HIV Causes AIDS

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AIDS, a new disease, was first recognized in 1981, clustered in male homosexuals, intravenous drug abusers, and hemophiliacs in the United States and among sexually active heterosexuals in some countries of equatorial Africa. Human immunodeficiency virus (HIV) was first discovered in 1983 and was definitively linked in 1984 to AIDS patients and to groups whose members were at high risk for developing AIDS. The serological test for antibodies to HIV was developed at this same time and showed that HIV infection in the United States was concentrated in those populations at highest risk for AIDS, namely, male homosexuals, intravenous drug abusers, and hemophiliacs (*1*).

The strongest evidence that HIV causes AIDS comes from prospective epidemiological studies that document the absolute requirement for HIV infection for the development of AIDS. It has been shown for every population group studied in the United States and elsewhere that, in the years following the introduction of HIV and subsequent seroconversion of members of that population, the features characteristic of progressive immunodeficiency emerge in a predictable sequence resulting in clinical AIDS (*2–4*). Furthermore, other epidemiological data show that AIDS and HIV infection are clustered in the same population groups and in specific geographic locations and in time. Numerous studies have shown that in countries with no persons with HIV antibodies there is no AIDS and in countries with many persons with HIV antibodies there is much AIDS (*3*). Additionally, the time of occurrence of AIDS in each country is correlated with the time of introduction of HIV into that country; first HIV is introduced, then AIDS appears.

It is also noteworthy that HIV infection, and not infection with any other infectious agent, is linked to blood transfusion-associated AIDS (*5*). Similarly, in HIV-infected pregnant women, mother-to-child perinatal transmission of HIV occurs approximately 50% of the time, and over 95% of HIV-infected infants develop AIDS by 6 years, while their uninfected siblings never develop AIDS (*3, 6*).

Support for the linkage of HIV infection and AIDS comes as well from the results of public health interventions where interruption of HIV infection almost completely prevented the further appearance of AIDS in blood transfusion recipients (*4*). After the introduction of the HIV antibody screening test in the United States, the transmission of HIV in the blood supply in the United States was reduced from as high as 1 in 1,000 infected units in some high risk areas to less than an estimated 1 in 40,000 units countrywide (*7*). (The recently recognized cases of virus transmission by blood transfusion are due to donors being missed by current antibody screening tests during the window of seroconversion. There is a period of about 4 to 8 weeks in which newly HIV-infected persons are capable of transmitting HIV, but have not yet developed antibodies.) As a result of the decrease in blood transfusion-associated transmission of HIV, the incidence of blood transfusion-associated AIDS among U.S. newborns showed a decline (*4*).

Thirteen of the cases of blood transfusion–associated seroconversion identified since the start of blood bank screening were recently investigated (*7*). In one of these cases, the recipient of one unit of blood was one of a pair of fraternal twins. This baby seroconverted and developed AIDS without any other risk factor. Her twin and her mother received no blood products, developed no HIV antibodies, and remained healthy. The blood donor became HIV seropositive and developed AIDS.

Scientists conclude that a virus causes a disease if the virus is consistently associated with the disease and if disruption of transmission of the virus prevents occurrence of the disease. HIV can be detected by culture in most AIDS patients and by culture or polymerase chain reaction in most HIV seropositive individuals (*8, 9*). Epidemiological data show that transmission of HIV results in AIDS and blocking HIV transmission prevents the occurrence of AIDS. Thus, we conclude that there is overwhelming evidence that HIV causes AIDS.

Knowledge of the cause of a disease (etiology) is important for control of that disease and gives a basis for understanding the pathology of the disease. However, knowing the cause of a disease does not mean that there is complete understanding of its pathology. Discovering the pathogenetic mechanisms of HIV in AIDS is a major focus for research.

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Duesberg's Response to Blattner and Colleagues

Blattner, Gallo, and Temin defend the hypothesis that HIV causes AIDS only with epidemiology and anecdotal clinical cases in which AIDS is correlated with antibody to HIV, but not with active virus. I submit that this is insufficient because such evidence cannot distinguish between HIV and other causes, unless there is also evidence for biochemical activity of HIV in AIDS.

