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HIV Is Not the Cause of AIDS

By Peter H. Duesberg

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Human immunodeficiency virus (HIV) is not the cause of AIDS because it fails to meet the postulates of Koch and Henle, as well as six cardinal rules of virology.

1) HIV is in violation of Koch's first postulate because it is not possible to detect free virus (1, 2), provirus (3-5), or viral RNA (4, 6, 7) in all cases of AIDS. Indeed, the Centers for Disease Control (CDC) has established guidelines to diagnose AIDS when all laboratory evidence for HIV is negative (8).

2) In violation of Koch's second postulate, HIV cannot be isolated from 20 to 50% of AIDS cases (1, 9-11). Moreover, "isolation" is very indirect. It depends on activating dormant provirus in millions of susceptible cells propagated in vitro away from the suppressive immune system of the host.

3) In violation of Koch's third postulate, pure HIV does not reproduce AIDS when inoculated into chimpanzees or accidentally into healthy humans (9, 12, 13).

4) In contrast to all pathogenic viruses that cause degenerative diseases, HIV is not biochemically active in the disease syndrome it is named for (14). It actively infects only 1 in 10⁴ to > 10⁵ T cells (4, 6, 7, 15). Under these conditions, HIV cannot account for the loss of T cells, the hallmark of AIDS, even if all infected cells died. This is because during the 2 days it takes HIV to replicate, the body regenerates about 5% of its T cells (16), more than enough to compensate for losses due to HIV.

5) It is paradoxical that HIV is said to cause AIDS only after the onset of antiviral immunity, detected by a positive "AIDS test," because

all other viruses are most pathogenic before immunity. The immunity against HIV is so effective that free virus is undetectable (see point 1), which is why HIV is so hard to transmit (9, 12, 13). The virus would be a plausible cause of AIDS if it were reactivated after an asymptomatic latency, like herpes viruses. However, HIV remains inactive during AIDS. Thus the "AIDS test" identifies effective natural vaccination, the ultimate protection against viral disease.

6) The long and highly variable intervals between the onset of antiviral immunity and AIDS, averaging 8 years, are bizarre for a virus that replicates within 1 to 2 days in tissue culture and induces antiviral immunity within 1 to 2 months after an acute infection (9, 17). Since all genes of HIV are active during replication, AIDS should occur early when HIV is active, not later when it is dormant. Indeed, HIV can cause a mononucleosis-like disease during the acute infection, perhaps its only pathogenic potential (9, 17).

7) Retroviruses are typically not cytotoxic. On the contrary, they often promote cell growth. Therefore, they were long considered the most plausible viral carcinogens (9). Yet HIV, a retrovirus, is said to behave like a cytotoxic virus, causing degenerative disease killing billions of T cells (15, 18). This is said even though T cells grown in culture, which produce much more virus than has ever been observed in AIDS patients, continue to divide (9, 10, 18).

8) It is paradoxical for a virus to have a country-specific host range and a risk group-specific pathology. In the United States, 92% of AIDS patients are male (19), but in Africa AIDS is equally distributed between the sexes, although the virus is thought to have existed in Africa not much longer than in the United States (20). In the United States, the virus is said to cause Kaposi's sarcoma only in homosexuals, mostly Pneumocystis pneumonia in hemophiliacs, and frequently cytomegalovirus disease in children (21). In Africa the same virus is thought to cause slim disease, fever, and diarrhea almost exclusively (22, 23).

9) It is now claimed that at least two viruses, HIV-1 and HIV-2, are capable of causing AIDS, which allegedly first appeared on this planet only a few years ago (20). HIV-1 and HIV-2 differ about 60% in their nucleic acid sequences (24). Since viruses are products of gradual evolution, the proposition that within a few years two viruses capable of causing AIDS could have evolved is highly improbable (25).

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Blattner and Colleagues Respond to Duesberg

Biology is an experimental science, and new biological phenomena are continually being discovered. For example, recently some RNA molecules were shown to act as enzymes, ribozymes, even though biochemistry text books state that all enzymes are proteins. Thus, one cannot conclude that HIV-1 does or does not cause AIDS from Duesberg's "cardinal rules" of virology. In fact, the Henle-Koch postulates of 1840 and 1890 were formulated before the discovery of viruses. They are a useful historical reference point, but were not regarded as rigid criteria by Koch himself and should not be so regarded today (1).

Duesberg's description of the properties of viruses is in error and provides no distinction between knowing the cause of a disease, that is, its etiology, and understanding the pathogenesis of this disease. Duesberg is noted for his discoveries about the viral oncogene src. There is no question that the expression of this gene in chicken fibroblasts results in sarcomas. However, no one can yet explain how the expression by the src oncogene of an altered tyrosine protein kinase results in a cell becoming neoplastic. Similarly, there are many unanswered questions about the pathogenesis of AIDS, but they are not relevant to the conclusion that HIV causes AIDS.

