectious (communicable) diseases that were the greatest threat to world health. Rather "non-communicable [non-infectious] diseases (NCDs)" had become "the most important cause of mortality worldwide". Calitz et al. (2015) observed that "chronic non-communicable diseases (NCDs) cause the majority of premature deaths, disability, and healthcare expenditures in the U.S." That being noted, funding for research on the prevention of such conditions, they estimated to account for less than 10% of the annual budget of the National Institutes of Health. They concluded that with "the current burden of disease" there is a "funding misalignment" — too much spending on infectious diseases and too little on the prevention of the more costly NCDs. So, prior to the SARS-CoV-2, what was driving the huge upswing in NCDs? The relevant research suggests straightforwardly that NCDs were and are mainly being caused by toxic exposures coming to the unsuspecting public mostly through prescription drugs, medical procedures, and through the most protected industry of all, vaccines.

Now the SARS-CoV-2 pandemic has removed all doubt that the horrendously expensive DURC and GOF research agendas were fraught with mortal danger. Money would have been better spent studying ways to prevent toxic exposures and to strengthen natural human immune defenses. Instead, the vast community of medical researchers, trusting in and joining with the cumbersome, ill-informed, and inefficient government bureaucracy invested billions (and now trillions with the SARS-CoV-2) of dollars in research aiming to transform one or many "potential pandemic pathogens" (PPPs) into bioweapons. The result? Either a real pandemic or an illusion so complete that it has had the impact of a genuine pandemic devastating world economies and resulting in the greatest transfer of wealth from lower and middle classes to rich global technocrats in the history of the world (see the Child Heath Defense paper in this issue).

The intrinsic flaw in the DURC/GOFROC agenda is that the bureaucrats who approved it knew from the start that they did not have the capacity to manage the sort of monsters they were hell bent on creating. The irony of their efforts to understand bioweapons that might be produced by some wicked enemy is that the peace-loving public in the USA was duped into believing the absurd proposition that a monster created by the "good guys" would somehow be easier to manage than one created by an evil state or a non-state cabal. Yet that absurd proposition was backed only by fear of threats and empty government promises about "mitigating regulations" that actually never had a chance of being enforced to prevent a manufactured pandemic.

Intensifying Pathogens

The ongoing DURC research sponsored by the United States government (though not necessarily conducted in the USA), was loosely described in a wordy policy statement in 2012 as "research that involves one or more of the agents or toxins . . . which pose the greatest risk of deliberate misuse with most significant potential for mass casualties or devastating effects to the economy, critical infrastructure, or public confidence, and produces, aims to produce, or is reasonably anticipated to produce one or more of the [following] effects" (EPA 2012):

> a) Enhances the harmful consequences of the agent or toxin;
>
> b) Disrupts immunity or the effectiveness of an immunization against the agent or toxin without clinical or agricultural justification;
>
> c) Confers to the agent or toxin resistance to clinically or agriculturally useful prophylactic or therapeutic interventions against that agent or toxin or facilitates their ability to evade detection methodologies;
>
> d) Increases the stability, transmissibility, or the ability to disseminate the agent or toxin;
>
> e) Alters the host range or tropism of the agent or toxin;
>
> f) Enhances the susceptibility of a host population to the agent or toxin; or
>
> g) Generates or reconstitutes an eradicated or extinct agent. . . .

Focusing on the Most Dangerous Pathogens

Disease causing agents listed in that 2012 EPA document included the following:

> Avian influenza virus (highly pathogenic) [bird flu];
>
> *Bacillus anthracis* [Anthrax];

Botulinum neurotoxin [a neurotoxic poison produced by *Clostridium botulinum* and related bacteria];

Burkholderia mallei [a bacterium causing lesions in mucosal membranes, knowns as "glanders disease", which is fatal to 95% of animals infected, and up to 50% of the humans even if treated with antibiotics, believed to have been used as a bioweapon by the Germans in WWI and WWII, and the Russians in the Sovitet-Afghan War 1982-1884];

Burkholderia pseudomallei [an infectious bacterium believed to be the biological precursor of Burkhoderia mallei and which causes melliodosis fatal to about 10% of persons treated for it and 40% for those not treated]; Ebola virus [a virus of "unknown" origins causing "hemorrhagic fever" — bleeding from the body orifices or skin, followed by death in about 25% to 90% of cases];

Foot-and-mouth disease virus [causes blisters in the mouth and feet of cattle and animals with a cloven foot];

Francisella tularensis [causes tularemia also known as "black plague"];

Marburg virus [caused hemorrhagic fever believed to originate in African fruit bat];

Reconstructed 1918 Influenza Virus [the alleged source of the Spanish flu, later asserted to be the H1N1 influenza A virus];

Rinderpest virus [causes cattle plague in animals with even number of toes, fatal to nearly 100%];

Toxin-producing strains of *Clostridium botulinum* [can cause sudden paralysis of vital organs];

Variola major virus [a smallpox virus];

Variola minor virus [another smallpox virus];

Yersinia pestis [causes the diseases known loosely as "the plague" — if it infects the lungs, then it is called "pneumonic plague"; if the blood, then, "septicemic", and if the lymph nodes, "bubonic"] . . . (EPA 2012)

Creating Unenforceable "Mitigating Measures"

So, how did the government propose to protect its people from the "enhanced" pathogens created by its own bioweapons research program? According to that same Federal document published by the US Environmental Protection Agency in 2012, "risk mitigation measures" might include the following laundry list of barely intelligible and certainly unenforceable government gobbledygook distributing responsibility across unnamed "Federal departments and agencies" along with similarly unknown and unnamed "other departments and agencies" for the following empty assurances:

(i) . . . . modifying the design or conduct of the research, . . . applying specific or enhanced biosecurity or biosafety measures, evaluating existing evidence of medical countermeasures (MCM) efficacy, or conducting experiments to determine MCM efficacy against agents or toxins resulting from DURC, and where effective MCM exist, including that information in publications, . . . referring the institution to available DURC educational tools such as http://oba.od.nih.gov/biosecurity/biosecurity.html [an html document, which is, as of November 11, 2020, no longer available from the EPA at the URL supplied] . . . regularly reviewing, at the institutional level, emerging research findings for additional DURC. . . . requesting that institutions notify funding departments or agencies if additional DURC is identified, and propose modifications to the risk mitigation plan, as needed . . . determining the venue and mode of communication (addressing content, timing, and possibly the extent of distribution of the information) to communicate the research responsibly . . . reviewing annual progress reports from Principal Investigators to determine if DURC results have been generated, and if so, flagging them for institutional attention and applying potential mitigation measures as described above, as necessary. . . . if the risks posed by the research cannot be adequately mitigated with the measures above, Federal departments and agencies will determine whether it is appropriate to . . . request voluntary redaction of the research publications or communications . . . classify the research: . . . In accordance with National Security Decision Directive/NSDD-189, departments and agencies will make classification determinations . . . . Actions taken to restrict the publication of technology may have implications under export control laws and regulations (e.g., 15 CFR parts 730-774 and 22 CFR parts 120-130) . . . . the scope of their

> classification authorities and appropriate classification guidelines or may consult with other departments and agencies to make these determinations.
>
> (ii) Departments and agencies may consider whether to refer classified research to another department or agency for funding. (EPA 2012)

Benevolence v. Harm

In 2014 there was an update on the EPA bioweapons policy of 2012. The later formulation opened with a defense followed by a definition of "dual use research":

> Despite its value and benefits [which are hoped for future outcomes], certain types of research conducted for legitimate purposes can be utilized for both benevolent and harmful purposes [the latter being real outcomes of accidents or deliberate releases as in the Anthrax case]. Such research is called "dual use research."

The "benevolent" part is never really spelled out. Apparently it was to consist only of blocking "harmful purposes" intrinsic to research aiming to increase the power of manipulated pathogens to do harm. Driven by *the fear of possible harm* the US Department of Health and Human Services promoted and continues to promote the very research that has the power to turn the feared possibilities into *genuine realities*.

Paraphrasing the Protective Game Plan

The argument works like this: to prevent the horrible potential damage of a Frankensteinian virus that might be created by some known or unknown unfriendly evil power, the USA should create monsters of our own in order to discover their inner workings so we will be able to defend ourselves against them. The bureaucrats spelled out their plan:

> Dual use research of concern [DURC] is a subset of dual use research defined as: "life sciences research that, based on current understanding, can be reasonably anticipated to provide knowledge, information, products, or technologies that could be directly misapplied [note that there is no friendly application of such knowledge] to pose a significant threat with broad potential consequences to public health and safety, agricultural crops and other plants, animals, the environment, materiel, or national security" [quoted verbatim from the 2012 document]. The United States Government Policy for Institutional Oversight of Life Sciences Dual Use Research of Concern articulates the practices and procedures required to ensure that dual use research of concern is identified at the institutional level and risk mitigation measures are implemented as necessary. (US Department of Health and Human Services 2014)

Thus, realizing that it is dangerous to create monsters that we do not yet know how to defeat, they claimed that it is necessary to take such dire risks, threatening the whole world population, in order to protect ourselves from the monsters that someone else *might be* trying to create. Following the initial defense and the self-sustaining definition of dangerous "dual use research" the government document of 2014 invoked the fear of weaponized pathogens to justify research to produce such "potential pandemic pathogens" (PPPs) in order to learn how, after we have created them, to defend against them. Also, the creators of the doctrine underlying DURC expressed the confidence that the governmental or private agencies engaging in DURC would figure out how to mitigate the dangers of doing the research on the fly, so to speak. It is a little like protecting yourself from falling off the face of a cliff by climbing higher and higher in the hope that you will be able to prevent yourself from falling by constantly increasing the risk of a deadly fall right up until the point when you fall from the cliff. Or, such a program of research could be compared to starting a potential

conflagration in a city or forest in the hope that you will be able to learn how to extinguish the fire once it is fully on the verge of getting completely out of control.

