Stealth Adapted Viruses Can Incorporate Renegade Cellular and Bacterial Genetic Sequences: Public Health Implications

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Abstract

The cellular immune system normally responds to relatively few of the different structural components that comprise the complete virus. Mutation or deletion of the genes coding for these few antigenic components is an immune evasion mechanism termed "stealth adaptation." I initially used this term to describe a virus derived from an African green monkey simian cytomegalovirus (SCMV). This article provides an extended discussion of the Public Health relevance of previously reported findings relating to this virus. Of particular significance, the virus did not evoke inflammation in the chronic fatigue syndrome (CFS) patient from whom the virus was repeatedly cultured, nor in virus inoculated animals. The viral genome consists of multiple fragments of double stranded DNA with lengths of approximately twenty thousand nucleotides (20 kb). This is in marked contrast to the > 225 kb size of the normal SCMV genome. Purified virus DNA was cloned, and sequence data were subsequently obtained. Most of the cloned sequences match to regions corresponding in their entirety to only approximately half of the originating SCMV genome. These matching sequences are very unevenly distributed along the SCMV genome. Moreover, there are significant genetic sequence differences between clones matching to identical regions of the SCMV genome. In addition to the SCMV matching sequences, there are sequences that match to regions of the human genome. There are also sequences that match closely to genes of bacterial origin. The major sources of the bacterial sequences in the initially cultured stealth adapted virus are from *Mycoplasma fermentans* and *Ochrobactrum quorumnocens* bacteria. These findings have extended the generic concept of stealth adaptation to include not only the loss or mutation of portions of the originating virus genome, which would have otherwise resulted in cellular immune recognition, but also the potential incorporation/acquisition of additional "renegade" genetic sequences from cellular genes and from other microbial genomes. The apparent acquisition of cellular genetic sequences by stealth adapted viruses may potentially lead to the infectious transmission of genetically determined illnesses. The inclusion of bacterial sequences is also concerning since it indicates possible bacteria mediated transmission of infectious stealth adapted viruses. Furthermore, the transmissible bacterial sequences can potentially result in the mistaken diagnosis of a stealth adapted virus infection for a bacterial disease. Examples are likely to include chronic Lyme disease and PANDAS, a severe childhood psychiatric illness. Public Health officials should respond urgently to the existence of stealth adapted viruses.

Keywords: Stealth adapted viruses, Stealth adaptation, African green monkey simian cytomegalovirus, SCMV, KELEA, Kinetic Energy Limiting Electrostatic Attraction, Alternative Cellular Energy, ACE pigments, Chronic fatigue syndrome, CFS, chronic Lyme disease, Gulf War Syndrome, PANDAS, polio vaccine, *Mycoplasma fermentans* , *Ochrobactrumquorumnocens* , *Borrelia burgdorferi* , viteria

Abbreviations: ACE – Alternative Cellular Energy, KELEA - Kinetic Energy Limiting Electrostatic Attraction, SCMV - African green monkey simian cytomegalovirus, CFS – chronic fatigue syndrome, CPE – cytopathic effect, CSF – cerebrospinal fluid, kb – kilobases, MI – mental illness

Discussion

Research conducted within my laboratory into a viral cause of the chronic fatigue syndrome (CFS) has

faced political difficulties primarily because the initially identified virus in a CFS patient is derived from an African green monkey simian cytomegalovirus (SCMV).[1] Kidney cell cultures from cytomegalovirus infected African green monkeys were routinely used in the production of live polio virus vaccines.[2] Stored bulk lots of several approved batches of polio vaccines have been shown to contain SCMV DNA, although Public Health authorities were unable to culture replicating viruses.[3,4] About 10% of the cultures of polio vaccine viruses were apparently discarded by the manufacturer because of SCMV contamination (personal communication from an employee). A relevant FDA official was seemingly unaware of the high incidence of SCMV contamination, which should have justified the imposition of more stringent detection methods. The detailed characterization of a cytomegalovirus-related cytopathic virus cultured from a CFS patient was reported in 1994 in the American Journal of Pathology.[5] The unequivocal derivation of this virus from SCMV was described in 1995 and confirmed in several subsequent publications.[1,6-8]