Duesberg presents six (or nine) cardinal rules for viruses. Most are not relevant to the question of etiology and are misleading or wrong about viruses in general and HIV in particular.

1-2) It was formerly true that evidence for the presence of HIV-1 could not be found in all AIDS patients. But the overwhelming seroepidemiologic evidence pointing toward HIV as the cause of AIDS spurred research to improve the sensitivity of the detection methods. Better methods of virus isolation now show that HIV infection is present in essentially all AIDS patients (2).

The CDC definition of AIDS has been revised several times as new knowledge has become available and will undoubtedly be revised again. The 1981 CDC definition of AIDS did not mention HIV, since no strain of HIV was known until 1983. Many cases of AIDS are diagnosed on clinical grounds alone because of the lack of availability or expense of HIV-1 antibody testing or because HIV testing is discouraged in some communities. Thus, rates of confirmation of AIDS cases by HIV testing in the United States vary geographically as reflected in CDC surveillance statistics.

3) It is true that HIV does not cause AIDS in chimpanzees. Most viruses are species-specific in host range and in capacity to produce disease. For example, herpes B virus, yellow fever virus, and dengue virus cause serious diseases in humans, but produce no disease symptoms during infection in many species of monkeys (3). Moreover, a virus closely related to HIV, simian immunodeficiency disease virus or SIV, causes an AIDS-like disease in rhesus macaques, but seldom, if ever, causes immunodeficiency in African Green monkeys (4, 5).

HIV-1 does indeed cause AIDS when inoculated into humans with no underlying medical condition. Accidental needlestick injuries with HIV-contaminated needles have resulted in HIV seroconversion and then clinical AIDS (6).

4) It is true that HIV infects only a small fraction of T cells. However, about 15% of the macrophage and monocyte cells from AIDS patients are positive for a viral protein, p24 (7), and the high concentration of this protein in the blood of AIDS patients indicates virus activity (8). The exact mechanism of CD4 cell depletion in AIDS patients is not known, but several indirect mechanisms are known by which HIV can cause CD4 cell depletion in laboratory studies and could operate in vivo.

5-6) Many viruses are highly pathogenic after evidence of immunity appears. For example, reactivated herpes zoster virus causes shingles, and reactivated herpes simplex virus causes local lesions as well as lethal necrotizing encephalitis; moreover, hepatitis B virus causes chronic active hepatitis, equine infectious anemia virus causes anemia, and visna virus causes central nervous system degeneration after the appearance of specific neutralizing antibodies (3, 9). (The last two viruses are lentiretroviruses as is HIV.) These diseases also can have long and variable latent periods.

7) It is true that some retroviruses, in particular, the highly oncogenic retroviruses of the kind that Duesberg has worked with, are not cytotoxic and promote cell growth. Most retroviruses have no effect on cell growth (9, 10). However, Rous-associated virus-2, spleen

necrosis virus, visna virus, and HIV kill infected cells in culture and can establish a chronic stage of infection in which surviving infected cells continue to divide (11).

8) It was apparently "paradoxical for a virus to have a country-specific host range and a risk group-specific pathology." The properties of HIV resolved this paradox because the distribution of AIDS was found to mirror the distribution of HIV. The nature of the spread of the virus and the type of the AIDS-related clinical syndrome depend on social and environmental factors. Sexually active gay men and parenteral drug abusers were the first conduit for spread of HIV in the United States, whereas in some developing countries of Africa, young heterosexually active men and women were the major focus of spread. It is common for life-style to be a major determinant for the spread of an infectious agent. For example, until a vaccine became available, hepatitis B virus was clustered among the same U.S. populations that are now infected by HIV.

The underlying pathology in AIDS is immune deficiency. The nature of the opportunistic agents that invade the susceptible host is a function of which agents are most prominent in a particular population. For example, in the United States Pneumocystis is most prominent in affluent gay men, while human mycobacterial infections and toxoplasmosis are more frequent in socially disadvantaged Caribbean immigrants. Other agents, such as Cryptococcus, are more prominent in developing countries.

9) It is true that there are two viruses that cause human AIDS, HIV-1 and HIV-2. The origin of these HIVs is an interesting scientific question that is not relevant to whether or not HIV causes AIDS. Since a primate lentiretrovirus also causes an AIDS-like disease in rhesus monkeys, just as a cat lentiretrovirus, feline immunodeficiency virus, causes an AIDS-like disease in cats (12), one can suggest either that there is strong selection among retroviruses for this kind of pathology (13) or that the virus ancestor to HIV already had this property. In favor of the first hypothesis is the existence of feline, murine, and primate AIDS caused by retroviruses in a different subfamily from the lentiretroviruses (14).

In summary, although many questions remain about HIV and AIDS, a huge and continuously growing body of scientific evidence shows that HIV causes AIDS.

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HIV Causes AIDS

W. Blattner, R. C. Gallo, H. M. Temin

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 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 68 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 generic 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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