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A new virus, a new disease?

Recent discoveries in immune depression have implications for research on AIDS and other new diseases.

A new member of the herpes virus family has been isolated in the laboratory of Dr. Robert Gallo at the National Cancer Institute, according to the Oct. 31 issue of *Science* magazine. This new human B-lymphotropic virus (HBLV) was isolated from the white blood cells of six patients, two of whom were seropositive for the AIDS virus, and four of whom were seronegative. All of these patients had either leukemia, lymphoma, or some other abnormality of the white blood cells, known as lymphocytes.

The new virus has the physical appearance (morphology) of a herpes virus and may be related to these viruses, which cause cold sores, genital herpes, chicken pox, shingles, and mononucleosis, and are involved in the production of a number of cancers. HBLV is distinguished from the other herpes viruses of animals and man, by an apparently restricted host range, infecting only fresh B-lymphocytes, and apparently killing them, as opposed to another herpes virus, the Epstein-Barr virus (EBV), which transforms infected B-lymphocytes into cancer cells.

The appearance of a new member of the herpes virus group of viruses has a number of implications. All members of the herpes virus family are associated, in one way or another, with derangements of the immune system. One member of this family, the cytomegalovirus, was an early candidate as the cause of Acquired Immune Deficiency Syndrome, because of its widespread prevalence in AIDS patients, and its ability to produce further immunosuppression in already immunocompromised hosts. This immunosuppressive characteristic is shared by the Epstein-Barr virus.

The other members of the herpes virus family—herpes simplex, which causes cold sores and oral ulcers; herpes genitalis, which causes genital herpes; and Varicella-Zoster virus, which causes chicken pox and shingles—all produce life-long latent infections which become active when the immune system is suppressed by infections or stress.

The initial evidence suggests certain parallels with the AIDS virus. As noted above, the HBLV apparently attacks B-cells in culture and destroys them. This is somewhat analogous to the situation with the AIDS virus (HTLV-III), which destroys T-cells in culture, as opposed to immortalizing them as the Human T-cell Leukemia Virus (HTLV-I) does.

One disease in which the new virus may play a part is a chronic mononucleosis-like syndrome which has come to the attention of the medical profession in the United States over the past two to three years. This syndrome, which some physicians attribute to a chronic Epstein-Barr virus infection, gained recent prominence with the report of 150 cases diagnosed in the Lake Tahoe area of Nevada between the fall of 1984 and the fall of 1985.

The disease is characterized by a cluster of non-specific symptoms including chronic fatigue, headache, swollen glands, recurrent headaches, and sore throats. In addition there are neurological complications which resemble those seen in AIDS patients, such as loss of memory and inability to concentrate or perform once routine mental tasks. Like mononucleosis, chronic mononucleosis syndrome appears to be highly contagious.

Many of these patients have evidence of exposure to most, if not all, of the herpes viruses, and it is difficult to distinguish between those with the disease and healthy individuals, on the basis of serological evidence of exposure to Epstein-Barr or other viruses.

Whether or not HBLV will turn out to be the "cause" of chronic mononucleosis-like syndrome is now under investigation at the National Cancer Institute and by clinicians in Boston, New York, Houston, Fort Lauderdale, Miami, and Tahoe. The more intriguing possibility, however, is that the disease may be the cause of the virus.

The discovery of the AIDS virus has tended to obscure the large body of data on environmental, nutritional, and other factors associated with the development of Acquired Immune Deficiency Syndrome. There is a large body of literature which documents the ability of viruses to recombine in an infected host and produce new viral strains. The ability of the herpes viruses to infect immunosuppressed individuals, and to then produce additional immunosuppression, is well documented.

It may not be entirely fortuitous that we see the emergence of yet another immunosuppressive v rus in areas where AIDS is already a problem, and where the general health environment, sanitary infrastructure, and nutritional level have declined. If this decline continues, the molecular biologists may have many more interesting viruses to write about—assuming they are still around to do the writing.