Open Letter to the U.S. Intelligence Committee & Scientific Community

COVID-19 is a Covert CCR5 Gene Silencing Experiment using a Genetically Engineered Virus Containing Decoy Parts

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The scientific community and the world at large have been misled about the origins of SARS-CoV-2, its design as it relates to its primary mode of action, and the reasons behind the intentional leak of this virus on humanity. This pandemic has killed roughly 3 million people worldwide and has compromised the health, safety, and livelihood of all of humanity. Confronting the truths of this matter is both urgent and essential, and so, despite my desire to present a full research article on this matter (which will be presented soon, God willing), I write this letter to call the scientific community and all agencies investigating the origin of SARS-CoV-2, to consider my research findings, and further, search for the truthfulness of these findings during the investigative process.

I have written extensively about these topics which has been published in a research paper and a book (1-2). For brevity, I will pull excerpts from both as well as express new points which I will elaborate on in the forthcoming research.

Background:

- In 2008, a letter was published in Nature discussing the global trends in emerging infectious disease (EID). Under the direction of Peter Daszak, President of the Eco. Health Alliance, the researchers analyzed a database of 335 EID events which occurred between 1940-2004. Their research showed emerging diseases mainly consisted of those that originate in wildlife and bacteria. They also noted a correlation between the rise in EID events and the HIV pandemic and attributed this to socio-economic, environmental, and ecological factors. *They further noted that EIDs place a huge burden on global economics and public health and that methods need to be employed in order to control EID to prevent global economic collapse and widespread disease* (3).

- Four years later in 2012, Jennifer Doudna and her colleague, Emmanuelle Charpentier, created CRISPR-Cas9 gene editing technology, in which scientists all over the world would attempt to create useful applications for.

- One year later, RaTG13, the virus which laid the basis for the natural origin theory of SARS-CoV-2, was said to be discovered in 2013 (4), though a structural and biochemical analysis has since proven that a bat virus like RaTG13 would not bind effectively to human ACE2 receptors (5).

- In 2017, Scientists at Harvard University applied for a patent which would cause a "loss of function" to CCR5 co-receptor gene to protect against HIV infection using CRISPR-Cas9 technology (6).

- Luc Montagnier, the virologist who co-discovered HIV, stated via *European Scientist* that SARS-CoV-2 contains elements of HIV and Malaria in genome and that it (SARS-CoV-2) may have been made in an effort to develop a vaccine for AIDS.

Assertion & Evidence:

- SARS-CoV-2 is a genetically engineered virus:

    - Possibly made using templates and as explained in the second Yan et al. report, and I concur with that report regarding the engineering of S2 region of SARS-CoV-2 (7).
    - The engineering possibly included the sequencing of HP1/S2 bacteriophage of Nontypeable Hemophilus Influenzae (NTHI) (8).
    - Previous research from 1958 has demonstrated how to change an organisms' genome through bacteriophages using the hosts own opportunistic bacteria such as NTHI (9).
        - **Why target NTHi?**
            - SARS-CoV-2 (a temperate bacteriophage) will not completely destroy the cell wall of NTHi, a gram-negative bacteria. This allows NTHi to continue function as NTHi and SARS-CoV-2 become a lysogen, allowing SARS-CoV-2 to replicate, as SARS-CoV-2 becomes a prophage (2).
            - mRNA COVID-19 vaccines can disguise prophage inducing chemicals as seemingly harmless ingredients (i.e., salts, chloride, lipids) (2).
            - Hemophilus influenza was used in the discovery of restriction enzymes (enzymes that can recognize and cut specific sequences of DNA) it was also sequenced during the Human Genome Project (10).
            - Additionally, it has been found that genes encoding high molecular weight adhesion proteins of NTHi are part of important gene clusters, specifically Hmw1 and Hmw2 genes which encode B ORFs and C ORFs of NTHi. A study of bacteriophage HP2 of Hemophilus influenza has demonstrated the ability of prophages

to inhibit gene expression (11). These factors make NTHi an easy and viable target for a covert gene editing experiment.

