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Stealth adapted viruses and the epidemic of chronic illnesses

Abstract

Stealth adapted viruses elude recognition by the cellular immune system due to the loss or mutation of genes coding the relatively few components typically targeted by the cellular immune system. Political barriers to accepting the existence of these viruses arose when it became apparent that some of the viruses had originated from the cytomegaloviruses that commonly infected monkeys used to produce poliovirus vaccines. Many virologists are seemingly unaware of the restricted targeting of viral components by the cellular immune system or that genetically defective viruses can continue to replicate and cause cellular damage. Some immunologists may also be somewhat reluctant to acknowledge possible non-immunological virus defense mechanisms. There are growing concerns regarding the increasing incidence of major chronic illnesses. Patient support groups are continually advocating for more research on the cause of specific disease entities. There is also a growing sense that some industries may have unintentionally imposed toxic exposures on the public leading to chronic illnesses. Relief from such exposures is being demanded by various Health Freedom movements. This article is intended to better inform the Health Freedom movements and various chronic illness support groups about the existence of stealth adapted viruses. A broader understanding of these viruses and their incorporated renegade cellular and microbial sequences will facilitate therapeutic endeavors, especially those based on the Alternative Cellular Energy (ACE) pathway.

Keywords: Alternative cellular energy (ACE) pathway, chronic fatigue syndrome, chronic Lyme disease, autism, PANDAS, amyotrophic lateral sclerosis, Gulf War syndrome, Health Freedom, SCMV, vaccine safety, polio vaccine, *Mycoplasma fermentans, Ochrobactrum, Porphyromonas gingivalis*, SV-40, FDA, GenBank, public health

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Abbreviations: ACE, alternative cellular energy; CFS, chronic fatigue syndrome; CSF, cerebrospinal fluid; PCR, polymerase chain reaction; SCMV, African green monkey simian cytomegalovirus; RhCMV, rhesus monkey cytomegalovirus; HHV-6, human herpesvirus-6

Introduction

Health freedom advocates cite increases in the incidences of many chronic illnesses including autoimmune diseases, depression and anxiety disorders, neurodegeneration, autism, learning and behavioral problems, as well as gender identification problems in children, obesity, allergies, diabetes, liver cirrhosis, and chronic fatigue syndrome (CFS).[1–4] Certain cancers are viewed as not only being more common but growing faster than normal,[5] such that they are sometimes referred to as turbo cancers. Incomplete recovery is occurring in approximately a quarter of those clinically infected with the COVID-19 virus.[6] Additional support for deterioration in health is provided by recent rises in overall mortality numbers in all age groups.[7]

There is a growing concern that the Government and pharmaceutical industry are unwilling to acknowledge and address possible underlying causes. Patient advocacy groups collectively highlight a wide range of potential explanations. These include reduced nutritional value and inclusion of synthetic chemicals in processed foods, exposure to a range of toxic chemicals in the home and workplace, higher levels of manmade electromagnetic radiation, adverse effects of vaccines and prescribed pharmaceuticals, lack of regular exercise, social isolation, insomnia, envy evoking advertising, and increased levels of economic difficulties and environmental stress.[8–13]

What is noticeably absent from the listing of potential causes is consideration of an infectious etiology, specifically due to stealth adapted viruses.[14] These viruses differ from the viruses from which they are derived in that they do not normally evoke inflammation the hallmark of most infectious diseases. The immune evasion is attributed to the loss or mutation of the viral genes that code for the relatively few virus components that are generally targeted by the cellular immune response. The best-characterized stealth adapted virus is a derivative of an African green monkey simian cytomegalovirus (SCMV).[15] Beginning in the early 1960s, cultured kidney cells from SCMV-infected African green monkeys were routinely used to produce live poliovirus vaccines.[16]

Cultured kidney cells from these monkeys replaced the prior use of cultured kidney cells from rhesus monkeys, which were also commonly infected with rhesus monkey cytomegalovirus (RhCMV). The use of rhesus monkeys was specifically discontinued when kidney cell cultures from many of these monkeys were shown to be infected with simian virus-40 (SV-40), a potential cancer-causing virus.[16] Interestingly, at least one polio vaccine manufacturer defied this ban and continued to use rhesus monkeys in polio vaccine production.[17]

It is understandable, yet not justified for Public Health authorities to refrain from publicly expressing doubts regarding the safety of vaccines. The generic argument is that criticisms of vaccines will discourage their use with more adverse consequences than those of using a possibly defective product. USA and UK Governmental efforts failed to culture SCMV from previously released polio vaccines in which the DNA of SCMV could be detected.[17,18] My request to the Food and Drug Administration (FDA) for access to these vaccines was denied because of proprietary restrictions imposed by the vaccine manufacturer.