Concentrating GOF research on Airborne PPPs

By 2014, the government's new policy was re-focused on the rapidly reproducing viruses constituting "enhanced" pathogens — ones with sufficient lethality to possibly cause a world-wide pandemic. They began to direct most of their attention to pathogens that could be transmitted in the air. This brought into focus the influenza viruses along with the corona viruses SARS, MERS, and so on because of their facility to spread rapidly from one person to another in the human population (Armesto et al. 2011; Evans 2013). With such particularly infectious viruses explicitly in mind, not only did the US Department of Health and Human Services construct a new policy statement in 2014, but, in view of the obvious dangers associated with that shifting focus, the Obama White House officially proposed a "pause" on "new funding" of "gain-of-function" studies aiming specifically to increase the lethality of such viruses for human beings while at the same time it authorized on-going efforts already underway to continue in order to protect the public (US Department of Health and Human Services 2014). Let's think this through: they ordered a halt because of the dangers, but, oh wait a minute, they ordered the continuation of any projects that were aiming to mitigate the known dangers the ongoing work might lead to. Indeed. Does that make any sense? The wisdom of our government was to suppose that risks were worth taking only if they were severe enough to actually threaten the security of the whole nation. Here, in their own words, are the crucial parts of that policy statement coming straight from the Obama administration:

> In light of recent concerns regarding biosafety and biosecurity [note the euphemistic nature of these terms when what is really at stake is the potential of an accidental release and the likelihood of an intentional one], effective immediately, the U.S. Government (USG) will pause new USG funding for gain-of-function research on influenza, MERS or SARS viruses. . . such that the virus would have enhanced pathogenicity and/or transmissibility in mammals via the respiratory route. (The White House 2014)

But they qualified the alleged "pause" in funding by allowing the only kind of "exception" that would conceivably justify any such "gain-of-function" research in footnote 1 saying effectively that whatever ongoing projects might be judged necessary to protect the public from the threat of such pathogens should be allowed to continue without regard for the alleged pause in funding:

> An exception from the research pause may be obtained if the head of the USG funding agency determines that the research is urgently necessary to protect the public health or national security. (The White House 2014)

So, what justifies the continuation of the dangerous research? The very fact that it is dangerous was invoked to justify going forward with it irrespective of the pause.

Another Committee Promising Mitigation of Deadly Threats

By 2015, within the National Institutes of Health, under the umbrella of the ubiquitous US Department of Health and Human Services, to deal with the growing likelihood that one or more PPPs might be released, whether intentionally or by accident, the government created another committee, the National Science Advisory Board for Biosecurity (NSABB; Stanley 2015). Their charge according to the 2014 policy statement was

> to draft . . . recommendations for gain-of-function research that will be reviewed by the broader life sciences community. The NSABB will serve as the official federal advisory body for providing advice on oversight of

> this area of dual use research, in keeping with federal rules and regulations. As a second step, coincident with NSABB recommendations, the National Research Council (NRC) of the National Academies then will be asked to convene a scientific conference focused on the issues associated with gain-of-function research and will include the review and discussion of the NSABB draft recommendations. This NRC conference will provide a mechanism both to engage the life sciences community as well as solicit feedback on optimal approaches to ensure effective federal oversight of gain-of-function research. The life sciences community will be encouraged to provide input through both the NRC and NSABB deliberative processes. The NSABB, informed by NRC feedback, will deliver recommendations to the Secretary of Health and Human Services, the Director of the National Institutes of Health, and the heads of all federal entities that conduct, support, or have an interest in life sciences research (including the Assistants to the President for Homeland Security and Counterterrorism and for Science and Technology). The final NSABB recommendations and the outcomes of the NRC conference will inform the development and adoption of a new U.S. Government policy governing the funding and conduct of gain-of-function research. Upon adoption of a federal gain-of-function policy, the U.S. Government will declare the end of the research funding pause. (US Department of Health and Human Services 2014)

Protests and Promises

Apparently, the assurances coming from the NSABB did not make independent researchers like Evans, Lipsitch, and Levinson (2015) feel sufficiently safe. They were particularly concerned about aerosol PPPs that might spin off from DURC and GOF research. They proposed

> an ethical framework for evaluating biosafety risks of gain-of-function (GOF) experiments that create novel strains of influenza expected to be virulent and transmissible in humans, so-called potential pandemic pathogens (PPPs). Such research raises ethical concerns because of the risk that accidental release from a laboratory could lead to extensive or even global spread of a virulent pathogen. (Evans et al. 2015)

On May 6, 2016 the NSABB Working Group, a select panel of experts, concluded with a summary of seven "findings":

> Finding 1: There are many types of GOF studies and not all of them have the same level of risks. Only a small subset of GOF research — GOF research of concern (GOFROC) — entail risks that are potentially significant enough to warrant additional oversight. [Evidently, the expert panelists concluded that they were competent to say in advance which infectious pathogens could safely be made more lethal than ever before while other lethal pathogens would be made unsafe by increasing their lethality. In effect, as Rampton and Stauber (2001) put it in their tongue-in-cheek title, the government's message to the public was, *Trust Us: We're Experts.*]
>
> Finding 2: The U.S. government has several policies in place for identifying and managing risks associated with life sciences research. There are several points throughout the research life cycle where, if the policies are implemented effectively, risks can be managed and oversight of GOF research of concern could be implemented. [So, the panel of experts here again play the *Trust Us: We're Experts* card. They claim their expertise will protect the trusting public because they can discern between really dangerous PPPs and safe PPPs.]
>
> Finding 3. Oversight policies vary in scope and applicability, and do not cover all potential GOFROC, therefore, current oversight is not sufficient for all GOFROC. [Uh oh. Finding 3 suggests the experts cannot be blamed if things go haywire. They issued their own "Get Out of Jail Free" card. It can be played in this game of government monopoly just in case the *Trust Us: We're Experts* card fails to convince.]
>
> Finding 4. An adaptive policy approach is a desirable way to ensure that oversight and risk mitigation measures remain commensurate with the risks associated with the research and the benefits of the research are being fully realized. [By maintaining a certain plasticity, they will be able to bend the policy to accommodate any eventuality. It's a convenient policy for that reason. This is the "One Size Fits All" card that can be played even if the PPP gets way out of control.]

> Finding **5.** There are life sciences research studies, including possibly some GOF research of concern, that should not be conducted because the potential risks associated with the study are not justified by the potential benefits. Decisions about whether specific GOFROC should be permitted will entail an assessment of the potential risks and anticipated benefits associated with the individual experiment in question. The scientific merit of a study is a central consideration during the review of proposed studies but other considerations, including legal, ethical, public health, and societal values are also important and need to be taken into account. [This is the recipe finding: if a certain mix does not work, the panel of experts proposes to add other ingredients on the basis of their expertise until everything is fine. This is the "The Best Exotic Marigold Hotel" rule: if everything is not perfect the recipe is just not finished yet.]
>
> Finding 6. Managing risks associated with GOF research of concern, like all life sciences research, requires both Federal-level and institutional oversight, awareness and compliance, and a commitment by all stakeholders to safety and security. [This one is the "We Feel Good About This So Everything Will Be Fine" rule. They took a vote and the group agreed so it must be right and true and safe and good.]
>
> Finding 7. Funding and conducting GOF research of concern involves many issues that are international in nature. [This finding is the "Other Nations May Be Harmed: So Let Them Beware" caveat.] (National Science Advisory Board for Biosecurity 2016)

Then on December 19, 2017, the Department of Health and Human Services (DHHS) issued its *Framework for Guiding Funding Decisions about Proposed Research Involving Enhanced Potential Pandemic Pathogens* and the funding pause (as noted by Evans 2018) was officially ended. The requirements for on-going funded research were that it had to be "scientifically sound", to involve "a credible source" of a PPP, to justify itself by a risk benefits analysis, also to show that there is no way to get the benefits with less risk, that the PI and institution can handle any breaches of security or other problems that might occur, that the researchers promise to communicate their results, that Federal oversight is assured, and that the whole project is ethically justifiable (see Office of Science and Technology Policy 2017, and Evans 2018). The upshot was that all the world should feel quite safe. The experts of the NSABB are on the job and have provided safeguards.

Rushing in Where Angels Fear to Tread

By 2019, the phrase "gain-of-function" research seemed to have swallowed up the former DURC and GOFROC categories because, logically speaking, contrary to Casadevall and Imperiale (2014) as well as Imperiale and Casadevall (2020), *there is no way to do any experimental GOF (genetic engineering of PPPs) research without introducing the danger of turning a "potential pandemic pathogen" into an uncontrollable actual pandemic catastrophe*. The proponents of such research — including Casadevall and colleagues as well as the amorphous US Department of Health and Human Services — nevertheless, have insisted as a matter of policy that the inherent risks of experimental GOF research are compensated by the assertion that hypothetically worse things can somehow be avoided by boldly creating real and actual "enhanced" pathogens in order to avoid the threat of hypothetical developments that might occur:

> Gain-of-function (GOF) studies, or research that improves the ability of a pathogen to cause disease, *help define the fundamental nature of human-pathogen interactions* [my italics to show that the government is talking about assessing something of which researchers are presently ignorant], thereby enabling assessment of the pandemic potential [a hypothetical risk] of emerging infectious agents [ones feared and not fully understood], informing public health and preparedness [future hypothetical] efforts, and furthering medical countermeasure development [non-existent ones, against entities to be created by the researchers doing GOF].