- **RBM Decoy:** Tampering done to the receptor binding motif (RBM) region of S1/spike to lead scientists to think virus was optimized for ACE2 receptors.
  - Recent research has shown that the furin cleavage of SARS-CoV-2 is not needed for infection (12).
  - Another research has pointed out that SARS-CoV-2 has dual action capability, in that it can bind to ACE2 receptors as well as act as a co-receptor dependent phagocyte (13).
  - ACE2 biding decoy used to distract attention away from ICAM-1 binding.
    - Recent research on the structure of SARS-CoV-2 shows that accessory protein ORF7a has structural homology to ICAM-1, the receptor that becomes upregulated during an invasive NTHi infection (14).

The following contains excerpts from Chapter 6 of my book; "Informed Consent: Exposing the Eugenics Cult & their Latest Experiment on Mankind: COVID-19":

COVID-19: CRISPR-Cas9 *in vivo*

It is my belief that COVID-19 is an attempt to intervene in the process of natural selection through the simulation of plague-like conditions from the Middle Ages in order to trigger a CCR5 delta-32 mutation via a CRISPR-Cas9 gene editing system. The CCR5 delta-32 mutation makes its carriers resistant to diseases like HIV, Smallpox, The Bubonic Plague (Black Death) (15), and most likely COVID-19. As previously mentioned, the CCR5 delta-32 mutation is more prevalent in the Caucasian population as 1% of them have two copies of the mutation and up to 20% of them have at least one copy of the mutation. In the U.S., Canada and Australia, the frequency amongst Caucasians is 8% to 10%, but less than 1% in African American populations (16). Consequently, COVID-19 is particularly harmful to African Americans and other minority groups due to the low prevalence of the CCR5 delta-32 mutation and pre-existing conditions like anemia, diabetes, heart dis-ease, and cancer. There are many CRISPR-Cas9 CCR5 editing systems that exist, however there is one which was created by scientists at Harvard University that would (in my opinion) offer the most comprehensive and effective tools to carry out a large-scale experiment like COVID-19.

Harvard's CCR5 delta-32 Patent* (*Funded by the NIH)

On August 18, 2020, Harvard University was granted a patent for its CCR5 gene editing systems, by the United States Patent and Trademark Office. Their systems involve several methods and chemical compositions which can cause "loss-of-function" to CCR5. One of these systems suggest use of the CRISPR-Cas9 gene editing tool to cause a loss-of-function to CCR5

via mutation. According to the researchers, this may be accomplished by introducing the CRISPR-Cas9 system via **mRNA virus particles**. Further, in order to promote effective gene targeting, they also suggest combining bacteriophage Mu along with a mRNA fusion (6). Unsurprisingly, evolutionary genes to proteins in bacteriophage Mu are present in the bacterial genomes of Hemophilus influenzae. Bacteriophage Mu also plays a critical role in the DNA repair process.

DNA Repair via HDR

The CRISPR-Cas9 system often uses Homology Directed Repair (HDR) to make precise edits in the genome. HDR is a system within cells that allows it to recognize and repair double stranded DNA breaks. In order to utilize this system for gene editing, a product including gRNA, Cas9, and a DNA template for up and downstream genes must be introduced to the host. This way, the cell has exact instructions on how to repair the break. Keep in mind, these instructions may include add-ons or deletions of specific genes. Now, HDR is not always guaranteed and in fact, the efficiency rate is low, as less than 10% of alleles get modified using this method. To counteract low HDR, fusions may be added to the product which includes cytosine base editors (CBEs) and adenine base editors (ABEs). These base editors are essential in making very small yet, precise edits within the genome. If a scientist wanted to further ensure the purity and effectiveness of HDR, they might consider adding additional fusions to the product which would increase the product purity such as the DNA glycosylase inhibitor (UGI) or the bacteriophage Mu- derived Gam protein (Mu-GAM) (6).