1) My opponents say that "following introduction of HIV in a population . . . immunodeficiency emerges in a predictable sequence." Instead, epidemiological surveys show that the annual incidence of AIDS among persons with antibody to HIV varies

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from almost 0 to over 10%, depending on factors defined by lifestyle, health, gender, and country of residence (see point 8 of my preceding statement). Among antibody-positive Americans the average conversion rate is 1% [10,000 to 20,000 (*1*) of 1 to 2 million (*2, 3*)] but that of certain hemophiliacs (*4*) or male homosexuals (*5*) is 10% or higher. These discrepancies between the epidemiologies of HIV antibody and AIDS indicate that neither HIV nor antibody to it is sufficient to cause AIDS.

2) The argument that HIV, "not . . . any other infectious agent," is linked to AIDS in blood transfusion recipients and in congenitally infected children is presumptuous for several reasons. Blood transfusion does not distinguish between HIV and "any other infectious agent" or blood-borne toxin. Further, it is presumed that the recipient had no risk factors other than HIV during the average of 8 years between HIV transfusion and AIDS symptoms. The transfusion evidence would be more convincing if AIDS appeared soon after a singular transfusion in generally healthy recipients. Transfusion AIDS cases, however, only occur very late after infection and mostly in persons with health risks, such as hemophilia, that are not representative of healthy individuals. Likewise, it is presumptuous to assume that HIV was the cause of AIDS in antibody-positive children, of whom 96% had other health risks, such as mothers who are prostitutes or addicted to intravenously administered drugs or blood transfusions for the treatment of hemophilia or other diseases (*1, 6*). The references to these cases would have been more convincing if antibody-negative controls had been included, having none of "the broad range of clinical diseases . . . and the diversity of signs and symptoms of patients infected with HIV" (*6*).

3) According to authoritative sources, the primary defect of AIDS is a T cell deficiency induced by HIV infection (*3, 7, 8*). Therefore, it comes as a surprise that the primary clinical symptom of the children with AIDS was a B cell, not a T cell, deficiency (*6*). In fact, one of these same sources reports that "to fit observations from children into definitions for adult patients is unwise" (*3*). I wonder whether there is truly any disease that, in the presence of antibody to HIV, would not be called AIDS.

4) They claim that "interruption of HIV infection almost completely prevented the further appearance of blood-transfusion-associated AIDS." However, according to the CDC, transfusion-associated AIDS cases in adults have doubled to 752 cases and pediatric cases tripled to 63 in the year ending May 1988 compared to the previous year (*1*). This happened 3 years after antibody-positive transfusions were reduced 40-fold with the AIDS test (*9*). The steep increase in transfusion AIDS cases despite the great reduction of HIV-contaminated transfusions argues directly against HIV as the cause of AIDS.

5) In addition to the correlation that "in countries with many persons with HIV antibodies there is much AIDS," it is necessary to demonstrate some HIV-specific biochemical activity at the onset of AIDS to prove that HIV causes AIDS. All other viruses and microbes are very active when they cause fatal, degenerative diseases similar to AIDS. There is also abundant genetic evidence that this activity is necessary for pathogenicity. Antibodies are evidence for the absence of an active virus, not a prognosis for future disease or death. Prior claims for etiology without genetic or molecular evidence for activity proved to be some of the most spectacular misdiagnoses in virology: (i) Based on epidemiological evidence, "scientists concluded" that Epstein-Barr virus was the cause of Burkitt's lymphoma—until the first virus-free lymphomas were found (*10*). (ii) On epidemiological grounds, human and bovine retroviruses were believed to cause leukemia after bizarre latent periods of up to 40 years in humans (*11*)—but finding these viruses in billions of normal cells of millions of asymptomatic carriers has cast doubt on this view (*12*). It is scarcely surprising that these leukemias arose from virus-infected cells. Consistent with this view, these "viral" leukemias are clonal and not contagious, behaving like virus-negative leukemias, and the associated "leukemia" viruses are not biochemically active (*12*). (iii) "Slow viruses" were accepted as causes of Alzheimer's, kuru, and Creutzfeldt-Jakob disease (*13*) on the basis of the same kind of epidemiology and transmission evidence used here for HIV—but these viruses have never materialized. These examples illustrate that correlations without evidence for biochemical activity are not sufficient to prove "etiology."

6) I fully support the view that "knowledge of the cause of a disease (etiology) is important for control." Since the cause of AIDS is debatable, the control of AIDS may not be achieved by controlling HIV. This is particularly true for the highly toxic "control" (preventive or therapeutic) of AIDS with azidothymidine (AZT)—AZT is designed to inhibit viral DNA synthesis in persons who have antibodies to a virus that is not synthesizing DNA (*14*).

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