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Mini Review

A Cautionary Tale: What if Stealth Adapted Viruses Incorporate the Covid-19 Spike Protein Coding mRNA into Their Genome?

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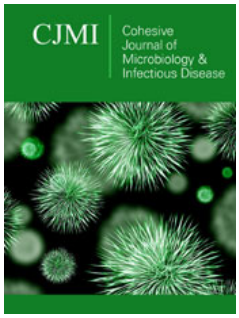

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Stealth adaptation is a generic immune evasion mechanism, which can potentially occur with all viruses [1-4]. It involves the deletion or mutation of the genes coding for the relatively few virus components typically targeted by the cellular immune system. A further characteristic of certain stealth adapted viruses is the incorporation of additional genetic sequences of cellular and bacterial origins [5-8]. These "renegade" genetic sequences can be subsequently transmitted between humans as components of infectious stealth adapted viruses. The best characterized stealth adapted virus, referred to in GenBank as stealth virus 1, is a derivative of an African green monkey simian cytomegalovirus (SCMV) [2,4,9]. It was repeatedly cultured from a patient with the chronic fatigue syndrome (CFS) [10]. A similar virus (stealth virus-2) was isolated from the cerebrospinal fluid of a comatose patient with a 4-year history of a bipolar psychosis [11]. Stealth virus-1 was cloned and the complete or partial DNA sequences of 248 clones were submitted to GenBank. Of these clones, 200 of the sequences correspond to regions of the SCMV genome. The sequences are unevenly distributed over the genome of the originating SCMV, with clear evidence of major deletions and genetic instability. Fourteen clones contained genetic sequences that were derived from non-coding regions of the human genome. These incorporated cellular sequences have also undergone genetic changes indicative of the genetically unstable stealth adapted virus genome [12].

Thirty-four sequences from stealth virus-1 correspond to modified bacterial protein-coding sequences. These sequences may have arisen from coinfection of cells with both stealth adapted viruses and intracellular bacteria, with the bacteria-derived sequences providing specific metabolic functions and/or substituting for some of the ordinary virus capsids proteins. The overall genome of stealth virus-1 is genetically fragmented leading to the likelihood of the cellular and bacteria-derived sequences becoming incorporated into the virus genome by the cross-linking of fragments from the originating SCMV. Cellular sequences incorporated into the stealth adapted viruses isolated from certain other CFS patients (designated in Gen Bank as stealth virus numbers 3,4,5) include sequences from the rhesus monkey genome [13-16]. There are also human-derived cellular sequences in stealth virus-5 [13,14].

The presence of these human-derived sequences is consistent with homologous recombination, which would allow for the replacement of some of the original rhesus-derived sequences in stealth virus-5 with human cellular sequences. A similar explanation can apply to the finding of human cellular sequences instead of African green monkey cellular sequences in stealth virus-1 [7-8]. The incomplete inclusion and genetic instability of the originating virus sequences, along with further replacements of virus sequences with cellular and bacteria-derived sequences have impeded efforts by many virologists to detect these

viruses in patients. The existence of stealth adapted viruses has unfortunately been disregarded by public health authorities despite the unequivocal DNA sequence data. The biased, not-wanting-to-know ignorance of the public health system led to the declaration in 2002 that the clinical culturing of stealth adapted viruses had put the Nation's health into Immediate Jeopardy. All clinical testing had to cease under threat of criminal charges and financial penalty.

The purpose of this article is to raise the possibility of stealth adapted viruses capturing the Covid-19 S-protein mRNA sequence used in the Pfizer and Moderna vaccines [15,16]. This could lead to the overproduction of the S-protein and to a major disruption in the functioning of the angiotension-converting enzyme-2 (ACE2) receptor [17-19]. Arguably, there could be either excessive activation or inhibition of the functions of the ACE2 receptor. In either event, the biological effects would be potentially profound. Additionally, there could be continuing formation of antigen-antibody complexes as well as enhanced tissue-destructive cellular immunity directed to the Covid-19 S protein. Because stealth adapted viruses are infectious, the potential deleterious effects of a Covid-19 S protein coding stealth adapted virus could spread throughout the human and animal world. A reasonable request to those responsible for approving Covid-19 vaccines is to seriously consider the potential worldwide risks caused by the foreseeable interactions of the vaccines with stealth adapted viruses.

At a minimum, the FDA should be formally required to scientifically respond to the concepts embodied in this article. Stealth virus-1 is available from the American Type Culture Collection (VR-2343, deposited September 17, 1991). Candidate mRNA vaccines could be added to stealth virus-1 cultures to determine the ease with which the Covid-19 S protein coding mRNA can become included into a replicating stealth adapted virus. Additional considerations should also be given to other possible interactions between the Covid-19 vaccine and its included adjuvants and stealth adapted viruses. Vaccine science has previously failed the world, including the likely inadvertent development of HIV. It is still reluctant to address vaccine-derived stealth adapted viruses as an explanation for the rising incidence of brain damaging illnesses, including autism and CFS. Although typically non-immunogenic, residual components on stealth adapted viruses can become antigenic targets if the immune system is sufficiently stimulated with adjuvants. This can explain the instances of vaccine induced neurological illnesses. It would indeed be tragic if the future of mankind were to be further imperiled by a continuing FDA disregard of the existence of stealth adapted viruses.

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