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AIDS epidemiology: Inconsistencies with human immunodeficiency virus and with infectious disease

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ABSTRACT **The newly defined syndrome AIDS includes 25 unrelated parasitic, neoplastic, and noninfectious indicator diseases. Based on epidemiological correlations, the syndrome is thought to be due to a new, sexually or parenterally transmitted retrovirus termed human immunodeficiency virus (HIV). The following epidemiological data conflict with this hypothesis. (*i*) Noncorrelations exist between HIV and AIDS; for example, the AIDS risks of infected subjects vary >10-fold with their gender or country. Abnormal health risks that are never controlled as independent AIDS causes by AIDS statistics, such as drug addiction and hemophilia, correlate directly with an abnormal incidence of AIDS diseases. Above all, the AIDS diseases occur in all risk groups in the absence of HIV. (*ii*) American AIDS is incompatible with infectious disease, because it is almost exclusively restricted to males (91%), because if it occurs, then only on average 10 years after transfusion of HIV, because specific AIDS diseases are not transmissible among different risk groups, and because unlike a new infectious disease, AIDS has not spread exponentially since the AIDS test was established and AIDS received its current definition in 1987. (*iii*) Epidemiological evidence indicates that HIV is a long-established, perinatally transmitted retrovirus. HIV acts as a marker for American AIDS risks, because it is rare and not transmissible by horizontal contacts other than frequent transfusions, intravenous drugs, and repeated or promiscuous sex. It is concluded that American AIDS is not infectious, and suggested that unidentified, mostly noninfectious pathogens cause AIDS.**

Epidemiology is like a bikini: what is revealed is interesting; what is concealed is crucial.

AIDS is a newly defined syndrome of 25 old parasitic, neoplastic, and noninfectious diseases, including in the United States 53% pneumonia, 19% wasting disease, 13% candidiasis, 11% Kaposi sarcoma, 6% dementia, 3% lymphoma, and 2% tuberculosis (1). These unrelated diseases are grouped together because they are all thought to be indicators of an acquired immunodeficiency (2). In America AIDS is almost completely restricted (91%) to males (1). About 90% of all AIDS patients are 20- to 40-year-olds, 30% are intravenous drugs users and their children, 60% are male homosexuals and some heterosexuals who frequently use oral psychoactive drugs (3–7), and 7% are hemophiliacs and other recipients of transfusions (1).

As of 1982, the Centers for Disease Control (CDC) considered AIDS infectious because it appeared to be transmitted among intravenous drug users and homosexuals by sexual contact or by contaminated blood (8). Among infectious agents, cytomegalovirus and various bacteria were proposed as causes of AIDS (6, 8, 10). In 1983 Montagnier and coworkers (11) suggested lymphadenopathy-associated virus [now termed human immunodeficiency virus (HIV)] and Gallo *et al.* (12) human T-cell leukemia virus (HTLV) as causes of AIDS. However, psychoactive drugs, like aphrodisiac nitrite inhalants ("poppers"), were also proposed as causes for Kaposi sarcoma and pneumonia in homosexuals (3–7, 9).

In April 1984 the Secretary of Health and Human Services announced that HIV was the cause of AIDS, and an antibody test for HIV, termed the AIDS test, was registered as a patent by Gallo and collaborators (13–15). This happened before even one American study on HIV had been published (13). According to this view HIV is a lymphotropic retrovirus that is sexually transmitted (16–20). On average 10–11 years after infection and appearance of neutralizing antibodies, HIV is postulated to cause immunodeficiency by killing billions of T cells (16–21). Only then, indicator diseases are said to develop from which patients die on average within 1 year (21–26). Thus HIV became the first virus for which a positive antibody test is interpreted as an indicator for primary diseases that have yet to come. Antibodies against conventional viruses typically signal protection against disease and those against some persistent viruses also signal a small risk of secondary disease upon virus reactivation (27, 28). Although no retrovirus has ever been shown to be pathogenic in humans (29), HIV is thought to be 50–100% fatal, more than any other human virus (16–21). The novelty of AIDS is postulated to reflect the novelty of HIV. The large variety of indicator diseases are postulated to reflect underlying immunodeficiency, and the almost exclusive concentration of AIDS in 20- to 40-year-olds (1) is postulated to reflect sexual or parenteral transmission of HIV (16–20).