"Biosafety" and "Biosecurity": Read, "Dangerous" and "Insecure" Research

Now, having offered the best argument they could muster for deliberately creating PPPs that might kill millions of people who have no way of knowing what is about to hit them, the government in its wisdom — tempering public fear by "getting out ahead of it" with wise-sounding journalistic language — "comes clean" on the evil it is about to unleash with a subtle bald-faced lie sneaking in with a parade of known facts:

> Gain-of-function studies may entail biosafety and biosecurity risks [what!!!! they certainly do entail such risks by definition and by the historical incidents that have already occurred]; therefore, the [very real] risks and [the wish-list of hypothetical] benefits of gain-of function research must be evaluated, both in the context of recent U.S. [real] biosafety incidents [ones that have already occurred] and to keep pace with new technological developments [that enable a host of new and dangerous GOF/genetic experiments with PPPs], in order to determine which types of studies should go forward and under what conditions. (US Department of Health and Human Services 2019)

By such cleverly composed phrasing, possibly confusing its authors along with the rest of the CDC and government pundits, the potential victims of GOF research are supposed to be assured that the government itself can protect them. By its own declaration it is the sole line of defense against its own dangerous creations. The government must authorize and fund GOF research to effectively weaponize PPPs in order to assess the unknown risks of harm that are certain to follow — all this, in order that the ginned up fear of potential harm may protect the public from the real harm that GOF researchers are being authorized and funded to create — all this for the greater good of the public.

Following the Line of GOF Research to SARS-CoV-2

According to Casadevall and Imperiale (2014), focusing on influenza viruses primarily spread through the air and impacting the respiratory systems — but also taking into consideration the coronaviruses, especially, SARS — boldly assert without any evidence whatever that GOF research with such PPPs is "of utmost importance to societal well-being" (p. 1). They also claim that there has been "a vigorous debate over so-called 'gain-of-function' (GOF) experiments involving pathogens with pandemic potential . . . about the value, safety, ethics, and validity of this type of research" (p. 1). Actually any such debate had scarcely begun to get underway.

Casadevall and Imperiale (2014) harked back to the 1975 conference at Asilomar in California where Paul Berg and others met to consider the potential dangers of genetically engineered monsters made possible the discovery of how to use recombinant DNA to modify and combine genomic components of diverse organisms (Berg et al. 1975; Berg 2008; Yi 2015). Casadevall and Imperiale proposed in 2014 that there should be another such conference to "finding a way to allow GOF research to go forward with minimal risk and maximal benefit" (p. 4). They suggested a "need to lower the level of rhetoric" in order to avoid "the potential to hinder future research and leave society more vulnerable" (p. 4). But it is not the power of the non-existent "debate" to do harm, but of real pandemic pathogens created by microbiologists in the 21st century. Nor can debate protect us from the ongoing SARS-CoV-2 "pandemic" (real or imagined).

GOF Researchers Transform Feared Fictions into Dangerous Realities

GOF research begins with real pathogens with a history of actually causing morbidity (infection or harm) and mortality (death) in human populations, and, possibly, in other species that humans depend on such as farm animals and food crops. The tortured rationale for enhancing, actually, weaponizing, dangerous pathogens such as *Anthracis bacillus*, *Burkholderia mallei*, *Yersinia pestis,* and so on — or the more easily delivered influenza and corona viruses that are in focus in the most recent GOF experiments — is to better understand their inner workings. The strangeness of maximizing the threat of lethal pathogens so those threats can be minimized is hardly removed by noting that the "epistemological value" of such "experiments" is that "they directly imply causality" (Casadevall and Imperiale 2014:2). That is to say, if a newly created GOF pathogen from chickens, bats, or monkeys leads to a pandemic causing wide-spread morbidity and mortality in humans, intelligent people will at least be able to infer that the researchers caused the new disease.

So, anyone not steeped in GOF research might ask how the risk-benefits analysis works for that kind of research. From an actuarial perspective, or using elementary algebra, the general objective of preventing harm from pathogens is to maximize the benefits of a medicine or procedure while minimizing the risks of deploying it. In a perfect world, risks would be reduced to all the way to zero, and benefits would be raised to the level of the greatest possible multitude of multitudes merging at continuity, along the lines of C. S. Peirce (Peirce 1898; Putnam and Peirce 1992; Zalamea 2003). The ratio of risk to benefit would be incalculably great favoring the positive side of the leger. But, of course, we do not live in a perfect world, so what is the effect of changing the ratio of risk to benefits by GOF research? Casadevall and Imperiale (2014) describe that ratio as a "conumdrum" (p. 2). They claim that the risks and benefits of GOF research are "quantitative" but say there are "problems in calculating the numerator [risks] and denominator [benefits]" (p. 3) and that proponents in the hypothetical "debate" tend to magnify the "benefits" in a "risk-benefit assessment" while the opponents focus on "risks" (p. 4).

However, the hypothetical "vigorous debate" about GOF research is irrelevant. It is not taking place while the world-wide SARS-CoV-2 crisis is already underway. With respect to quantification of risks and benefits there is also a huge difference in the calculation of the numerator (risks) that are known to be real — experimental GOF modifications that simultaneously increase (1) transmissibility, (2) host range (from bats to humans, say), and (3) virulence — produce real increases in the numerator of any risk-benefits ratio. By contrast, the denominator is based only on hypothetical benefits, ones that only follow if the GOF research actually reduces the numerator. We are talking about a real strange algebraic construction here.

Whereas the real risks are actualized deliberately by the experimenters engaging in the GOF research — to enable the all important cause-effect inferencing — the future "benefits" of such research are entirely hypothetical, unknown, and incalculable. The hypothetical motive for such research that (Casadevall and Imperiale 2014) lift to a great height is to increase, supposedly, our preparedness to avoid and/or deal with a world-wide pandemic. The difficulty from the "risk-benefits" analysis they are asking us to bet our lives and the the future of the world on, is that it is grounded in non-existent promised future winnings in the form of remedies for any real pandemic that is virtually certain to be caused by GOF researchers. If we think through the actual reality associated with the GOF game plan over time, the longer it persists, the greater is the likelihood approaching certainty at a limit,

that it will actually produce a real pandemic. The logical reality is that the numerator keeps getting bigger irrespective of whatever the denominator is construed to be. Suppose the denominator represents health, wealth, and well-being for all the billions of people on earth, but GOF research continues to increase the risk factor (the numerator) until a genuine pandemic occurs. There is no logic by which the denominator consisting of hypothetical benefits described as "the greater good" can compensate for the certainty that GOF research continues over time to increase the likelihood of an accidental or intentional release of a PPP that can become a real pandemic. The risks-benefit analysis by any reasonable interpretation suggests that GOF research should be shut down in the USA and throughout the world. Existing PPPs should be annihilated, not cultured in poorly regulated laboratories.

Reasonable Probability Estimates: The Known v. Unknown

The funding of ongoing GOF research is like hiring arsonists in a huge city (the whole of civilization) to start fires in order that the fire department (the governments of the world, fractionated as they are) can learn more about how to prevent the whole of civilization or a large part of it from being incinerated. As pointed out by Edwin Thompson Jaynes (1957a, 1957b, 1963, 1965), in his proofs and explanation of "the maximum entropy principle", whenever a "probability distribution" must be estimated on "the basis of partial knowledge . . . the maximum-entropy estimate . . . [that is] the least biased estimate possible on the given information . . . is maximally noncommittal with regard to missing information" (1957a:620). The upshot of the Jaynes' proofs is that the known risks in GOF research so vastly outweigh the hypothetical unrealistically promised benefits, the analysis can only come down on the side of the opponents of such research. Its hypothetical benefits, like the hopes of a compulsive gambler, can only lead toward, never away from, the much feared catastrophe the deranged gambler keeps betting against. In GOF research the maximum entropy principle applies with a vengeance.

The approach of the advocates of GOF research is something like hiring professional fire fighters (some one or more of whom may be arsonists) provided with optimal incendiaries and explosives (known deadly pathogens), to start controlled fires here and there (in GOF laboratories) threatening a large city (the whole human population) in the hopes that doing so will enable fire fighters (virologists, medical professionals, vaccine manufacturers) to protect the city (the world's human population) from the fire or explosion (pandemic) that is certain to reduce large parts of the city to ashes. The law of maximum entropy assures us that such an approach can only trend toward the feared disaster that the GOF research is supposedly aiming to prevent.

Facts on the Real Side of GOF Research (Actual Risks)

Bearing in mind the maximum entropy principle of the mathematician and physicist, Jaynes, it does not require advanced math to see that GOF researchers begin their experiments like an arsonist bent on starting hotter and less containable fires that are likely to maximize damage. They begin their work with lethal pathogens that they know have led to epidemics in the past and that, with the sort of experimental manipulations the researchers are able to come up with, have the potential to lead to a world-wide epidemic, a pandemic like the SARS-CoV-2 crisis. So how is that working out for the proponents of GOF research?

In Search of an Adequate Theory of Causation

It is difficult to fix any particular point for the beginnings of GOF research, but some dates for the discovery of key pathogens are known. For instance, the discovery of *Anthracis Bacillus* is known to have occurred at least by 1877. What is disputed is who got to it first, and just when it was determined, if it was ever actually proved that the bacterium isolated from diseased animals or humans, was the sole cause of the disease. It seems that Heinrich Hermann Robert Koch [born 1843- died 1910], who cultured and injected the pathogen into animals that promptly developed the disease, and Louis Pasteur, who is credited with developing a vaccine for Anthrax, both laid claim to the discovery that *Anthracis Bacillus* causes Anthrax disease (Carter 1988). That particular controversy would also call attention to Koch's postulates about experimentally demonstrating pathogenic causation as now being practiced in GOF research. Likewise, the intimacy of the relation between the theory underlying vaccines and the research with pathogens involved in their development was clarified in the dispute between Koch and Pasteur.