Polio vaccine viruses were earlier grown in kidney cell cultures obtained from rhesus monkeys.[9] The switching to the use of African green monkeys occurred in 1963 following the detection of simian virus-40 (SV-40) infection in many rhesus monkeys.[10-12] Dr. Albert Sabin had previously cultured an atypical virus contaminant in the rhesus monkey-derived CHAT experimental polio vaccine.[13] This vaccine was subsequently shown to contain DNA of rhesus cytomegalovirus.[4] Since the atypical virus was difficult to culture, it was likely a stealth adapted rhesus cytomegalovirus. The difficulty in culturing may explain why the presence of the virus was denied by the CHAT vaccine manufacturer. This vaccine had been extensively tested in African chimpanzees, as well as in African children during the late 1950s. As described elsewhere, the human immunodeficiency virus (HIV) likely arose from chimpanzees inoculated with the contaminated CHAT polio vaccine.[15]The ominous transmission of rhesus monkey cellular sequences has occurred in stealth adapted virus infected humans.[16-18] Certain of the originally transmitted primate sequences appear to have been subsequently replaced by human genetic sequences, presumably by homologous recombination.[16,17]

There is a notable finding with regards to the detected rhesus cellular sequences in different stealth adapted virus cultures. It is the close matching of several of the rhesus sequences detected in the cultures from three unrelated CFS patients.[16,17] This finding implies that stealth adaptation may be a relatively rare occurrence with further selection of more highly infectious viruses.

The initially cultured stealth adapted virus consists of genetically unstable, DNA fragments with a size of approximately 20 kb.[5,6] This is less than a tenth of the 226 kb size of SCMV and more in keeping with the size of large RNA viruses. This finding along with the significant sequence differences in the clones matching to the same region of the SCMV genome implies the probability of RNA to DNA replication presumably involving an endogenous reverse transcriptase. This would also explain the minor sequence variations between matching polymerase chain reaction (PCR) amplified products in other stealth adapted virus cultures.[16,17] Direct RNA to RNA replication may, however, be occurring with certain other SCMV-derived stealth adapted viruses. For example, a positive PCR results with a patient's culture required prior RNA to DNA reverse transcription.[19] Of considerable concern is the potential of stealth adapted viruses to acquire highly pathogenic, transmissible cellular sequences.[16-18,20,21] These can arise by either genetic mutation or the acquisition of sequences from abnormal cells, including cancer cells. Stealth adapted virus infections can pass reciprocally between humans and animals providing additional opportunities for the infectious transmission of genetically determined illnesses.[22] Public Health epidemiologists should be monitoring for unanticipated rises in the incidences of various chronic illnesses in humans and animals.

CFS and similar chronic illnesses have been attributed to various types of bacteria. This conclusion has been primarily based on positive serology but also supported by genetic detection methods.[23-31] Included as a possible causal bacteria is *Mycoplasma fermentans,* which is also described as a common co-pathogen in HIV infected AIDS patients.[32-34] The evidence of infection by *Mycoplasma fermentans* is not restricted to CFS and HIV infected individuals.[33] Portions of its DNA have been detected in many patients with other illnesses including amyotrophic lateral sclerosis, Gulf War syndrome, rheumatoid arthritis, autism, and chronic Lyme disease.[24,35-39] The identification of *M. fermentans* derived genetic sequences in the initially cultured SCMV-derived stealth adapted virus suggests a different interpretation.[21,40-43] Rather than indicating a

bacterial infection, the detected *Mycoplasma*- related genetic sequences in a wide range of illnesses may be components of infecting stealth adapted viruses. Indeed, I was privately informed that the sequencing studies on CFS and chronic Lyme disease diagnosed patients revealed unanticipated minor discrepancies from authentic*M. fermentans* DNA sequences. The other major source of bacterial sequences in the initially cultured SCMV-derived stealth adapted virus is closely related but not identical to *Ochrobactrumquorumnocens* , an alpha proteobacterium.[21]Somewhat related bacteria have been implicated as a possible cause of Morgellons disease,[44] although there is currently more emphasis on this illness being potentially due to *Borreliaburgdorferi* ,[45] the bacteria that causes acute Lyme disease.