This virus–AIDS hypothesis was accepted by most medical scientists, in particular virologists, by 1986 (16–18, 30). Accordingly, the virus was named HIV by an international committee of retrovirologists (30) and became the only basis for the definition of AIDS: "Regardless of the presence of other causes of immunodeficiency, in the presence of laboratory evidence for HIV, any disease listed . . . indicates a diagnosis of AIDS" (2). AIDS is now diagnosed whenever antibody to HIV is detectable along with any of the 25 indicator diseases, even if no immunodeficiency or opportunistic infections are detected, as in cases of Kaposi sarcoma, lymphoma, dementia, and wasting syndrome (2, 18, 23–26, 31). Moreover, infection in the absence of any clinical symptoms is now termed, and often treated as, "HIV disease" (18). However, all efforts directed by the virus–AIDS hypothesis, for over 2 billion dollars annually, have failed to contain or cure AIDS (32, 33).

Since 1987 I have challenged the virus–AIDS hypothesis because HIV is latent and is present in only 1 out of 500 T cells during AIDS, because retroviruses typically do not kill cells, and because AIDS appears on average 10 years after the virus has been neutralized by antibodies (7, 29, 34). In response to this challenge it was agreed that the hypothesis cannot be defended in terms of orthodox virology, based on known genetic and biochemical properties of HIV (35–47). However, it was claimed that epidemiological evidence supports the

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Abbreviations: CDC, Centers for Disease Control; HIV, human immunodeficiency virus; HTLV, human T-cell leukemia virus.

virus–AIDS hypothesis (35, 36, 38–41, 44–47) and that Gallo (48), Montagnier (editorial comment in ref. 7, p. 5), and possibly others (editorial footnote in ref. 34, p. 755) would prove it. Here this epidemiological evidence is called into question.

Noncorrelations Between AIDS and HIV

AIDS Risks of HIV-Infected Persons Differ 10- to 65-Fold Depending on Their Country. If AIDS is caused by HIV, the ratio of infected to diseased carriers should be similar in different countries. However, in the United States about 10% (or 100,000) (1) of 1 million HIV-positives (16, 18, 49, 50) have developed AIDS since 1985, but in Uganda only 0.8% (or 8000) of 1 million (51), and in Zaire only 0.15% (4636) (52) of 3 million HIV-positives (34). Although AIDS surveillance by some African countries has been questioned, surveillance by Uganda is reported as "highly successful," providing "the highest number . . . of cases . . . in Africa" (51). Since the HIV epidemics of both the United States and Africa are said to be new and to have an African origin (17, 20, 36, 45), but the AIDS risks of infected Americans are 10- to 65-fold higher than those of Africans, country-specific pathogens are necessary for AIDS. Moreover, if AIDS equaled opportunistic infections resulting from immunodeficiency, more, instead of less, AIDS per HIV carrier would be expected in Africa than in the United States.

AIDS Risks Among HIV-Infected Americans Differ About 10-Fold Based on Gender. Since 1985 the U.S. Army has tested 1.15×10^6 male and female 17- to 19-year-old recruits for antibodies to HIV. Every year 0.03% of the males and females in this age group were found to be HIV-positive (53). Although HIV has been distributed equally between the sexes among 17- to 24-year-old Americans for the last 5 years, about 10 times more AIDS cases have occurred in males (1, 53). Ninety-five percent of these were either intravenous drug users or homosexuals (1, 53).

Abnormal Health Risks Correlate Directly with the Incidence of AIDS Diseases. Since 97% of the American AIDS patients come from behavioral or clinical health-risk groups and since the incidence of AIDS indicator diseases in risk-matched, HIV-free control groups is never reported (1), it may be argued that pathogens associated with abnormal lifestyles are causing AIDS (3–7, 32, 34, 54)—particularly because the diseases are risk-specific (see below).

In response to this, it has been pointed out that AIDS occurs outside the major risk groups in Americans and Africans. However, to date only 3900, or about 3%, of all American AIDS cases have come from groups without health risk (1). These represent 25 conventional diseases that occurred in a country of 250 million inhabitants over the last 9 years (1). To determine whether these diseases are due to HIV, their incidence must be compared with that in the normal, HIV-free population. However, this has not been done, because these diseases are only reportable and recorded by the CDC as AIDS if HIV is present (1). Thus there is no controlled epidemiological evidence that HIV is pathogenic in the normal United States population. The same is true for Africa. The cumulative incidence of AIDS from 1986 to 1989 in Uganda was only 8000 (0.8%) out of 1 million HIV-positives (51), or about 0.2% annually. (Most AIDS statistics are cumulative and thus always increasing.) Since >90% of these cases are common African diseases like slim disease, fever, diarrhea, and tuberculosis (34, 51) and their incidence in HIV-free controls is not reported, it is uncertain whether HIV is pathogenic in Africa.