In my research paper; “COVID-19: The Plague that Strikes Behind the Ear”, I identified the Mu-GAM, though I did not realize it at the time. In my paper, I described how a NTHi / COVID-19 co-infection would cause ICAM-1 to become upregulated. ICAM-1 then coordinates bonding between LFA-1 and CD4 T-Cells. LFA-1 activation causes the release of “Leukotoxin” or LtxA which is a protein secreted by the oral bacterium, Aggregatibacter acinomycetemcomitans. LtxA is the GAM protein of the bacteriophage Mu (6). This information provides more insight into the pathophysiology of a NTHi / COVID-19 co-infection. In this scenario, LtxA would increase the infectivity of CD4 T-cells which would grant COVID-19 more access to CCR5 co-receptors. LxtA is secreted as part of the immune response, however in this gene editing experiment, it would facilitate effective HDR through its ability to bind to the double strand breaks thereby protecting against the degradation of the DNA in the places where the edits are made. In this way, COVID-19 can use mechanisms and secretions elicited by the host’s adaptive immune system to not only facilitate easy access to the gene target(s), but it also uses these mechanisms to carry out the gene editing procedure. This would be thought of as brilliant to a mad scientist. However, to sane people, its just *evil*.

Oftentimes, the alteration of a network of genes or sequences is required in order to effectively alter the targeted gene. Harvard University’s patent suggests such alterations be made in “all possible PAM sequences” in the genome in order to generate CCR5 mutations making a NTHi a perfect target for this COVID-19 experiment due to the Pam3Cys. P6, a main feature of this system, is a dual orientation peptidoglycan-associated lipoprotein (PAL) which is part of the Tol-

Pal System. P6 has long been studied as a potential vaccine antigen. It is located on the outer membrane of NTHi and when triggered, it will activate the host's Toll-like receptor 2 (TLR2). The TRL2:P6 signaling axis responds to a NTHi infection by mounting an inflammatory response in which cytokine-secreting P6 specific T-cells (i.e. IFN-y ((Th1)) and IL-17 ((Th17))) are summoned giving COVID-19 access to CCR5 co-receptors. It is during this process that the Tol-Pal compounds can facilitate the penetration of bacteriophage Mu particles (from LtxA) into NTHi. During this process transcription can occur as Aggregatibacter acinomycetemcomitans contains genes encoding the Tol-Pal system – (ybgC – tolQ – tolR – tolA)(tolB-pal-ybgF). Transcription occurs from promoters located upstream of the 5' ends of genes ybgC and tolB. This DNA sequence or "PAM motif" as described within Harvard's patent, is required for Cas9-DNA interaction (6, 17-18).

sRNA Targets

During the inflammatory response, iron is sequestered as part of the nutritional immunity phase. In NTHi, the ferric uptake regulator (Fur) acts to regulate and balance iron levels throughout the body. Fur also can positively affect gene expression via a small RNA (sRNA) intermediate called HrrF. HrrF targets genes whose byproducts are involved in molybdate uptake, deoxyribonucleotide synthesis, and amino acid biosynthesis.

Orthologs, which are evolutionary genes of HrrF only exist in members of the Pasteurellaceae Family of which NTHi and Aggregatibacter acinomycetemcomitans are both members. It is critical to mention the unknown dangers of editing orthologs. These are genes which have been essentially passed down from our ancestors', generation after generation, and have retained the same function throughout that evolution. Alterations of these genes can have devastating, long-lasting effects and further, it can be fatal. HrrF target genes include the modABC gene cluster, asnA, asnC, and nrdB. Some of these orthologs encode major iron utilization systems including hitABC, hxuABC, hfeABCD, tbpA and tbpB, hgpB, hgpC, and hgpD. Fur regulated sRNAs in Aggregatibacter acinomycetemcomitans include JA01 though JA04 (19).