Isolation and Inoculation

In their efforts to experimentally determine the cause of Anthrax disease, both Pasteur and Koch used the strategy of isolating the suspected "germ", purifying it in successive cultures, and then exposing healthy animals to it in order to see if they could be infected by it. This was not a new methodology, but it was Pasteur who doggedly pursued it, thinking aloud in various publications (see the notes in Carter, 1988), that he would be able to produce a "weakened" (attenuated) variant of the pathogen, one that would not have the power to return to virulence, that could be used to prevent the disease in healthy animals deliberately exposed to it (see Carter, 1988, and his references especially, in footnotes 49-52 on p. 51). It was in following out such a line of research that Pasteur proposed the generalization of Jenner's use of *Vaccinia virus* (the cowpox virus) to inoculate humans against *Variola virus* (the smallpox virus), to propose the term "vaccine" (Pasteur 1881b) and to suggest that, in theory, it ought to be possible to find a preventative vaccine for every known pathogen (Pasteur 1880a).

The approach of "isolation and inoculation" actually employed by Pasteur and Koch, as well as certain of their predecessors, as Carter (1985:354) points out, can show that a particular entity is a *sufficient* cause without being powerful enough to prove that it is a *necessary* cause:

> For our purposes, a phenomenon C is sufficient for a phenomenon E if the occurrence of C ensures the occurrence of E. A phenomenon C is necessary for a phenomenon E if the nonoccurrence of C ensures the nonoccurrence of E. It is obvious, but frequently overlooked, that if one wants to bring about some state of affairs, or to explain something that has happened, one seeks a *sufficient* cause, since by bringing that cause to bear one can be certain that the desired effect will follow. On the other hand, if one wants to prevent or to eliminate some state of affairs, or to explain why something did not happen, one seeks a *necessary* cause, since by preventing that cause one can prevent the undesired effect.

As Koch noted during the course of his polemics against the older Pasteur, and as Pasteur had patiently noted early in his generous praise of the younger man's efforts to "prove" that *Anthracis bacillus* is the sufficient and necessary cause of Anthrax disease, sufficiency had been shown adequately, but to prove the rod-like bacterium did not contain on its surface, or in its powers of generation, some other virus or toxin capable of causing the disease would require, in principle, an infinite number of experiments.

For that reason, it is logically quite impossible — that is to say, it is a mathematically provable impossibility — for any number of experiments to rule out all possible causes other than the one singled out for study. Pasteur acknowledged that *Anthracis bacillus* appeared to be the sole cause of the symptoms of what came to be known as Anthrax disease, but there was no way, he argued, to be sure that there was not some other agent on the surface of, or contained within the bacterium, or in some other way associated with it, that was the real causal agent. General proofs reaching all possible cases can sometimes be attained by abstract mathematical or exact logical reasoning, but they can never be attained by ever so many experiments, any more than we could prove that there is no gold on Mars by transporting ever so many handfuls of dirt from Mars back to the earth and finding no gold in any of them. By contrast, one valid experiment, finding a tiny bit of Martian gold, is sufficient to refute the general negative that there is no gold on Mars, just as a single experiment with vaccinated and unvaccinated animals is powerful enough, in principle, to rule out the general propositions (hypotheses) that the vaccine is always harmless (safe), or that it is universally beneficial (effective). Experiments can rule out false general propositions.

However, the empirical proof of a completely general proposition, for instance, that only *Anthracis bacillus* can produce Anthrax disease, would require the experimental examination of all possible agents that might cause it. Such an experimental program cannot be carried out. It could never be completed. This fact was noted by Pasteur early in the exchanges between himself and Koch, and was only later used as an oblique argument against Pasteur, by Koch in which Koch inadvertently refuted his own former claims of having proved empirically (an impossibility) that *Anthracis bacillus* is the only possible cause of Anthrax disease. The fact is that the conclusion might be correct, as virtually all the world now supposes to be the case, but the theory of proof on which it was based, was fatally flawed. Sad to say, that flaw seems to have been incorporated in current GOF and vaccine research and development. However, before connecting the dots all the way to the present SARS-CoV-2 epidemic — it is necessary to take note of Pasteur's notion of "attenuation" and its relevance to current vaccine theory and practice.

The Critical Element of "Attenuation"

Pasteur was the first vaccinologist to explore the possibility that a pathogen could be artificially manipulated in a way that would weaken its harmful effects. He introduced the term "attenuation" in its current use (Moulin 1992; Pasteur 1881a, 1881b) meaning essentially the opposite of "gain-of-function". Whereas GOF researchers, by contrast, are generally seeking to make pathogens more harmful, Pasteur aimed to make them less so in order to use "attenuated" pathogens safely to inoculate animals (or humans) either to prevent their being infected by the disease associated with that pathogen, or to weaken the impact of the disease by strengthening the immune response of the inoculated animal (or person) to the pathogen. Pasteur generalized the idea of weakening a pathogen by suggesting that Jenner's inoculation of humans with cowpox could be thought of as presenting them with an "attenuated" version of the *Variola virus*, the one that causes smallpox. He made this idea explicit in his paper about chicken cholera:

> I infer that vaccine [here speaking specifically of the *Vaccinia vir*us used against *Variola*] rarely acts as a complete preventative [for smallpox]. There are cases cited of vaccinated persons who have had the *Variola*, and there are even cases of persons who have had it, afterwards, as much as three times. (L. Pasteur 1881:55)

Gradation of Effects

Pasteur observed on the basis of his cholera experiments with chickens, that although just one inoculation with the attenuated cholera virus could not prevent the disease in all instances, it always had, in his view, an ameliorative impact: it lessened the effect of the disease, or prevented it entirely. He suggested that this was so for up to three inoculations of the experimental animals he studied. It is noteworthy, however, that he was never aiming to artificially intensify the virulence of the pathogens he was studying, but to render them less virulent experimentally in order to defeat them in their most virulent natural form. He was also concerned to discover whether the pathogens under study could return to their former virulence after being attenuated. If it occurred to him to ask whether *Anthracis bacillus*, cholera, rabies, or whatever pathogen he happened to be studying could be deliberately made more virulent, transmissible, or caused to jump from chickens, rodents, or canines, say, to humans, it was never his own purpose. Pasteur was, it seems, always aiming to prevent or lessen the impact of infectious disease, not to cause them. However, with respect to causation, he was more cautious than Koch who claimed to have ascertained based on a few experiments the sufficient and necessary — the only possible — cause of the Anthrax disease in the whole universe of possible causes.

Pasteur never made such a radical claim. He himself had experimented with bacterial pathogens that prior to his time were so small and difficult to detect that their discovery was denied by many even after their existence was no longer in doubt. As historical evidence of this fact, the work of Ignaz Semmelweis (1861) showing that invisible pathogens from cadavers were unintentionally being transferred by doctors to the epithelial tissues of living patients while they were assisting women in delivering babies, was rejected by mainstream doctors typified by Carl Edvard Marius Levy (while he was head of the leading maternity institution of Europe) for about another 20 years (Anon 2015) until the "germ theory" of disease could no longer be denied because of the work, mainly of Pasteur (Pasteur 1864, 1880a, 1881a). But, Pasteur himself refused to suppose that a bacterium sufficient to cause a disease such as Anthrax was necessarily *the only possible cause of it*. Apparently he kept in mind the fact that something much smaller than a bacterium — some pathogenic element, a toxin or another pathogenic organism accompanying the bacterium, attached to it, or inside it — might be causing the disease. In the case of Anthrax, it seems that Koch's claim that it was the only cause of the disease might have been correct, but it certainly would not have been correct in the case of rabies.

The Special Case of Rabies

Pasteur's empirical disproofs of the popular theory of "spontaneous generation" (1864) also demonstrated that microbes too small to be seen by the naked eye could cause disease. He put it this way in one of his now famous Sorbonne lectures of 1864:

> The air in this room is replete with dust motes, with those tiny nothings which ought not always to be despised, for they sometimes carry sickness or death, in the form of typhus, cholera, yellow fever, and many other kinds of flux (Pasteur, 1864:12).

Thus, having demonstrated the existence of certain microbes that had long gone undetected, Pasteur was cautious not to over-generalize his findings with *Anthracis bacillus* in relation to the symptoms of the disease it was evidently sufficient to cause. Perhaps, as the younger Koch insisted, that bacterium

was the sole cause of Anthrax, but leaping to that conclusion on the basis of a few experiments was still a severe error of logic, an over-generalization that would be false in the case of many diseases that involve viruses that are now known to interact with an organism's immune defenses in a great variety of ways, some of them involving deadly deceptions. Pasteur's work with what was known as *hydrophobia* or rabies was described by Plotkin (2014). He said, "Pasteur and his colleagues . . . most clearly formulated the idea of attenuation and demonstrated its utility, first with *Pasteurella multocida*, the cause of a diarrheal disease [cholera] in chickens [Pasteur 1880b], then anthrax in sheep [Pasteur, Chamberland, and Roux 1881] and most sensationally rabies virus in animals and humans [Pasteur 1885]" (Plotkin 2014:12283-12287). Pasteur inferred that the pathogen he was dealing with was much smaller than a bacterium and he consistently used the term "virus" to describe it in spite of the fact that the first virus, the tobacco mosaic virus, would not be found for several years (Iwanowski 1892) nor would any virus become visible before the advent of the electron microscope in the 1930s. However, Pasteur correctly inferred the existence of viruses, and in 1886, he and Chamberland, one of Pasteur's laboratory assistants developed and patented (Hansen 2016) a porcelain filter that could strain out bacteria leaving viruses and their reproducing particles, virions, behind. It was that filter which would enable Iwanowski to detect the tobacco mosaic virus six years later.