Further, it has been argued based on anecdotal cases that AIDS did not exist prior to HIV in clinical health-risk groups such as (*i*) hemophiliacs, (*ii*) other recipients of transfusions, (*iii*) children of drug-addicted mothers, and (*iv*) drug addicts (1, 18, 35, 36, 38, 44, 45, 47). However, competing causes for AIDS diseases have not been excluded in any of these cases.

(*i*) Transfusions of blood and factor VIII and intrinsic deficiencies associated with hemophilia, acting over the long time periods said to be latent periods of HIV, have all been identified as pathogenic factors for AIDS-like diseases in hemophiliacs (55–61). To determine whether HIV or clinical deficiencies and their treatment cause AIDS in hemophiliacs, either controlled epidemiological studies comparing matched hemophiliacs, with and without HIV, or epidemiological evidence that the mortality of hemophiliacs is increased by HIV would be necessary.

Surprisingly, in view of the many claims that HIV causes AIDS in hemophiliacs, there is not one controlled study that has found HIV to be a health risk. There is also no report from any country that the mortality rate of hemophiliacs has increased due to the infection of hemophiliacs with HIV (59). In fact, it appears to be lower than ever (56, 59). About 75% of the 20,000 severe hemophiliacs in the United States are reported to be HIV-positive at least since 1980 to 1982, owing to the administration of blood or factor VIII (16, 18, 59, 61, 62). Because the administration of plasma fractions prepared from large numbers of donors started in 1965, many hemophiliacs may have been HIV-positive for >10 years now (56, 59). Since 50–100% of HIV-infected persons are postulated to develop AIDS after an average latent period of 10 years (18, 21, 34), at least half of the 15,000 HIV-positive hemophiliacs should have died from AIDS. Yet <2% of the 15,000 HIV-positive hemophiliacs in the United States have died with a diagnosis of AIDS in 1988 and in 1989 (1).

The low annual incidence of AIDS-like diseases among hemophiliacs in the United States is likely to reflect hemophilia-related morbidity and mortality under a new name. Indeed, among American hemophiliacs infected for an average of at least 10 years with HIV, elapsed time as a hemophiliac stands out as the critical determinant for AIDS diseases. The median age of hemophiliac AIDS patients is 34 years, or 14 years higher (59) than that of their asymptomatic peers, which is 20–22 years (16, 59).

(*ii*) The thesis that HIV transfusions cause AIDS in other patients is also entirely uncontrolled (36). Indeed, a controlled study might be difficult because 50% of American patients (other than hemophiliacs) die within 1 year (58) and 60% within 3 years (63) after a transfusion—long before the average 10 years that HIV is said to require for pathogenicity have elapsed. The pathogenic conditions that necessitated the transfusions are obviously deadlier than the hypothetical pathogen HIV.

(*iii*) About 95% of the children with AIDS in the United States were subject to pathogenic conditions other than the putative pathogen HIV, either drug addiction of the mother or deficiencies of their own that required blood transfusions (1). The remainder probably reflects normal morbidity of infants born to healthy mothers who are perinatally (see below) or iatrogenically infected by HIV.

(*iv*) A rare controlled study showed that among 297 asymptomatic, HIV-poistive intravenous drug users the AIDS risk over 16 months was 3 times higher in those who persisted than in those who stopped injecting drugs (95).

Since the incidence of AIDS diseases in non-risk groups appears normal, and since the abnormal incidence of AIDS diseases in risk groups correlates directly with their abnormal health risks, there is no epidemiological evidence that HIV is pathogenic. Although longitudinal studies of selected risk groups claim, some even without published data or references (64), that HIV-positives have more AIDS, none of these studies have controlled the negatives for all health risks and selection biases (18, 47, 62, 65).

AIDS Indicator Diseases Occur Without HIV in All Risk Groups. Tsoukas *et al.* (61) observed severe immunodefi-

ciency in 6 of 14 HIV-free and 9 of 15 HIV-positive hemophiliacs. Ludlam *et al.* (60) described a group of 15 hemophiliacs who had acquired immunodeficiency before they were infected by HIV. Others observed HIV in only 7 of 19 (55) or 10 of 19 (66) hemophiliacs who had very similar immunodeficiencies. However, direct correlations were observed between the degree of immunodeficiency and the amount of clotting factor consumed, in both HIV-negatives and HIV-positives (60, 66).