COVID-19: CRISPR-Cas9 In Vivo (Rudimentary Overview)

1. COVID-19: a genetically engineered bacteriophage of Nontypeable Haemophilus Influenzae (NTHi)- carrying gRNA (small interfering RNAs or siRNAs) enters the host.
2. NTHi identifies COVID-19 and attempts to destroy it through the upregulation of ICAM-1.
3. ICAM-1 acts as a ligand to LFA-1.
4. LFA-1 becomes active on CD4 T-cells.
5. LFA-1 activation on target and infected CD4 T-cells enhance COVID-19 infectivity and transmission by promoting virus binding and cell to cell spread via the CCR5 co-receptor. COVID-19 now has access to the CCR5 target DNA sequence within the NTHi bacterium.
6. gRNA binds to the target sequence.
7. Cas9 enzyme binds to gRNA.

8. Host is exposed to treatments;

- Vaccines
    - Sodium, sugars, phosphate, chloride bind to Cas9 causing it to activate.
    - mRNA is introduced and causes the process of translation.
    - Cas9 functions to repress expression of the CCR5 delta-32 allele.
    - Cas9 cuts DNA
    - The cut is repaired introducing CCR5 delta-32 mutation.
    - New proteins are created from the mRNA template.
- Radiation Therapy
    - Cas9 enzyme binds to gRNA
    - Cas9 cuts DNA
    - The cut is repaired introducing CCR5 delta-32 mutation.

As I pointed out in my research paper, people who have iron deficient blood (anemia) have a higher risk for severe disease complications from COVID-19. This is because during the nutritional immunity phase iron is sequestered, and consequently, iron levels rapidly dip to dangerous levels. Though bad for the host, this is good for the gene editing experiment because HrrF gene expression is higher when iron levels are low. This is dangerous for those with even mild iron deficiencies, and as a reminder, *Blacks have moderate to severe anemia almost 3 times more than Whites and Hispanics*.

Attempts at CCR5 delta-32 mutations using certain base editors (i.e., AID mediated mutagenesis (TAM) and CRISPR-X) can cause evolutionary changes and hold many unknowns particularly for the next generation who may experience high rates of birth defects due to unintended alterations in chromosomal arrangements. Additionally, this experiment may be the cause for increased cancer rates due to higher levels of radiation and it can spur the emergence of new diseases. Further, it is well established that bacteria have memory that is transmittable to the next generation. In NTHI, P6 contains that memory, thus targeting the TLR2:P6 signaling axis is dangerous and will cause evolutionary changes to the human genome.

Closing Statement & Advice:

COVID-19 is biological terrorism against all of humanity, and a blatant violation of informed consent. Further, it is a Gain of Function gene editing experiment whereby: All test subjects (humans) are subjected to a change in the environment due to the intentional release of a genetically engineered virus (COVID-19), for the purpose of gene silencing using CRISPR-Cas9 Technology. Primary Therapeutic Target: CCR5 delta-32 allele.

COVID-19 is also a mass genocide attempt targeting: Persons of African descent, Hispanics, Native Americans, Asians, and Middle Eastern persons. COVID-19 is also a is a population control effort targeting the world's poorest members namely those in; Africa, India, and South America.

The perpetrators behind this attack on humanity need to be swiftly identified and held to account for their reckless and shameful actions. It is also imperative to catch them before they release additional strains of this virus on society. I do encourage those investigating the origins of SARS-CoV-2 to;

- Investigate any gain of function research involving (but not limited to) bacteriophages HP1/S2 and H2 of NTHi and coronaviruses done within the U.S. and abroad;
- Fully investigate research Peter Daszak and EcoHealth Alliance, research partners and associates, and funding institutions including Bill Gates, the Wuhan Institute of Virology, and the National Institutes of Health concerning research on NTHi, CCR5 Silencing, CRISPR-Cas9 technologies, HIV/AIDS research, P6, Influenza, and HIV vaccine development, genetic drug therapies, etc.
- Investigate all experiments involved in the creation of Harvard University's patent number # 10745677.
- Make public Moderna, J&J, and Pfizer & Biontech mRNA vaccine formulations and specific ingredients for mRNA publicly available by relaxing patent protections so that the larger scientific community may investigate these claims further.

Keywords: Nontypeable Hemophilus Influenzae, HIV, Biowarfare, Bioweapon, COVID-19, SARS-CoV-2

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