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Viruses Disguised as Self and/or as Bacteria

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ABSTRACT

This article extends the concept of stealth adaptation of viruses beyond the avoidance of cell mediated immune recognition due to deletion or mutation of the genes coding for the relatively few virus components normally targeted by the cellular immune system. A fragmented virus genome can incorporate additional cellular and bacterial genetic sequences, while continuing to delete the originating virus sequences. Virus replication and transmission extends to the incorporated and substituted sequences, which have seemingly abandoned their normal locations to essentially become viral. By analogy with deserting or with the switching of allegiance, the term renegade sequences is suggested to describe the incorporated sequences beyond those remaining from the initiating virus. The over expression of certain genetic sequences has been linked to the occurrence of particular types of illnesses, including the development of various cancers. Although yet to be clinically acknowledged, this is an inherent risk of transmissible, genetically altered cellular sequences. Humans have become infected with monkey-derived stealth adapted viruses. The monkey-derived cellular sequences in these viruses can potentially undergo homologous recombination with human germline DNA leading to multigenerational illnesses. These and other major implications of the existence in humans of reformed viruses being disguised as self, and/or as bacteria are discussed in this article.

Keywords

Stealth adapted viruses, Renegade viruses, African green monkeys, Rhesus monkeys, Simian cytomegalovirus, SCMV, Polio vaccine, Chronic fatigue syndrome, Chronic Lyme disease, PANDAS, Morgellons, Glioblastoma, Multiple myeloma, Alzheimer, Borrelia burgdorferi, Porphyromonas, Ochrobactrum

Introduction

Viruses have traditionally been viewed as being fundamentally different in their genetic composition from either the genome of cells or of bacteria [1-4]. This is in spite of there being a few genes in some of the larger viruses, which in the distant past almost certainly arose from cellular genes [5-9]. There are also examples of certain animal retroviruses acting as carriers of specific cellular oncogenes, as is the case with Rous sarcoma virus [10]. The major differences between virus and cellular genomes allow for the use of serological and molecular markers to detect, categorize, and treat the different viruses known to infect humans and animals. Negative findings using these serological and/or molecular virus markers have led to dismissing a virus cause of many chronic illnesses [11-13]. This is especially true for those illnesses in which there is no accompanying inflammation; the accepted hallmark of most infectious diseases [14]. Recent research on stealth adapted viruses indicates that these reasons are not sufficient to exclude a virus cause of any particular chronic illness. This is because many if not all of the originating virus sequences can potentially be replaced by cellular and/or bacteria-derived genetic sequences [15]. The substituting sequences are being referred to as renegade sequences in the sense that they have deserted from their normal location to become functional components of replicating, transmissible and disease-causing viruses. This article will briefly summarize this additional extension of the stealth adaptation process and review some of the more important potential consequences for human health.

Stealth adaptation

Stealth adapted viruses differ from the viruses from which they are derived in not evoking an inflammatory response [16-20]. This is because of the deletion or mutation of the genes, which code for the relatively few virus components normally targeted by the cellular immune system. Certain stealth adapted viruses arose from African green monkey simian cytomegalovirus (SCMV), undoubtedly as

an inadvertent consequence of the use of cytomegalovirus-infected monkeys in poliovirus vaccine production [21]. The best studied SCMV-derived stealth adapted virus was repeatedly cultured from a patient with the chronic fatigue syndrome (CFS). It has a fragmented and genetically unstable genome. Large segments of the virus genome have been deleted with uneven distribution of the remaining fragmented portions of the original genome [22]. The replicating virus has incorporated additional genetic sequences from parts of the cellular genome [15,23] and also from several bacteria [15,24]. These added renegade sequences have presumably replaced or have been substituted for some of the originating virus genetic sequences. Both the remaining originating virus sequences and the incorporated cellular and bacteria-derived sequences have undergone further mutations due to genetic instability. The incorporated sequences replicate and are transmitted as components of the reformed viruses. Incorporated cellular sequences can cross the species barrier, as shown by rhesus monkey sequences in the stealth adapted viruses cultured from three different CFS patient [15].