A prospective study from Canada identified immunodeficiency in 33 of 161 HIV-free homosexual men. Nine of these became subsequently infected by HIV (65). Further, a recent study identified initially 6 and now 12 HIV-free Kaposi sarcoma cases in male American homosexuals, some of which occurred in the absence of immunodeficiency (67, 68). Others recorded the occurrence of Kaposi sarcoma in AIDS risk groups long before AIDS (57). And recently, CDC workers have postulated a Kaposi agent other than HIV, because of the almost exclusive occurrence of Kaposi sarcoma in homosexuals among AIDS risk groups (69). Moreover, there is no trace of HIV even in the Kaposi sarcomas of patients in which antibody to HIV is confirmed (34). Thus HIV is necessary neither for immunodeficiency nor for Kaposi sarcoma in homosexuals, although their association is perceived as the hallmark of AIDS (68, 69).

Among intravenous drug users in New York representing a "spectrum of HIV-related diseases," HIV was not observed in 26 of 50 pneumonia deaths, 15 of 22 endocarditis deaths, and 5 of 16 tuberculosis deaths (70). Likewise, no HIV was observed in 24 of 54 prisoners with tuberculosis in New York State, of whom 47 were street-drug users (71). Similar neurological deficiencies were recently observed in 12 HIV-infected and 16 uninfected infants from drug-addicted mothers (72). In a group of 21 heroin addicts, of whom only 2 were infected by HIV, the ratio of helper to suppressor T cells was found to decline within 13 years from a normal of 2 to <1 (73), which is typical of AIDS (16).

Since all AIDS indicator diseases occur in AIDS risk groups in the absence of HIV, HIV is not a necessary cause for these diseases (except for their diagnosis as AIDS). Current AIDS statistics probably include additional HIV-free cases because HIV was confirmed up to 1989 in only about 73% of all American AIDS cases, and in only 39% of the AIDS cases from New York and 61% from California (74). Moreover, statistics are biased in favor of HIV-positive cases because AIDS is reportable whereas most HIV-free indicator diseases are not (57).

Inconsistencies Between AIDS and Infectious Disease

AIDS Diseases That Are Not Male-Specific Show a 10-Fold Preference for Males. The distribution of conventional sexually transmitted diseases is almost even between the sexes (75). By contrast American AIDS occurs almost exclusively (91%) in males, although none of the 25 AIDS diseases is male-specific (1). However, African AIDS appears largely compatible with infectious diseases that strike without preference for sex, risk, and age groups (17, 18, 51) and that hardly overlap with American AIDS (see below).

Latent Periods of 10 Years Are Not Compatible with Infectious Disease. Viruses, retroviruses, and HIV are immunogenic and biochemically most active within weeks or months after infection (27, 28, 34, 76, 77). There is no precedent for an infectious agent that causes primary diseases on average only years after it is neutralized by antibodies (27, 28, 38, 39). Yet HIV is claimed to cause AIDS on average 10 years after transfusion in adults and only after 2 years in children (18, 34, 62). The diversity of these latent periods is inconsistent with one infectious agent, and their magnitude is characteristic for diseases caused by chronic exposure to toxic substances.

Specific AIDS Diseases Are Not Transmissible Among Different Risk Groups. If AIDS diseases are caused directly by HIV or are due to HIV-induced immunodeficiencies and available parasites, all infected subjects should have the same risk of developing those AIDS diseases that are not associated with group- or region-specific parasites. However, 53% of American AIDS patients have *Pneumocystis* pneumonia and 13% have candidiasis (1), whereas 90% of the African AIDS patients have slim disease, fever, diarrhea, and tuberculosis but not pneumonia and candidiasis (34, 51), although *Pneumocystis carinii* and candida are ubiquitous in humans, including Africans (34, 78, 79). In addition, the AIDS diseases of American children are different from those of adults—namely, 50% pneumonia, 16% wasting disease, 12% dementia, and 20% bacterial infections, which are exclusively diagnosed in children (1). Further, in the United States, Kaposi sarcoma occurs 20 times more often in homosexuals than in hemophiliacs and intravenous drug users (69), often in the absence of immunodeficiency (23–26, 67). Thus, specific AIDS diseases are not transmissible among different risk groups, suggesting that distinct, nontransmissible pathogens must be primary causes.