So called chronic Lyme disease has many clinical features in common with CFS and fibromyalgia patients.[23,46] Extensive culturing of blood samples from patients diagnosed as having chronic Lyme disease showed the consistent presence of stealth adapted viruses. DNA sequence analysis was not performed on any of the cultured viruses to check for the presence of *Borrelia burgdorferi* genetic sequences. Chronic Lyme disease patients commonly show evidence for additional infections, which include *Bartonella, Ehrlichia, Mycoplasma,* and other bacteria and the *Babesia*parasite.[47] The evidence is more commonly serological rather than isolation of organisms. It has led to long-term antibiotic therapy with questionable benefits. Detailed sequencing of stealth adapted viruses cultured from diagnosed chronic Lyme disease patients is indicated.

Another possible example of the mistaken identification of stealth adapted virus infections as a bacteria-induced illness is PANDAS (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections).[48,49] From personal experience in reviewing laboratory testing data, it is not uncommon for there to be either anti-Streptolysin O (ASO) or anti-DNase B antibody but not both, as would be expected if there were an infection with viable bacteria. Positive Lyme disease serology has also been reported in association PANDAS.[50] Moreover, stealth adapted viruses were reliably cultured from children clinically diagnosed as having PANDAS. The generic term "viteria" has been introduced to help convey the concept of stealth adapted viruses being potential carriers of bacteria-derived genetic sequences.[21,40]

Unlike the cellular sequences, which are derived from non-coding regions of cellular genes, the bacteria-derived genetic sequences code for structural components with potential metabolic, enzymatic, and/or immunogenic activities. [21,40-43] It is also possible that some of these components have biophysical properties, including the indirect providing of a non-food source of cellular energy. A feature of stealth adapted virus cultures is that the virus induced cytopathic effect (CPE) becomes suppressed in the absence of regular replacement of the culture medium. The CPE quickly recurs upon the refeeding with fresh medium.[51] The suppression the CPE correlates with the cellular production of energy transducing materials that form into self-assembled, pigmented particles in the extracellular fluids. The materials can further develop into colorful threads and ribbons[51,52] Reactivation of the virus CPE does not occur if a few of these particles are first added to the refeeding culture medium.[51] The particles are fluorescent, especially in the presence of certain dyes, electrostatic, electron donating, and occasionally ferromagnetic. Similar particles develop in stealth adapted virus infected patients, in whom they can be misidentified as parasitic insects.[52,53]

Subsequent research led to the understanding that cells have the intrinsic capacity to derive cellular energy by means other than through the metabolism of food. This alternative cellular energy (ACE) pathway involves the transfer of an environmental energy force into both the body's intra- and extracellular fluids. The force is termed KELEA, an abbreviation for Kinetic Energy Limiting Electrostatic Attraction.[54-58] Many naturally occurring, and cell synthesized compounds with regional differences in their electrical charges may continually attract and release KELEA, possibly upon oscillatory movements. Such compounds, termed ACE pigments,[51,52] can increase the KELEA levels in nearby fluids, including water. They include the extracellular materials developing in long-term stealth adapted virus cultures[51] Certain forms of fluctuating electrical activities in the brain and muscles are likely to be the major ways that humans and animals normally receive KELEA, which is then transferred to the body's fluids.[59] Some of the KELEA attracting brain and muscle mediated activities can presumably be learned.