Questioning vaccine safety is probably not in the best interests of those dependent upon Government and pharmaceutical grants. Moreover, raising such questions is likely to impede academic promotions and appointments to influential committees. Vaccines are an income source for pediatricians and the refusal to comply with Government vaccine mandates can lead to disciplinary actions.

What is surprising and somewhat disconcerting is the very limited understanding and lack of open discussion of stealth adapted viruses by leadership in the Health Freedom Movement and various disease disease-specific patient support groups. It means that these viruses have not been sufficiently well explained to establish widespread confidence in their being a major contributing cause of chronic illnesses. The purpose of this article is to address this issue by providing an easy-to-follow chronology of the research leading to the description of stealth adapted viruses. The purpose of this article is to address both issues by providing a descriptive summary of stealth adapted virus research. Supporting data are provided in the cited publications. A subsequent article will update the therapeutic implications of the inhibition of stealth adapted and other viruses by the alternative cellular energy (ACE) pathway.[14,19,20]

Basis for initial studies

The research program was founded on the linkage of three 1986 publications. The first was the outbreak of an illness in Lake Tahoe, Nevada.[21] It had the clinical features of what certain medical practitioners in the US and Europe were calling myalgic encephalomyelitis[22] and loosely attributed to chronic infection with Epstein Barr Virus (EBV), a type of herpes virus.[23] This illness was subsequently called chronic fatigue syndrome (CFS). The second publication described the culturing of a human virus that soon became classified as human herpes virus-6 (HHV-6).[24] The third publication described the polymerase chain reaction (PCR), a very sensitive molecular diagnostic assay technique.[25] It uses small synthetic DNA primers to amplify known DNA sequences. A rather straightforward project was, therefore, to use the PCR assay to test local patients diagnosed as having CFS for evidence of HHV-6 infection.

Early findings

Using standard PCR conditions, the PCR results using HHV-6 primers were negative. However, by reducing the stringency of the PCR to allow for more cross-reactivities, positive responses were seen in about a third of tested patients but not in healthy controls.[26] Moreover, using low stringency conditions, certain primers designed to amplify either other herpes viruses or even other types of viruses also commonly yielded positive results in blood samples from patients with negative PCR findings in healthy controls.

Positive low stringency PCR findings were also seen in testing blood and cerebrospinal fluids (CSF) of patients with a range of neurological illnesses. These included the testing of CSF samples repeatedly collected from a newborn child in whom a shunt had been placed to alleviate elevated intracranial pressure. While there were some clinical features suggestive of a congenital viral infection, there was no accompanying inflammation. Another PCR-positive individual was an adolescent diagnosed as having herpes simplex virus (HSV) encephalitis. He was the subject of a lawsuit. His clinicians were being sued for withholding Acyclovir therapy early during his hospital admission. This therapy was only started after the development of major neurological indications of brain damage. The earlier hesitation to begin Acyclovir was because his CSF had no inflammatory cells.

The most striking example of positive PCR in the absence of inflammation was a brain biopsy obtained in early 1990 on a 39-year-old schoolteacher.[27] Magnetic Resonance Imaging (MRI) showed near-confluent periventricular lesions. The biopsy was obtained to exclude malignancy. Non-inflammatory histologic and electron microscopic signs of cellular damage were present. Were a virus to be present as indicated by the PCR finding, it seemingly had a stealth-like quality of evading the immune system.