Unlike New Infectious Diseases, AIDS Does Not Spread Exponentially. AIDS is said to be a new sexually transmitted disease (17, 18, 36, 45). The epidemiological hallmark of a new transmissible disease is that it spreads exponentially until it has saturated a susceptible pool of the population, a process described by Farr's law (80). Therefore AIDS would be expected to increase in the sexually active population at an exponential rate, provided there is at least some promiscuity. Yet since the AIDS test has been established and AIDS was given its currrent definition in 1987 (2), AIDS has spread very slowly, claiming among >100 million sexually active Americans only 20,000–30,000 cases annually (1).

Epidemiological Evidence That HIV Is Not Pathogenic

HIV Is Epidemiologically Not New. Since 1985, when HIV infection became detectable with the "AIDS test," the number of infected Americans has remained constant at about 1 million, or 0.4% of the population (16, 18, 49, 50). Likewise, the percentage of HIV-positive male and female U.S. Army recruits has remained constant at 0.03% for 5 years, although >70% of 17- to 19-year-olds are sexually active and about 50% are promiscuous (53, 62). The strikingly constant incidence of HIV indicates that it is epidemiologically not new in the United States and thus not a plausible cause for the new epidemic AIDS.

HIV Depends on Perinatal Transmission for Survival and Is Thus Not Likely to Be Fatally Pathogenic. Due to the absence of HIV in the semen of 24 of 25 HIV-positive men, with one provirus detected per 10^6 cells (81), and due to the chronic latency and presence of HIV provirus in only 1 of 500 susceptible lymphocytes (34, 82), HIV depends on an average of about 500 sexual contacts to be transmitted (83, 84). Perhaps even more contacts are necessary, because only about 15% of the wives of hemophiliacs are HIV-positive (20), although about 75% of severe hemophiliacs in the United States have been positive for 8–10 years. Therefore it is unlikely that HIV could survive by sexual transmission. Further, it is unlikely to be preferentially transmitted by homosexual males, since about 10% of both males and females frequently practice anal intercourse (62).

Based on animal and human models, retroviruses depend almost exclusively on perinatal transmission for survival. They are very difficult to transmit horizontally by immune competent animals and humans, because they are chronically suppressed, first by maternal antibody and then by the baby's own (76, 77), and possibly also by cellular suppressors (34). Even retroviruses with sarcomagenic or leukemogenic on-

cogenes have never spread horizontally in breeding colonies (29, 85). Therefore, specific strains of mice, chickens, or humans from geographically distinct regions are often marked for generations by distinct strains of perinatally transmitted, latent retroviruses (85, 86). For example, HTLV is endemic in certain islands of Japan and marks specific ethnic groups among mixed populations in the Caribbean (86). Wild animals (29, 85, 86) or humans (42, 43, 86) with an acute retrovirus infection are virtually never observed. Acute retrovirus infections result from experimental infection or horizontal infections among mass-bred animals, typically prior to immune competence with virus strains not covered by maternal antibodies (76, 77, 85).

Since perinatal transmission of HIV is at least 50% efficient (18, 20, 34, 62), and sexual transmission is <0.2% efficient, it appears that HIV, like other retroviruses, depends on perinatal transmission for survival. Therefore, it cannot be fatally pathogenic in most infections within 2–10 years, as postulated by the virus–AIDS hypothesis. This provides the only plausible explanation for the random distribution of HIV in even as few as 0.03% of 17- to 19-year-old healthy Americans (53) and in about 10% of Africans of all ages (31, 34, 49, 51). This explains why no more than 2456 AIDS cases have been recorded among about 75 million Americans under the age of 19 in the last 9 years (1), although at least 0.03%, or 25,000, can be estimated to be perinatally infected (53). It appears that >90% of perinatally infected Americans are asymptomatic for at least 19 years.

Antibody to HIV Is a Marker for American AIDS Risks. American AIDS risk groups and patients are marked by antibodies not only to HIV but also to many other viruses and microbes, such as cytomegalovirus, hepatitis virus, herpes simplex virus, HTLV, parvovirus, Epstein–Barr virus, genital papilloma virus, *Treponema, Neisseria*, amoebae, candida, and mycoplasma (1, 5, 6, 10, 12, 54, 57, 67, 75, 78, 87, 88). Among these, antibodies to HIV and HTLV are perhaps the most specific markers because their prevalence in AIDS patients is 73% (74) and 25% (87), respectively, but only 0.03% (53) and 0.025% (86) in the general United States population.

