THE LAST WORD

A CHALLENGE TO THE AIDS ESTABLISHMENT

by Peter Duesberg

Acquired Immune Deficiency Syndrome (AIDS) has become a *cause célèbre* for journals, companies, and scientists, and the "deadly AIDS virus" has been sold to the public as the cause of AIDS with the confidence and authority that is usually derived from absolute scientific proof. The bases of the virus-AIDS *hypothesis* are that this retrovirus was originally isolated from an immune-deficient patient, that 80–90% of AIDS patients have antibody to the virus, and that there is a reasonable correlation between antiviral antibodies and persons in high-risk groups for the disease. The hypothesis is appealing because it appears to fit a 100-year-old tradition of triumphs in medicine that proved viral and bacterial parasites to be the causes of contagious disease. But this appearance is deceptive.

Three criteria need to be met before a virus can be said to function as a pathogen. It must be biochemically active. It must infect or intoxicate more cells than the host can regenerate or spare. And the host must be genetically and immunologically permissive. Yet almost none of the accumulated data on the "Human Immunodeficiency Virus" (HIV) demonstrate that the virus satisfies *any* of these criteria.

HIV is latent and inactive, not only in the 1–2 million Americans who test positive for antibody to the virus, but also in the 10,000 who annually develop AIDS and the 5,000 who die from it. Yet *all* other pathogenic viruses are known to be metabolically active when they cause disease. Latency is the mechanism by which parasites typically survive as passengers in asymptomatic hosts.

HIV also fails to meet the second criterion since it actively infects fewer than .01% of susceptible lymphocytes and since 5% of T cells are regenerated during the 2 days it takes the virus to infect the cell. Moreover, it is truly paradoxical that HIV is said to cause AIDS only after an asymptomatic incubation period of at least 5 years, although antiviral immunity is induced within a few weeks. Ever since Jenner discovered the principle of vaccination, anti-viral immunity (the basis of the AIDS test) has been considered the ultimate weapon against, rather than an indication for, future disease.

Consistent with the presence of antiviral antibody, there is very little direct evidence for the presence of the virus in persons with AIDS. There is not one report of a virus titer from an AIDS patient. Indeed, virus can only be isolated from 50% of symptomatic and asymptomatic carriers, and then only by techniques originally designed to activate latent viruses. The methods are to grow millions of cells in culture, away from the immune system and as yet unknown suppressors of the host, until at least one previously latent virus becomes active. This will then be multiplied by adding uninfected cells until a detectable titer is reached. Thus isolating virus from 50% of AIDS patients implies that 50% carried less than one latent virus in several million cells.

In fact, viral genomes have only been detected in about 15% of persons who test positive for HIV antibody; in these persons, about one proviral genome is found in 100–1000 susceptible lymphocytes. In other words, 85% of antibody-positive persons carry either less provirus than this or none at all. Moreover, most of these proviruses are dormant since only one in 10,000–100,000 susceptible cells express viral RNA in *both* symptomatic and asymptomatic carriers. In contrast, the titers of other known retroviruses are between 10^4 to 10^{12} infectious units per ml of serum or tissue when they function as pathogens.

The virus-AIDS hypothesis also totally fails to explain how the virus depletes T-helper cells, and why it takes at least 3–5 years to do so. Unlike all other animal viruses, retroviruses need mitosis to initiate infection. Moreover, no HIV gene remains inactive during replication, which takes about 1–2 days, as with all other retroviruses. Thus HIV would be expected to kill T cells and cause AIDS when it first infects an organism and not 5 years later when it is biochemically inactive and suppressed by antiviral immunity. The 5-year latency presents proponents of the hypothesis with two bizarre options: either old T cells die 5 years after infection, or the offspring must die in the 50th generation, given a one-month generation time for the average T cell.

Nevertheless, killing of T cells within weeks, not years, after infection has been observed in cell culture—in apparent agreement with the claim that the virus kills T cells. But this type of killing is fundamentally different from the unconditional cell lysis achieved by true cytocidal viruses. It involves cell fusion mediated via HIV antigens on the surface of infected cells and receptors at the surface of uninfected cells, and is conditional on the cells and virus isolates used. Further, it does not occur in chronically infected human T-cell lines that grow indefinitely in culture yet produce more virus than any other system, nor has it ever been observed in blood taken from an AIDS patient. In fact, limited cell killing by fusion is a common feature of retroviruses, none of which have as yet been claimed to cause AIDS.

It seems clear from the foregoing that the virus-AIDS hypothesis fails to make a case for sufficiency. It offers *no* explanation for why less than 1% of antibody-positive persons develop AIDS and why the mean latency between infection and disease is 5 years, whereas antiviral immunity is established in a few weeks. A latent period for pathogenicity that exceeds the latent period for immunity is unambiguous evidence for a co-factor or another causative agent altogether. Finally, the hypothesis cannot support a claim that the virus is even necessary for AIDS in view of the fact that it is barely present and consistently latent even in persons with the disease. Since the transmission of AIDS depends on frequent contacts involving the exchange of *cells*, the case for a viral cause remains open.

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