Positive virus cultures

Prior efforts to culture viruses from CFS patients yielded negative or only marginally equivocal results. Based on the positive PCR findings, added efforts were made to culture a virus from the blood of a 43-year-old woman subsequently diagnosed as having CFS. Repeated positive cultures were obtained from the patient's blood and designated as stealth virus-1.[28] The second positive culture was from the acellular CSF of a comatose 21-year-old female patient with a 4-year history of bipolar psychosis.[29] Her initial clinical diagnosis at age 17 was schizophrenia. The virus from her CSF and subsequently from blood samples was designated as stealth virus-2. Both stealth virus-1 and stealth virus-2 produced a similar cytopathic effect in multiple types of cultured cells and in cells of several species.[28] The positive cultures from these and other patients raised three questions.

1. What was the nature of the cultured virus?
2. Why was there no inflammation?
3. Why had my prior efforts at culturing viruses not been successful?

PCR on virus cultures

To answer the first question, the PCR assay that yielded positive responses in the testing of the blood of the culture-positive patient was performed on the patient's culture.[28] The assay yielded positive PCR products that could be easily identified in agarose gel electrophoresis. Two large PCR products were cloned and sequenced. The sequences were compared with the DNA sequences that were then available on GenBank a national repository of DNA, RNA, and protein sequences. One product showed 58% nucleotide identity with human cytomegalovirus (HCMV). The other product showed no significant DNA matching.[28]

Isolation, cloning, and sequencing of viral DNA

An isolated PCR product was radiolabeled and used as a probe to identify the virus in filtered, ultracentrifuged, and agarose gel-separated DNA and RNA. The DNA was double stranded, migrating with a size of either single or multiple fragments of approximately 20 kilobases (kb). Using restriction enzymes, the viral DNA was cloned into plasmids, which were either partially or completely sequenced.[28]

SCMV origin of the virus

At the time there were only limited DNA sequences of animal cytomegaloviruses, some of the cloned sequences could be aligned to those obtained from SCMV.[15,30] The alignment was in the order of approximately 95% identity, with significantly lower levels of identity to RhCMV. This established an unequivocal origin from SCMV with a significant degree of mutation. Of 349 partially or fully sequenced clones, 300 could be matched to regions of the SCMV genome. The matching, however, was unevenly distributed along the entire SCMV genome which measures 226 kb. Over 30 clones matched some SCMV regions, while many other regions had far fewer matching clones.[31] Approximately half of the SCMV genome had no matching cloned sequences. There were also significant sequence differences between clones that matched to the same region of the SCMV genome. It was, therefore, concluded that the virus had a fragmented, genetically unstable genome.[32]

Renegade cellular sequences

Fifteen clones had sequences that matched closely to non-coding sequences within the human genome.[33,34]

Given the SCMV origin of the virus, monkey-derived rather than human-derived cellular sequences might have been anticipated. Indeed, the cellular sequences were probably monkey-derived. This conclusion has come from sequencing of PCR products generated from stealth adapted virus cultures from several other CFS patients. A sequenced PCR product from stealth virus-3 and all six sequenced PCR products from stealth virus-4 matched near-identically to regions of the rhesus monkey genome. Of seven sequenced PCR products from stealth virus-5, four could be most closely matched to the rhesus genome and three most closely matched to the human genome.[35] These data are consistent with homologous recombination replacement of originating monkey sequences with matching human sequences.

There are important Public Health implications of stealth adapted viruses being able to transfer genetically unstable cellular sequences between species and between individuals of the same species, including humans and animals. One of these is the conversion of a genetic illness to an infectious illness, including cancer. Another is the possible occurrence of an entirely new disease entity attributed to a mutated cellular gene.

Renegade bacterial sequences

Thirty-four of the sequenced clones from stealth virus-1 matched with very high significance to bacterial sequences.[33,36] There were no bacteria in the cultures that could be maintained in antibiotic-free media. Ten of the bacterial sequences matched regions of the genome of *Mycoplasma fermentans*. These bacteria had been identified as co-pathogens in many HIV-infected patients.[37] Molecular evidence for the possible presence of these bacteria in a range of additional illnesses, including children with autism, adults with amyotrophic lateral sclerosis (ALS), chronic Lyme disease, and Gulf War syndrome.[38–42] Stealth adapted virus cultures yielded positive results on most tested patients with these conditions. Indeed, in the period between 1991 and 2002 several hundred positive cultures were obtained from children with autism[43] and patients diagnosed with chronic Lyme disease.[44]

Most of the remaining bacterial sequences in stealth virus-1 cultures match sequences in *Ochrobactrum* an alpha-proteobacteria.[36] Furthermore, some of the data are consistent with genetic recombination between sequences from different bacterial strains. It will be of interest to sequence stealth adapted viruses from chronic Lyme disease patients[44] to determine whether this diagnosis is based on the presence of renegade *Borrelia burgdorferi* sequences, rather than intact bacteria.