This research is leading to a new understanding of viruses in which it is possible that the substituted genetic sequences may potentially comprise the entirety of the replicating and transmissible pathogen. This can have several major consequences in addition to bringing into question the reliance on conventional serological and molecular methods for virus detection. i) It can lead to a false assumption of a bacterial origin of certain illnesses. ii) It raises the prospect of a virus and, therefore, a potentially infectious cause of many existing illnesses, including psychiatric diseases and cancers. iii) It bodes badly for the stability of the human genome with the prospect of an increasing array of future illnesses. Each topic will be briefly discussed in this article.

Illnesses mistakenly attributed to bacteria

A prime example of these illnesses is chronic Lyme disease. It is widely attributed to infection with the bacteria Borrelia burgdorferi [25]. Dr Joseph Burrascano provided the author with numerous blood samples from patients who he had diagnosed with chronic Lyme disease. Almost without exception, these blood samples tested positive for stealth adapted viruses. As an interesting aside, the foamy vacuolated cellular changes typically occurring in human fibroblasts cultures of stealth adapted viruses are highly permissive for the in vitro growth of Borrelia burgdorferi (unpublished). It is possible, therefore, that borrelia-related genetic sequences could have become incorporated during the early passages of a stealth adapted virus in a borrelia-infected individual. These sequences would subsequently be transmissible to other individuals leading to a misleading diagnosis of a bacterial, rather than a viral disease. A similar argument can be made for claims that Morgellons skin disease is also a borrelia infection [25-27]. Another example of an illness that was previously attributed to a bacterial infection is CFS. It was popular some years ago to link this illness, as well as the Gulf War Syndrome, to infection with mycoplasma [28-29]. An interesting informal comment was made at the time by a mycoplasma researcher. He stated that the mycoplasma sequences he was identifying in CFS patients were actually slightly different from those of the then known mycoplasma species. A further example of a potentially false bacteria disease diagnosis is PANDAS (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections) [30-31]. On close analysis, there is often a discrepancy between the anti-streptolysin O and anti-DNase B antibody testing for streptococcus (unpublished). This would not be expected if the streptococcus bacteria were present. Indeed, the PANDAS diagnosis is not dependent upon culturing Streptococcus from the patients. The bacteria Porphyromonas gingivalis and chlamydia have both been linked to Alzheimer disease and to rheumatoid arthritis [32-38]. The possibility exists for a dynamic process in which the bacteria sequences previously incorporated into a stealth adapted virus may be replaced by other bacteria sequences present in a newly infected host. The consumption of food can be a pathway for a wide range of soil-based bacteria [39]. This is consistent with the finding of Ochrobactrum quorumnocens sequences in an SCMV-derived stealth adapted virus [15]. Of major concern is the prospect of bacteria providing a mode of transmission of stealth adapted viruses [24].

Illnesses not yet generally considered as being viral

The brain is particularly susceptible to symptomatic illnesses caused by stealth adapted viruses. This is because of the spatial distribution of the functional activities of the brain making it difficult to compensate for limited, localized tissue damage by heightened activity elsewhere in the brain. Blood and occasional cerebrospinal fluid (CSF) samples from patients with a range of neurological and psychiatric illnesses were cultured over a period of several years. Prominent examples of illnesses from which stealth adapted viruses have been cultured include CFS, fibromyalgia, autism, multiple sclerosis, amyotrophic lateral sclerosis, bipolar depression, schizophrenia, Parkinson's disease, and Alzheimer disease. Positive culture results were also reported on blood samples from occasionally referred hospitalized patients with an otherwise unexplained severe encephalopathy. Acute flaccid myelitis is an illness in which there is often some indications of a prior enterovirus infection but with inconsistent evidence of an active ongoing enterovirus infection at the time of diagnosis [40-43]. Along with some other illnesses, the inability to detect an active infection in these patients is sometimes ascribed to the virus acting in a hit-and-run manner. Rather, it could be a replacement of sequences from the originating enterovirus with cellular sequences. If so, the patients would still be positive in cultures for stealth adapted viruses. It is even possible that prions might induce a discernable CPE in stealth adapted cultures [44]. Positive stealth adapted virus cultures in patients with neurological illnesses can be extended to animal inoculation studies, as was done with the prototype virus cultured from a CFS patient [45].