The ACE pathway provides a non-immunological defense mechanism against stealth adapted viruses. It can also suppress other types of infections.[51,52,60-63] Moreover, the physiological functions of the ACE pathway

extend well beyond the control of infectious diseases.[64] It can be converted into chemical energy[58] and, thereby, help compensate for illnesses due to impaired metabolic activity. It may also enhance the biophysical processes that allow for the detection of changes in the physical environment. It can support tissue regeneration in the absence of scarring,[65] and help sustain a higher level of brain activity including conscious awareness. [66]

Although CFS was the early focus of research on stealth adapted viruses, positive cultures were obtained from patients with more widely recognized major neurological and psychiatric illnesses. Indeed, the second SCMV-derived stealth adapted virus was cultured from a 23-year-old woman who was initially diagnosed at age 19 with schizophrenia.[67] The diagnosis was changed to bipolar psychosis largely based on her clinical response to lithium therapy. She had required residential care since her diagnosis. She acutely deteriorated into coma prior to admission to the Los Angeles County Hospital in early 1991. The stealth adapted virus was cultured from an acellular cerebrospinal fluid (CSF) sample obtained upon her hospital admission. She remained comatose till her death several years later. Illnesses among family members of CFS patients also support an etiological role of stealth adapted viruses in major neurological and psychiatric diseases.[68,69] Prominent examples include Alzheimer diseases occurring in grandparents, CFS and amyotrophic lateral sclerosis (ALS) in parents, and severe learning and behavioral disorders in children. Another example is Parkinson's disease in a grandparent, CFS in both parents, and schizophrenia in a teenage son. Upon inquiry it is not uncommon to learn of CFS-like illness in mothers of children with autism. Stealth adapted viruses were consistently cultured from children with autism.[70]

Stealth adapted viruses were also cultured from all tested patients diagnosed with multiple myeloma.[71] This may explain the preceding or concurrent neuropsychiatric symptoms that are seemingly common among patients diagnosed with multiple myeloma. Either direct or circumstantial evidence also suggests a major role of stealth adapted viruses in glioblastoma multiforme and in certain leukemias. Potentially any type of virus can undergo stealth adaptation possibly with a particular likelihood of occurring with herpesviruses because of their large size.

It is important that the culturing and detailed sequencing of stealth adapted viruses be undertaken. Moreover, the testing should not be restricted to patients with neurological or psychiatric symptoms. Thus, while relatively few virus-coded components are targeted by the cellular immune system, a much wider array of components can typically evoke anti-viral antibodies. Such antibodies may provide a barrier to stealth adapted viruses from infecting the brain.[22] Because of genetic instability, there can be a range of structurally diverse antigenic components coded by the viral and acquired bacterial genes. The resulting antibodies can be reactive with a range of different viruses and bacteria. They can also lead to antigen-antibody immune complexes with the potential of inducing renal, joint, and other diseases. Stealth adapted virus infection can also potentially trigger common autoimmune diseases, including systemic lupus erythematosus (SLE), multiple sclerosis Hashimoto's disease, Addison's disease, Type 1 diabetes, idiopathic thrombocytopenia (ITP), etc.

Stealth adapted viruses can also be an important cofactor in many common illnesses in addition to those that have been previously mentioned. The similarity of symptoms, including fatigue and cognitive deficits, between CFS patients and many of those with the Long Covid Syndrome[72] warrant the culturing of these patients for stealth adapted viruses. This possible association is further supported by the concurrent elevations in anti-herpesvirus antibodies in certain patients diagnosed with the Long Covid Syndrome.[73,74]

Public Health authorities prohibited my clinical culturing of stealth adapted viruses in 1972 stating that I had put the Nation's Health into Immediate Jeopardy. Certain CDC, NIH, and FDA officials have repeatedly shied away from my requests that they confirm the culturing of stealth adapted viruses. It is time for a more proactive response.

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