Leading up to the development of that filter, Pasteur's most "sensational" inoculations with an attenuated pathogen involved the yet to be discovered rabies virus with which he began his experiments in 1882. His work would anticipate the differentiation of injections with an attenuated pathogen as contrasted with injections containing antibodies that pathogen harvested from the blood or tissues of a host that was formerly infected by it. His method of inoculation in his rabies experiments combined both types of inoculation. He not only found that pathogens in general can be rendered less virulent by merely exposing them to oxygen or heat, but he also inferred that tissue harvested from an infected animal could be passed through a series of laboratory animals to produce a further attenuation of the pathogen by what would come to be known by the term "passaging" (Anon 2020f). He found that by harvesting rabies from the marrow of a rabid dog, then injecting it into the brain of a healthy laboratory rabbit, would lead to the rabbit developing rabies within about 15 days. Then, he would similarly harvest the infection from that rabbit and inject it in another healthy one. After repeating this cycle through 20 to 25 successive rabbits, he reached what he believed to be the purest attainable form of the pathogen. If it were injected, it would produce rabies in a healthy rabbit within about seven days. Having thus isolated and purified the rabies virus as much as he believed possible, he found that he could attenuate it more and more by exposing it to dry air for longer and longer periods. It was with 13 graded levels of decreasingly attenuated virus that he was able to inoculate the nine year old boy, Joseph Meister, bitten by a rabid dog two and a half days earlier, over a ten day period of treatments, thus saving him from the horrible death of rabies. What worked in immunizing dogs against rabies also worked for humans.

On the Edge of Life?

Pasteur's research with the pathogens that were eventually judged to be the respective causes of Anthrax, cholera, and rabies, led him to speculate that cowpox is like an attenuated variant of smallpox (L. Pasteur 1881). A couple of years earlier, one of Pasteur's co-workers inadvertently allowed a culture of *Vibrio cholerae* to age for a month before injecting chickens with it. He reportedly

(VBI Vaccines, Inc. 2016) went on vacation and failed to inject the experimental chickens until he returned. But, when he did so, the chickens only became slightly ill and later recovered. Pasteur wondered if the aged bacteria, exposed to the air, oxygen in particular, had been weakened, "attenuated", over time. With that in mind he injected the same chickens, now recovered, with full strength laboratory cultured *Vibrio cholerae*. When they did not become ill, he inferred that the inoculation with "attenuated" bacteria, led to the strengthening of the immune defenses of the chickens. With that discovery, in 1879, the field of immunology in medicine was born.

Later on, with the discovery of the unbelievably intense biosignaling functions of nuclei acids and proteins interacting across time, the similarities of *Vaccinia* and *Variola* viruses could be examined in detail (Aguado, Selmes, and Smith 1992; Silverman et al. 2008). It would also be discovered that viruses, much smaller than a bacterium but vastly larger than the nucleic acid strings from which they are constructed, are, as Rybicki put it, "at the edge of life" (1990:182). They are peculiarly small complex molecular entities that depend on their power to invade the essential signaling systems of living hosts in order for the viruses themselves to propagate and thus to survive. In the cells of human beings, the central biosignaling systems attacked by pathogenic viruses are known to fall ultimately under the supervision of nuclear DNA deeply protected within barrier after barrier clear down inside the nucleolus of each nucleated cell's intelligence system of nucleic acids (Oller and Shaw 2019). While some of the RNA viruses can replicate in protoplasm, the cancer-causing viruses, according to current research, in keeping with the "depth hypothesis" Shaw and I proposed in our 2019 paper, must penetrate the barriers protecting the nucleoli of infected organisms and deceive the host's systems of replication into regarding the virus itself as worthy of multiplication.

As noted by Rybicki, viruses are necessarily "intracellular parasites" — that is to say, in order to survive they must not only be associated with a living host, but to reproduce their own genomes, whether consisting of either DNA or RNA — they must commandeer "host cell machinery to synthesize specialized self-assembled particles called virions, whose function is to contain the genome [of the virus] and transport it from cell to cell" (Rybicki 1990:182). Though some viruses, like bacteria (Dietert 2014; Min and Rhee 2015; Rao and Gershon 2015; Shamriz et al. 2016), may be "pathogenic", many are not harmful (Robinson and Pfeiffer 2014; Voelkner 2019), and like the notorious *E. coli* bacterium which is essential to a healthy large intestine in humans, many viruses are not just useful but may even be essential to optimize rather than diminish the health and well-being of the host (Piast 2019).

So Where Is the Center of Life?

Thus, comes the question, if viruses are at the "edge of life", where is the center? Biologists in general, virologists included, are unable to offer a definitive answer, though speculations abound. For example, Piast (2019) hypothesizes a continuum moving from Rybicki's "edge" deeper into the systems of complex organisms. Other ideas are offered by theoreticians like Barbieri (2014, 2019, along with Vega 2018). They end up arguing for a mechanical explanation of life and others have urged quantum complexities in an effort to make sense of the fractal like patterns within patterns and loops within loops that seem to connect things in ways that defy linear thinking (Rajagopal et al. 1999; Salari et al. 2011; Ho 2011, 2012; Li, Walker, and Michaelides 2011; Maleeh 2015; Rogers 2016; Montagnier et al. 2017; Hatano and Ordonez 2019). These last several theories seem to deny any edge or boundary separating living from non-living matter (Krylov 2017). However, one thing

that seems to be agreed upon is that dynamic interpretations of meanings expressed in biosignaling, alias biosemiotic, systems manifested in genomes, proteins, organelles, cells, tissues, and whole organ systems nonetheless remain essential to life and entail an orderliness that seems to reach out and touch, if not embrace, the whole cosmos. If it is supposed that there is a continuum from inert matter merging with viruses at a limit of minimal organization, what would we find at the opposite end of such a continuum?

The Highest Known Level of Symbolic Organization

In the biosphere it is plain to see that there is no form of organized representation that rises above the complexity of human discourse as manifested by the human language capacity. It is that capacity which enables us to probe the mysteries of our own lives and the universe in which we find ourselves (Chomsky 2011; Berwick and Chomsky 2017a, 2017b). Approaching the problem of life from that highest perspective — a "linguistic" or "discursive" perspective — we can define a kind of "gold standard" of the immensely complex systems by which living organisms, from microbes to human beings, wax and wane with respect to the much desired well-being that we refer to generically as "life". The human language capacity led Berwick and Chomsky to pose a deceptively simple question: *Why Only Us?* Why is the language capacity with its amazing complexities enabling us to probe the mysteries of life limited to human beings? Likewise, how are the complexities of that highly abstract system of systems somehow embedded in our genome?

At the center of the well-formed representations upon which viable living systems depend are the unique logical systems known as true narrative representations (TNRs). Such well-formed dynamic systems have three essential components each of which represents and virtually contains the whole: TNRs have the sort of relation to each other that each of the parts invariably represents the whole. It was this fact, evidently, like the persons of the Trinity in Christian theology that led C. S. Peirce to his various conceptions of what he called firstness, secondness, and thirdness (Peirce 1908; Oller 1984; Oller and Collins 2000; Robinson 2010; Slater 2013). It was Peirce, also, who showed more clearly than any other mathematician why Euclid's fifth "common notion" that the whole must be greater than the part is false because, as in TNRs, the part may be equal to the whole. In well-formed TNRs we always find three parts: (1) at the most abstract level, there is a string of symbols manifested in some material way (like the base-pairs of DNA, or the amino acids of a protein, the words of a true story, and so forth); (2) in the middle connecting the other two components of the TNR there are indexical lines in a tensional equilibrium connecting the abstract string of symbols to the concrete complex of facts referred to by that string; and (3) there must be a complex arrangement of real material facts that are faithfully represented by the manifested string of abstract symbols with which those particular facts are connected through the indexes that link them to the abstract symbols of the TNR. Among the unique logical properties that are only found in TNRs, and in no less well-formed representations are the power to determine the facts they represent, to be well connected to the facts they represent, and to be generalizable to all similar facts exactly to the extent of their similarity with the facts of the TNR at hand.

Ordinary Truth: An Abstract, Spiritual Foundation

The edge of life may be a boundary of great uncertainty, but it has been demonstrated with algebraic certainty (Peirce 1897; Tarski 1941, 1949; Oller 2014, 2020) that at the pinnacle of successful

communications of any kind — and, therefore, of the life, health, and well-being of living organisms in general — we find valid TNRs connecting manifest (real) sequences of abstract symbols with and through concrete complexes of material facts in the real world. The logicomathematical proofs of Peirce and others show that all fictions, errors, lies, and even nonsense strings depend for whatever semblance of meaning they may have on the extent of their resemblance to known TNRs. That is to say, all of the less interpretable sequences of symbols that may be constructed in any language, or in any language-like system whatsoever, must be (are mathematically proved to be) parasitic. Another way of summing up the argument is to say that to the less well-formed representations (fictions, errors, lies, and nonsense of varying degrees) depend for whatever meaning they may acquire on their resemblance to well-formed TNRs. From such a standard, it is possible to refine the meaning of Pasteur's "attenuation" in terms of the degree of departure of the derived form from its starting point.

Thus, it follows that the center of living systems is not to be found in the parasitic entities at the edges, nor in the many boundaries of the containers of containers that define living organisms as systems of systems (Oller and Shaw 2019). On the contrary, it seems that the center of organization of any any living thing can only be found in the enormously complex but well balanced TNR that is dynamically adjusted on the fly as that organism maintains the validity of its biosignaling systems over time. We can say that a coherent biosignaling system of systems must reside at the center of every healthy living organism, or we can turn the proposition the other way around and say that health and well-being are defined by a coherent system of biosignaling systems. The empirical demonstration of the existence of such a living system of systems consisting of TNRs at their basis is the fact that disruptions of those systems universally trend toward disorders, diseases, and the inevitable catastrophic failure known as death — an outcome, sad to say, which is guaranteed by the cumulative effect of disruptions of the biosignaling systems of every living thing (Oller 2010; Gryder, Nelson, and Shepard 2013; Oller 2014b). Life, therefore, dependent as it must be on TNRs, as I have argued (Oller 2010) is the logical antithesis of entropy. Or, putting the case in the opposite form, the antithesis of life, the kind of organization found in TNRs, is the sort of attenuation, decay, degeneration, and so forth, that leads ultimately to the complete dissolution of organization which can be thought of as death.