Because AIDS patients carry antibodies to many more viruses and microbes, in particular, rare ones such as HTLV, than the general population, it is arbitrary to delineate HIV as an etiologic agent of AIDS by the presence or titer of antibody alone. In addition, this hypothesis is inconsistent with the typical sequence of events in which antibodies follow rather than precede viral pathogenicity (27, 38, 39), incompatible with HIV-free indicator diseases, and implausible in the absence of HIV activity (34, 82). As tens of thousands of positive tests for antibody and hundreds of negative tests for free virus have shown (34), HIV remains typically dormant in "T-cell reservoirs" even during AIDS (82). The simultaneous occurrence of HIV viremia and antiviral antibodies was reported in some AIDS patients in 1989, but this observation has not been replicated by others (42, 43). More and more of the AIDS-associated parasites are now named as AIDS cofactors of HIV, most recently HTLV and mycoplasma (45, 88).

A consistent alternative explanation for the high prevalence of antibody to HIV (and other microbes) in AIDS risk groups and AIDS patients proposes that HIV is a marker for American AIDS risks (34). The probability of becoming HIV antibody-positive correlates directly with the frequency of injecting unsterile drugs (34, 62, 70, 89–91), with the frequency of transfusions (59–61), and with promiscuity (62, 65, 89, 92). However, in America, only promiscuity aided by aphrodisiac and psychoactive drugs, practiced mostly by 20- to 40-year-old male homosexuals and some heterosexuals, seems to correlate with AIDS diseases (3–7, 62, 67). HIV would thus be a marker for these drugs and also for the frequent infections by conventional venereal diseases such as gonorrhea and syphilis (5, 6) which are not part of the AIDS definition (2), and for the corresponding therapeutic and prophylactic medications. In fact, HIV was named as a marker for homosexual promiscuity (92) and recently for an "unknown sexually transmitted agent" that is presumed to cause Kaposi sarcoma in male homosexuals (45, 67–69).

However, not all HIV antibody-positives above those expected from perinatal transmission (e.g., 0.03% in the United States) must reflect promiscuity and parenteral infection. Instead, perinatally infected persons may develop antibodies only with age, as latent proviruses become activated by transient immunosuppression or other stimuli. This predicts that the percentage of antibody-positives among provirus-positives increases with age. The lower incidence of antibody to HIV in 1- to 14-year-old Zairen children (1–2%) compared with adults (4–10%) (31) is a case in point. The incidence of antibody to HTLV also increases with age in countries where HTLV is endemic, although HTLV is just as difficult to transmit sexually as HIV (86).

An Alternative Hypothesis

Numerous correlations have linked American AIDS with the consumption of drugs. The CDC reports that 30% are intravenous drug users (1) but does not report that another 50–60% have used oral psychoactive drugs (3–7) and medical drugs, above all the DNA-chain terminator 3′-azido-3′-deoxythymidine (AZT) (7, 34). AZT is currently prescribed to 125,000 sick and healthy HIV-positive persons worldwide, including about 80,000 Americans, based on annual sales of $284 million and a wholesale price of $2200 for a year of AZT at 500 mg/day (Burroughs Wellcome Annual Report 1990 and Office of Public Affairs, personal communication). Therefore it is proposed that either drug consumption (frequently associated with malnutrition) by recently established behavioral groups or conventional clinical deficiencies and their treatments are necessary and sufficient to cause indicator diseases of AIDS. This hypothesis resolves the many paradoxes of the virus–AIDS hypothesis. (*i*) American AIDS is new because of the recent dramatic increase in the consumption of psychoactive and medical drugs (4, 7, 70, 91). For instance, cocaine-related hospital emergencies increased 5-fold from 1984 to 1988 (93). (*ii*) American AIDS is prevalent in 20- to 40-year-old men, although not one AIDS disease is male-specific, and this age group is the least likely to develop any diseases. The reason is that men of this age group consume 80% of hard psychoactive drugs (94). (*iii*) The vastly different AIDS diseases are caused by different pathogens, pathogenic conditions, and their treatments. This also explains "AIDS diseases" that do not depend on immunodeficiency and occur without it, including Kaposi sarcoma, lymphoma, dementia, and wasting disease (1, 2, 23–26, 34, 67). (*iv*) African AIDS would be old diseases caused by malnutrition and parasitic infections under a new name, the reason why it is equally distributed between the sexes (16, 51). (*v*) The long and unpredictable latent periods between infection by HIV and specific AIDS diseases are the product of functionally unrelated events: the pathogenic events necessary to reach an individual's threshold for AIDS diseases, and infection by the marker HIV.

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