Cancer is an illness in which genetic changes can be limited to a single cell. In a small study, stealth adapted viruses were cultured from all 10 tested patients with multiple myeloma, with a control group of 10 individuals having no positive cultures [46]. This finding is consistent with the frequency in which prior or ongoing neurological illnesses occur in patients diagnosed with multiple myeloma. A strikingly positive culture was also obtained in an

individual with an aggressive lymphoma and also in a patient with a glioblastoma. Other tumor types in which it will be informative to perform specialized stealth adapted virus cultures include breast, prostate, colon, and salivary gland. Obviously, the finding of a positive culture in a cancer patient might be due to a coincidental infection. Even if this were the case, it is conceivable that a secondarily infecting stealth adapted virus could acquire a human cellular oncogene, which could subsequently be passaged to other cells and other individuals.

Cancers typically arise from a single cell that has acquired a driver mutation in its genome [47]. This is different from infections in which multiple cells, even of different cell types, are simultaneously infected. Because of the monoclonal nature of cancer, there needs to be some additional cellular changes beyond the actual infection to trigger tumor cell growth. This is consistent with the multiple hit explanation for cancer, in which the virus is providing one of several hits. An additional requirement for cancer development can also be a failure of apoptosis, with the assumption that the initiation of transformation to malignancy normally leads to apoptosis [48]. A useful clue as to the possibility of a stealth adapted virus infection in a cancer patient is the occurrence of symptoms, such as excessive fatigue, cognitive impairment, etc., which persist even after the tumor is surgically removed. This feature has been described in several survivors of breast cancer [49].

Modifying the human genome

More than ninety percent of the RNA made from the human genome is from non-protein-coding regions [50]. These RNA molecules regulate many cellular activities [51]. Specific mutations occurring in certain non-coding genetic sequences have been linked to a wide range of clinical illnesses, including cancer. The imposition onto the human genome of a virus-delivered cellular sequence, which may be animal-derived, would not only affect the normal quantitative level of this sequence but could also affect its activity through any additional mutations or interspecies differences. Homologous recombination allows for the replacements of chromosomal sequences with closely related extrachromosomal sequences [52]. This can potentially occur in stealth adapted virus infected cells. Hereditable changes in the human genome can arise if the virus-delivered sequences become integrated into the genome of a subsequently fertilized germ cell. Stealth adapted viruses have been cultured from semen and virus particles have been seen by electron microscopy in close association with sperm. Since stealth adapted viruses can have a wide range of infectivity, which may extend to insect cells and to bacteria, the potential adverse effects of infectious modified cellular sequences are not necessarily confined to humans. Conversely, the human genome is at risk for being modified by genetic sequences from many foreign species.

Rethinking the production of vaccines for human use in cells from virus infected animals

Serious concerns were raised regarding the decision to use monkeys to produce live poliovirus vaccines. The major concern was the possible transmission of animal viruses to human. This occurred with the unforeseen contamination of rhesus monkey kidney cells with SV-40 virus [53-54]. The issue of SCMV contamination of poliovirus vaccines produced in African green monkeys was faced in 1972 when kidney cultures from all eleven tested monkeys were found by the FDA and the vaccine manufacturer to be infected with SCMV. No decisive action was taken with the excuse that any major problems would have been apparent in the previous decade of use of the vaccine. SCMV-derived stealth adapted viruses were reported to the FDA in 1995, but still without any constructive response other than an added incentive for the eventual replacement in the US of the live poliovirus vaccine with a killed vaccine [55]. It will be interesting to observe the FDA's response to the existence of infectious, mutated cellular sequences, presumably also contributed to by the development of virus vaccines.

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