Amplifying the Concept of "Attenuation"

With the just stated logicomathematical basis in mind a more explicit articulation of what is meant by Pasteur's "attenuation" can be offered along with a simple articulation of experimental ways to assess and measure the degree of attenuation achieved in specific instances: *the attenuation of a known disease causing agent can be judged (measured) in principle (theoretically) by the reduction in its resemblance to its own formerly virulent genome (its former self), and in practice (experimentally) by the extent of reduction in virulence (after attenuation) with respect to organisms known to be susceptible of infection by that pathogen.*

The reasoning underlying this amplification of the concept of attenuation, as first clearly articulated by Pasteur (Pasteur 1881a, 1881b), is grounded in proofs developed in 2014 showing that any given TNR of whatever complexity it may possess, all else being held equal, can be successively "attenuated" (Peirce would have used the logical term "degenerated"; Peirce 1877) to produce one or many (1) fictions, by reducing one or more of the material elements of the starting TNR to turn it into a less well-formed representation where some part or parts of the material facts represented, are

not real, and must be imagined; (2) errors, by accidentally replacing some fictional element with a material or abstract element making that part of the representation false; and (3) lies, by deliberately (as in human discourse) or accidentally (as in the hypothetical progress of evolution) polishing up the false parts of an error so as to make it better resemble a TNR in order that it might be mistaken for one. We can take a further step of degenerating the surface form of any meaningful string of symbols by scrambling them or otherwise damaging them so as to reduce them bit by bit toward randomness.

One of the virtues of the logicomathematical proofs in which the theory just articulated is grounded, is the fact that the transitivity of the progression still holds if the starting point, say the genome of a bacterium (such as *Anthracis bacillus*, *Vibrio cholerae*, or whatever) or any virus (say, rabies, cholera, or smallpox), is construed as an "error" or as a deliberate destructive deception (a "lie") to begin with. According to the Nobel Prize winning research of David Baltimore (Huang and Baltimore 1970; Baltimore 1975), the replication of viruses by commandeering the replication systems of their hosts involves a lot of mistakes, errors where bits and pieces of the original virus end up in the cytoplasm of the host. The whole history of viruses, according to Baltimore and mainstream theoreticians, is a progression that theoretically sorts through a multitude of errors, making functional sense of some of them. Bearing in mind that an average-sized bacterium can contain hundreds of average-sized viruses (Oller 2020:94), it is little wonder that disease causing viruses were more difficult to discover than pathogenic bacteria, and it is also unsurprising that some viruses are much more likely to be transmitted through the air in multitudinous ways. Their much smaller size also enables viruses in general to multiply faster than the much larger bacteria that commonly contain and transport them (Guerin and Hill 2020). Whereas the Baltimore method of classifying viruses according to their means of replicating or transforming themselves by combining their genomic identity with some other virus, seemed to be the industry standard for a while, recent work with "structure-based analysis" superimposing viral capsid proteins one on another suggests a simplification from as many as seven or even 15 ranks (International Committee on Taxonomy of Viruses Executive Committee 2020) down to as few as four distinct classes according to Ravantti, Martinez-Castillo, and Abrescia (2020). The authors of the latter study, however, acknowledge "the difficulty of establishing structural 'self' traits for enveloped viruses" notably including the "coronaviruses" and especially SARS-CoV-2.

Regardless, no matter the complexity of the starting composite of "self traits" that might be associated with a given viral pathogen, the mathematical proof showing that all meaningful strings of signs in any language or language-like system whatever, must derive their meaning from TNRs also proves conclusively that the details of any fiction, error, lie, or even chaos of any degree of complexity can be represented in TNRs, but the reverse does not follow. Jaynes (1963) showed that his principle of "maximum entropy" in irreversible processes such as the kind of recombinant experimentation GOF researchers engage in on a regular basis, mathematically guarantees the transitivity of Pasteur's concept of attenuation, or Peirce's logical degeneration, even if applied to the corrupt and degenerate form of an error or a deliberate lie as a starting point. Baltimore's research with corrupted composite of "self traits", as seen in the multitudinous fragments of a virus that are about as apt to be replicated as the original virus, show the meaning of "attenuation" in the case of a recombinant viral pathogen. The pragmatic force of the progression from any given TNR to derived fictions, errors, or lies must also hold for the progression from any selected pathogen to attenuated

variations of it. The theoretical shape (say the genome) of a given pathogen (bacterium or virus) and the infective powers of such an entity are just as subject to the transitivity of attenuation (degeneration) as are any of the other categories of representations that can be derived from TNRs.

GOF Research Is a Game Changer

Following the line of Baltimore's thought about viral replication and its progression across time, it is not entirely irrational to suppose that the accidents of biochemical history can result in fragments of DNA, RNA, and proteins that may quite by chance, over fairly long periods of time, sort themselves into an array of infective pathogens consisting of viruses or even prions (Diener, McKinley, and Prusiner 1982; Prusiner et al. 1982, 1983; Scheckel and Aguzzi 2018). However, when intelligent researchers introduce deliberate experimental manipulations aiming to exploit suspected or known weaknesses of mammalian or human immune defenses against known PPPs in order to cause them to gain in virulence, transmissibility, or to leap across barriers from one species to another increasing the range of hosts infected, inoculation of potential hosts with such beefed up PPPs introduces a far greater likelihood that some one of these "enhanced" pathogens will produce a real pandemic.

As even the proponents of GOF research are quick to admit (Casadevall and Imperiale 2014; Imperiale and Casadevall 2020), there are two classes of uncertainty: on the one hand there is the risk of an inadvertent release of a real PPP similar, for instance, to the SARS-CoV-2 virus of the SARS-CoV-2, and, on the other hand, there is the virtual certainty that nefarious powers aiming to develop offensive bioweapons will be at pains to gain control of the products of GOF research. Meantime, decades of research with influenza vaccines have not produced any notable success in developing effective vaccines to prevent influenza or the common cold. Because of the demonstrated instability of viruses themselves (Huang and Baltimore 1970, et. seq.), and of the vaccines manufactured to emulate and thus help immune systems to defeat them (Kumru et al. 2014), developing stable forms of intrinsically unstable viruses to be delivered in some kind of "attenuated" (less potent) form in a vaccine is a risky enough proposition all by itself.

Is the Reduction in Virulence Adequate in Influenza Vaccines?

Very often, as Eaton et al. (2018) demonstrated (inadvertently, see my discussion of their results; Oller 2020:285–86), the instability factor combined with adjuvants such as aluminum hydroxide (Santiago et al. 2015), evidently sets up anti-viral vaccines such as the monovalent H1N1 influenza vaccine and the trivalent influenza vaccine (TIV) to bring about instabilities in the recipient. For the pregnant women studied by Eaton et al. (2018), either of the vaccines, H1N1 or TIV, on the average, were associated with developmental irregularities ranging from "preterm birth" to "congenital anomalies" and "still birth" (death of the baby) in 71.19 and 67.93 cases per 1,000 administrations, respectively (Oller 2020:285). With such vaccines, the objective of research and development is to achieve sufficient stability to lessen the risk of infection and harm from the pathogen. It is, in the case of influenza vaccines, doubtful whether the the risks to unborn babies and their mothers are lessened or increased by the vaccines.

Unfortunately, Eaton et al. did not compare data for healthy pregnant women receiving neither of the vaccines against the results for those who did get vaccinated. The fact that such a comparison is the obvious basis for making the judgment call as to whether or not a vaccine is safe or effective is

plain in Pasteur's experiments with animals in developing vaccines for Anthrax, cholera, and rabies. He systematically compared vaccinated cases against unvaccinated. Setting aside the fact that current vaccine research almost universally compares one vaccine against another — as Eaton et al. did with H1N1 compared against TIV — and avoids any comparison of vaccinated against unvaccinated persons (E. P. I. C. Magazine 2017; Habakus, Holland, and Rosenberg 2011; Olmsted, Blaxill, and Kirby 2011; Miller and Blaylock 2017) on the ground that it would be unethical to permit, much less encourage, anyone not to be vaccinated, it is notable that the FDA sees no problem in administering dangerous "challenges" (US Food and Drug Administration 2019:5–6) in the form of untested vaccines to human volunteers. If human beings are judged to have enough sense to take a voluntary risk that might inform researchers about the impact of an inadequately tested vaccine, why is the reverse scenario, volunteering to reject vaccination, judged to be unethical?

What If the Aim Is to Increase the Virulence of the Pathogen?

Setting aside the earlier question pertaining to the government's efforts to promote the creation of prophylactic procedures (especially, vaccines) to lessen the threat of pandemic disease from airborne viral pathogens, what about the risks of research aiming to increase the likelihood that some dangerous pathogen can be turned into a real pandemic disease? Proponents may deny that the goal of GOF research is to increase the risks euphemistically referred to as "biosafety" and "biosecurity" (Casadevall and Imperiale 2014:2), but the definition of GOF research ensures that outcome. There is, on the one hand, no guarantee that anything good (any benefit) will come of GOF research, but it is a lead pipe cinch that GOF research increases the risks of producing something very bad — namely something exactly like the SARS-CoV-2. The bio-risk euphemized as "biosafety" is the intensification, weaponization, of dangerous pathogens that is necessary to qualify research as the GOF kind, and the bio-risk dressed up as "biosecurity" is that conducting and publishing GOF research is an open invitation to evil-doers to figure out some way to gain access to one or more weaponized pathogens, or to learn how to create them on their own hook. More importantly, if we have learned nothing else from the recent SARS-CoV-2, government protections for the public are totally inadequate in both categories.