Other neurological illnesses possibly wrongly attributed to bacterial infections include PANDAS (Pediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcus),[45] Alzheimer disease associated with evidence of *Porphyromonas gingivalis,*[46] *Desulfovibrio* bacteria in Parkinson's disease.[47] The association could be either due to autoimmune cross-reactivity between bacterial and neuronal proteins or direct enzymatic activity of the bacterial renegade genetic sequence incorporated into the stealth adapted virus genome.

Redefining stealth adaptation

The original definition of stealth adapted viruses focused on the loss or mutation of viral genes, including those coding for the relatively few components that are normally targeted by the cellular immune system. This definition has been extended to include the additional potential acquisition of renegade, transmissible genetic sequences, which can be derived from cellular genomes or other microbes. Both the originating viral sequences and the acquired renegade sequences are genetically unstable and subject to major changes from homologous recombination. The brain is particularly susceptible to symptomatic illness caused by stealth adapted virus. This is due in large part to the spatial distribution of major functional regions of the brain along with its complex networking. Organs with more uniformity of functions can internally compensate for limited localized virus cellular damage. Malignancy is an exception in which a genetically modified single cell can potentially lead to a fatal illness. The induction of autoimmunity is another example of organ-wide damage due to stealth adapted viruses.

Virus infection in pregnant women is a major risk for brain damage occurring in the developing fetus. Even if the direct virus damage is minimal, the infected cells may still express some viral components against which there is normally no cellular immune recognition. Intensifying immune reactivity using other vaccines and their adjuvants can potentially evoke fresh immune recognition of these minor residual components. This is consistent with vaccine-induced triggering of autism in infants and of neurological illness in adolescents receiving human papillomavirus vaccine.[48] A related explanation can potentially explain certain adverse effects in adults receiving COVID vaccines.

Stealth adapted virus infections are typically characterized by periods of severe exacerbations and near-complete remissions. Moreover, the predominating symptoms can also vary over time. Clinical recovery occurred in the absence of inflammation in stealth virus-1 inoculated cats.[49] These observations point to a potent non-immunological anti-virus defense mechanism. This mechanism has been characterized in terms of the alternative cellular energy (ACE) pathway. Means that are available to enhance the ACE pathway in humans are effective in suppressing illnesses from conventional viruses, as well as bacterial pathogens.

Conclusion

This article is primarily written for those who may not have encountered information describing stealth adaptation to a virus immune evasion mechanism. Even a cursory review of the available information indicates that certain of these viruses are undoubtedly derivatives of the cytomegaloviruses infecting monkeys used to produce polio vaccines. Moreover, it is understandable that stealth adaptation is likely to be a generic process applicable to all viruses. Further review shows that stealth adapted viruses were near-uniformly cultured from blood samples obtained from patients diagnosed as having CFS, chronic Lyme disease, Gulf War syndrome, autism, and multiple myeloma. This article is also addressed to those in the Health Freedom Movement and to those who manage various patient support groups that have listened to discussions on stealth adapted viruses but show no further interest. Rather, they persist in emphasizing the dangers of currently administered vaccines, toxic chemicals, psychosocial factors, etc. A widespread collective effort acknowledging the existence and importance of stealth adapted viruses would help expedite the restoration of health to many of those infected. Effective educational programs regarding stealth adapted viruses will help maintain or even increase the memberships of the Health Freedom Movement and patient support groups. The prospect of an infectious cause creates degrees of urgency and vulnerability beyond those associated with chemical exposures. Benefits can also come from the sharing of observations relating to infections in

different individuals and animals. Facilities are needed that will allow for the culturing and sequencing of stealth adapted viruses. A final benefit of acknowledging the existence of stealth adapted viruses is the opening provided for major clinical trials to optimize therapies based on the alternative cellular energy (ACE) pathway.

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Conflicts of interests

The author declares that there are no conflicts of interest.

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