Laboratory Accidents

Documented incidents of accidental and deliberate releases of weaponized pathogens (N. G. Evans et al. 2015; Silver 2015; N. G. Evans 2018) from level 3 and 4 laboratories is where this whole public discussion began to capture public interest. Now, with the SARS-CoV-2, the whole world is interested in the largely unenforceable regulations in place for the most advanced GOF research laboratories in the USA and the world (Centers for Disease Control and Prevention National Institutes of Health 2020). The fact is that levels 3 and 4 laboratories (Jahrling et al. 2009; Risi et al. 2010; Günther et al. 2011) have not achieved the safety and security record that should be demanded by the public if GOF research is to be permitted anywhere on the globe. A malingering question that seems likely to be answered definitively as the greatest lapse of biosafety in world history is whether the virus credited with the causation of the SARS-CoV-2, attributed to SARS-CoV-2, was owed to an accidental release or a deliberate release of a manipulated (laboratory intensified) variant of SARS-CoV (Zhan et al. 2020).

Digging up and Reviving the 1918 Influenza Virus

The CDC (Centers for Disease Control and Prevention 2019e) and related resources (Scull 2020) identify four modern and well-documented "pandemics" preceding the one we are calling SARS-CoV-2. All of them were attributed to airborne influenza viruses — H1N1 (the "Spanish flu if 1918), H2N2 (the "Asian flu" of 1957), H3N2 (an unnamed 1968 epidemic), and another H1N1 (epidemic of 2009). All of these and other airborne viruses, in recent years, have been the focus of genetic engineering in GOF research laboratories. Curiously, the first and probably the most deadly epidemic in modern history was the 1918 influenza epidemic attributed to H1N1. It is known to have killed about 17.4 million persons based on records examined by Spreeuwenberg, Kroneman, and Paget (2018) although the Centers for Disease Control and Prevention (2019a) commonly multiply that number up to five times suggesting that the H1N1 virus may have killed as many as 40 to 100 million (Taubenberger, Reid, and Fanning 2000; Hagemann 2020).

Reconstructing the 1918 Virus to Develop Measures Against Future Pandemics?

The H1N1 virus believed to have been the primary cause of the 1918 epidemic (though most of the deaths were caused by secondary bacterial infection, according to Taubenberger et al. 2000), was literally dug up by Johan Hultin in 1997 (see Jordan, Tumpey, and Jester 2019). The virus was extracted in fragments from the lungs of a victim buried in November of 1918 beneath the Alaskan permafrost (Taubenberger et al. 1997). In the year 2000, GOF researchers at the Armed Forces Institute of Pathology in Washington, DC stressed the importance of receptor binding between the proteins of the host and the H1N1 virus (Taubenberger et al. 2000:242). By 2005 the putative deadliest influenza virus ever known, H1N1 of 1918, was fully reassembled by genetic engineering thanks to funding from the National Institutes of Health and the Armed Forces Institute of Pathology (Taubenberger et al. 2005:893). The public justification for this re-assembly was "to protect public health and to develop measures effective against future influenza pandemics" (Taubenberger et al. 2005:80). It did not take long for the next so-called "pandemic" to come along, and it just happened to be named the H1N1 flu of 2009 (Jordan et al. 2019).

Was it merely a coincidence that the "pandemic" proclaimed in 2009 by the CDC occurred just a few years after the reconstruction of the 1918 virus? Was there any connection with the H1N1 virus manufactured by the CDC and FDA using "reverse genetics" to create a "candidate vaccine" that planned in advance to "be sent out to manufacturers" so they could begin to produce "pilot lots of vaccine" at a cost of "about $1 billion for clinical studies of vaccine pilot laws and for commercial scale production of potential ingredients for a pre-pandemic influenza stockpile" (CDC Newsroom 2009)? Was there just an amazing series of lucky coincidences for the manufacturers of vaccines, or did the research involved in the reconstruction of the 1918 H1N1 virus have anything to do with the H1N1 "pandemic" of 2009? Latham, with a PhD in virology and a post-doc in genetics at the University of Wisconsin, Madison, and Wilson with a doctorate in molecular biology and genetics from Indiana University, surmised that

> the most coherent explanation for the H1N1 variant "swine flu" pandemic of 2009/10 that resulted in a death toll estimated by some as high as 200,000 (Duggal et al., 2016; Simonsen et al. 2013), is that a vaccine was improperly inactivated by its maker (Gibbs et al., 2009). If so, H1N1 emerged from a lab not once but twice.

Skipping over the lesser "pandemics" of H2N2 in 1957 (Asian flu beginning in Singapore, Centers for Disease Control and Prevention 2019b) and H3N2 in 1968 (bird flu beginning in the US, Centers for Disease Control and Prevention 2019c), the very return to the H1N1 nomenclature for the 2009 pandemic by the Centers for Disease Control and Prevention (2019d) is interesting. The series of events bringing about a billion dollars to vaccine manufacturers in 2009 is even more telling in view of the fact that GOF scientists had been seeking to intensify the H1N1 reconstruction at least from about 2004 (Stevens et al. 2004; Sun et al. 2010), along with H5N1 (Evans 2013; Fouchier et al. 2013; Tian and Zheng 2015), H7N9 (Fouchier et al. 2013; Tian and Zheng 2015), as well as corona viruses (Casadevall and Imperiale 2014; Imperiale and Casadevall 2020).

The Ubiquitous "Spike" Protein

Critical systems focused on by GOF researchers in PPP viruses of interest are those involved in the penetration of the host cell "mediated by a spike protein present in the virus envelope . . . In the case of influenza virus, the viral spike protein hemagglutinin (HA) mediates both receptor binding and membrane fusion" (Sun et al. 2010). By 2012 GOF researchers engineered an avian virus H5N1 to leap across from birds and infect mammals (Herfst et al. 2012; Imai et al. 2012). In August of 2013, a group of 22 scientists based in the Netherlands, the USA, Hong Kong, and the UK, wrote a letter to the editor of *Science* (Fouchier et al. 2013) concerning an avian influenza H7N9 infecting people in China. They claimed that the virus had accounted for 43 deaths in 130 cases of human infections. On that basis they argued in their letter that

> The A(H7N9) virus hemagglutinin protein has several motifs that are characteristic of of mammalian-adapted and human influenza viruses, including mutations that confer human-type receptor-binding and enhanced virus replication in mammals. The pandemic risk rises exponentially should these viruses acquire the ability to transmit readily among humans. . . . [Because] classical epidemiological tracking does not give public-health authorities the time they need to mount an effective response to mitigate the effects of a pandemic virus. To provide information that can assist surveillance activities — thus enabling appropriate public-health preparations to be initiated before a pandemic — experiments that may result in GOF are critical. . . . (Fouchier et al. 2013:612).

The authors proposed GOF research to "determine whether genetic changes [engineered by GOF researchers] that confer altered virulence [greater infectivity], host range [jumping from chickens, to ferrets, or, from ferrets, mice, or bats to humans] or transmissibility also change antigenicity [the power of the virus to resist or defeat the host's defenses]" (Fouchier et al. 2013:612). They spelled out their intention to use GOF research specifically to explore the "hemagglutinin cleavage site, that would enable circulating A(H7N9) viruses to become more pathogenic" (Fouchier et al. 2013:612). Focus on that kind of cleavage site had been associated with pathogenicity and the power of the virus to bind to host a receptor was also done with H7N1 (Sutton et al. 2014). Meantime work was also progressing to develop PPPs (bioweapons) from SARS and other lethal airborne corona viruses (Fouchier et al. 2013; Evans et al. 2015; Board on Life Sciences et al. 2016; Fears and ter Meulen 2016; Frank et al. 2016; National Science Advisory Board for Biosecurity 2016; Selgelid 2016; N. G. Evans 2018; Spieler et al. 2020). Perhaps the most disturbing aspect of that research was the hardly concealed collaboration between militarists in the Chinese Communist Party and GOF research proponents, notably the influential Anthony Faucci, insisting that the NIH and its international partners were all working together to prevent, not to produce, the weaponization of the PPPs under experimental investigation by GOF scientists (Breggin and Breggin 2020).

Is the Source of the SARS-CoV-2 Virus Discoverable?

The critical question addressed in this paper is whether the virus identified as the source of the COVID-19 epidemic, the SARS-CoV-2 virus, was (1) a product of nature, like a little green apple that grows in a tree, or (2) was it a man-made variation on something natural, like an apple pie cooked up in a bakery. Is SARS-CoV-2 an ordinary natural virus that just happened to make the leap to human beings, or was it a virus assisted to make the leap to humans through genetic engineering in one or more bioweapons laboratories. Attention has focused on "key interactions between SARS-CoV spike protein and its host receptor angiotensin-converting enzyme 2 (ACE2), which regulate both the cross-species and human-to-human transmissions of SARS-CoV" (Wan et al. 2020:e00127-20). The focus on that spike protein in GOF research and in vaccine development to try to counter-act the impact of SARS-CoV-2 in the COVID-19 epidemic, as Sørensen, Susrud, and Dalgleish 2020) warn, unless is etiology is taken into account, could cause more harm than good by "including the risk of antibody-dependent enhancement" whereby the virus attaches itself to inefficient or defective antibodies in a way that increases its power to invade healthy cells (Sørensen et al. 2020). To stress two points: the ongoing frantic vaccine research in response to COVID-19 shows the inevitable relation between the costly manufacture and distribution of vaccines as being motivated and facilitated by the GOF research aiming to create PPPs in the first place. If SARS-CoV-2 is a manufactured bioweapon, as many have suspected and as some are aiming to demonstrate empirically, it is a huge experimental proof of concept of the historical connection between modern bioweapons research and vaccine development.

Natural or Manufactured?

The public discussions coming from GOF research advocates suggested several possible intermediate animal species that might have facilitated the infection of the first human thus initiating the incubation and proliferation of SARS-CoV-2 leading to the COVID-19 epidemic. In defense of GOF research, and consistent with the virtually homogeneous mainstream publications on the "proximal origin" of SARS-CoV-2, Andersen et al. (2020), published in March 2020 (cited 652 times in the Web of Science database, and 1946 times according to Google Scholar, by December 1, 2020), concluded:

> Although the evidence shows that SARSCoV-2 is not a purposefully manipulated virus, it is currently impossible to prove or disprove the other [natural animal origin] theories (p. 452).

However, there is a logical problem with that claim: it asserts an empirical proof of a completely generally proposition — namely, that no demonstrable connection between SARS-CoV-2 and any intentional bioweapons research will ever be found. Further it is a self-refuting proposition: it asserts that no conclusive trail from a "previously used backbone virus" (p. 450) could "prove or disprove" (p. 452) what they claim to have already proved — that SARS-CoV-2 cannot possibly have been humanly constructed from some prior animal virus.

Their claim is an overgeneralization on both accounts and it is a self-refuting claim with respect to what they say is impossible to either prove or disprove. On the one hand, the 2020 argument of Andersen et al. it is like climbing several apple trees in search of apple pie, finding none, and claiming therefore to have proved that no apple pie exists. No number of failed searches for evidence that the genome of SARS-CoV-2 was a GOF construction can prove that it was not such a

construction. To pursue the analogy, finding an apple pie growing spontaneously in an apple tree would go a long way toward disproving the theory that apple pies do not grow on trees, whereas finding an apple pie in the oven at the bakery — say, at Wuhan laboratory aiming to convert corona viruses into PPPs — would pretty much demolish the natural origins theory that apple pies do not involve any participation from human bakers. Whereas any number of failed searches can never prove that what we may be looking for does not exist, just one successful search can show the opposite.

Cutting to the Chase

With the foregoing in mind, skipping over the hundreds of publications accepting the claim that SARS-CoV-2 could not possibly be a manufactured result of GOF researchers, a great deal of effort has ensued trying to show that SARS-CoV-2 must have originated from the horseshoe bat (Zhou et al. 2020), with a possible stop over in a snake, a civet, or a pangolin (Piplani et al. 2020). Attention was famously directed away from the Wuhan bioweapons laboratories to an outdoor meat market in Wuhan near the epicenter of the "outbreak" of COVID-19 where SARS-CoV-2 might have made its leap to humans. It was speculated that the first human infections might have occurred when buyers and sellers exchanged the meat of just the right SARS-CoV infected reservoir. However, as Latham and Wilson (2020) point out, citing the authoritative *Lancet* article by Huang et al. (2020), the very first case and 13 others among the earliest 41 cases that were infected had no contact with the Wuhan meat market. The fact that Wuhan is the epicenter of bioweapons research with coronaviruses harvested mainly from bats — combined with the fact that bat meat was not for sale at the market — led some researchers to suggest that SARS-CoV-2 may have been an engineered product of the bioweapons research with bat viruses going on in the Chinese Communist Party's military research in one of the Wuhan laboratories (Zhan et al. 2020).

Definitive Research?

While it may not be possible to rule out all of the conceivable ways that SARS-CoV-2 might have been created in a bioweapons laboratory from some prior corona virus, it may be feasible by making some invidious comparisons between proposed parent viruses and the gaps they would have to cross to complete the transformation from their starting genome to the known genome of SARS-CoV-2. Every researcher concerned with the etiology of SARS-CoV-2 has been considering routes by which some particular corona virus from a bat, especially the *Rhinolophus affinis* bat, according to Andersen et al. (2020), the source of RaTG13 which they claimed to be "~96% identical overall to SARS-CoV-2" (p. 450). That particular corona virus, with a genomic sequence "first submitted to *GendBank* on January 27th, 2020" (Yan et al. 2020a:3), however, has an interesting history going back to 2013. It was called "RaTG13" where the "Ra-" portion was taken from the *Rhinolophus affinis* bat, the "-TG-" from the location where it was discovered near "Tongguan" in the Yunnan Province extracted from one or more of three miners who died with COVID-19 like symptoms in 2013, thus completing the designation with the number "-13" commemorating the year of the supposed discovery of the RaTG13 virus (Swarajya Staff 2020). Strangely, as Yan points out, the discovery of the corona virus in question was not reported for seven years by China's famous "bat-woman" Zhengli Shi and her team at the Wuhan Institute of Virology (Hu et al. 2020) and despite its being credited with the deaths of half of the Mojiang miners infected by it in 2012 was not sequenced until

2018 (Cohen 2020; Yan et al. 2020a:5). Meanwhile, it was, along with other coronaviruses the subject of intensive GOF research for the development of PPPs at the Wuhan Institute of Virology (Daszak 2014-2019; Menachery et al. 2015; Breggin and Breggin 2020; Guterl 2020; Lin 2020). Moreover, certain US universities, notably the Galveston National Lab, University of Texas Medical Branch and Department of Epidemiology, University of North Carolina at Chapel Hill, teamed up with Zhengli Shi and her team at the Wuhan Institute of Virology to help them in their GOF/bioweapons research with PPPs, focusing specifically on coronaviruses.

To come to the bottom line, Yan et al. (2020) have produced persuasive evidence, perhaps sufficient for a legally probative case (though not a strict logical proof), that the Mojiang Mine theory proposed by Latham and Wilson (2020a) is grounded in a fictional viral genome, specifically the recently "sequenced" RaTG13 that Yan et al. believe was invented by Zhengli Shi on her laptop and then submitted to *GenBank* on January 27, 2020. Moreover, the corona viruses supposedly extracted from Malayan pangolins, and from bats, ones unnaturally similar in excessively improbable ways to RaTG13, are also very likely to be either laboratory creations, or fakes created to direct attention away from the growing conviction, plausible if not definitively demonstrated, that SARS-CoV-2 is a humanly manufactured bioweapon that either was deliberately or accidentally released from the Wuhan Institute of Virology. After that occurrence, which Yan et al. assert was deliberate, the plot thickens in a way that suggests very plainly that the Chinese Communist Party and the militarists at the Institute, headed up from January 2020 by China's bioweapon's expert Major General Chen Wei of the People's Liberation Army (Thomson 2020), decided to cut their own internal losses while performing an unrestricted biological warfare experiment of the rest of the world: they systematically restricted travel to and from Wuhan within China (covered up the epidemic occurring there and all of the research on the SARS-CoV-2 causative agent), while encouraging as much international traffic as possible to the rest of the world (Yan et al. 2020a:26–28).

Summary

Bioweapons research and development in the free world has been linked closely to vaccine research dating back at least to the creation of the the War Research Service in the United States in 1942. Prior to that time, it is true that vaccine research enjoyed the reputation of being "for the greater good" — with risks that seemed worth taking on the theory that worse things could be avoided. In the latter part of the 19th century, Louis Pasteur became an international celebrity of great renown for his work in demonstrating that some deadly pathogens could be killed with heat, and that ones as lethal as Anthrax, cholera, and rabies could be weakened, "attenuated", by exposing them to oxygen or heat. On the basis of the supposition that cowpox (*Vaccinia*) is regarded by the body as a weaker version of smallpox (*Variola*) — on account of the supposed resemblance of one pathogen to the other — Pasteur inferred that animals and humans inoculated with weakened versions of any pathogen could become immune to them. As Christopher A. Shaw has suggested (personal communication), by such logic, we could argue that the purpose of a vaccine is to trick the human immune defenses into treating the attenuated pathogen as if it were the real thing. In this way, vaccinologists are inadvertently producing an experimental proof of concept of TNR-theory with every inoculation: for the deception to work at all, as the logical theory requires, the attenuated (substitute) pathogen, must resemble the real thing. The fact that the resemblance can never be perfect except in the case of the natural pathogen representing itself, can be hypothesized to account

for the relatively shorter lived resistance engendered by vaccines as contrasted with naturally occurring pathogens.

Pasteur also did something that is not required in the current climate of frantic, catch-up vaccine development running behind GOF research: he systematically compared vaccinated against unvaccinated animals to experimentally assess the efficacy and safety of the vaccine he was developing. The inoculation of Joseph Meister after he was bitten by a rabid dog was Pasteur's most sensational success though it may have worked in part by not only exposing the child to successively more virulent forms of the virus but also exposing him to antibodies effective against rabies coming from the formerly infected laboratory animals. That question aside, immunoglobulin capable of binding and possibly destroying a pathogen, is almost certain to be safer than deliberate exposure to the pathogen itself. Also, immunoglobulins in general would, according to current research (Akazawa-Ogawa, Nagai, and Hagihara 2018), retain their integrity under the drying out protocol for attenuation used by Pasteur.

All that being said, with respect to GOF research, it seems that Pasteur's careful reasoning and his ethical concern for the lives of animals and human beings, have fallen by the wayside as vaccine developers frantically chase after the PPPs being cranked out and released, whether by accident or on purpose, from bioweapons research laboratories. Does it really make sense to maximize the virulence, transmissibility, and host range of the most dangerous pathogens in the biosphere in the hope that doing so will enable the well-intentioned researchers of the world to outsmart the evil ones and also to outsmart themselves by figuring out how to contain the monsters they are helping to create?

Competing Interests

The author declares no competing interests.

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