

AIDS Acquired by Drug Consumption and Other Noncontagious Risk Factors

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Abstract

The hypothesis that human immunodeficiency virus (HIV) is a new, sexually transmitted virus that causes AIDS has been entirely unproductive in terms of public health benefits. Moreover, it fails to predict the epidemiology of AIDS, the annual AIDS risk and the very heterogeneous AIDS diseases of infected persons. The correct hypothesis must explain why: (1) AIDS includes 25 previously known diseases and two clinically and epidemiologically very different epidemics, one in America and Europe, the other in Africa; (2) almost all American (90%) and European (86%) AIDS patients are males over the age of 20, while African AIDS affects both sexes equally; (3) the annual AIDS risks of infected babies, intravenous drug users, homosexuals who use aphrodisiacs, hemophiliacs and Africans vary over 100-fold; (4) many AIDS patients have diseases that do not depend on immunodeficiency, such as Kaposi's sarcoma, lymphoma, dementia and wasting; (5) the AIDS diseases of Americans (97%) and Europeans (87%) are predetermined by prior health risks, including long-term consumption of illicit recreational drugs, the antiviral drug AZT and congenital deficiencies like hemophilia, and those of Africans are Africa-specific. Both negative and positive evidence shows that AIDS is not infectious: (1) the virus hypothesis fails all conventional criteria of causation; (2) over 100-fold different AIDS risks in different risk groups show that HIV is not sufficient for AIDS; (3) AIDS is only "acquired," if at all, years after HIV

is neutralized by antibodies; (4) AIDS is new but HIV is a long-established, perinatally transmitted retrovirus; (5) alternative explanations disprove all assumptions and anecdotal cases cited in support of the virus hypothesis; (6) all AIDS-defining diseases occur in matched risk groups, at the same rate, in the absence of HIV; (7) there is no common, active microbe in all AIDS patients; (8) AIDS manifests in unpredictable and unrelated diseases; and (9) it does not spread randomly between the sexes in America and Europe. Based on numerous data documenting that drugs are necessary for HIV-positives and sufficient for HIV-negatives to develop AIDS diseases, it is proposed that all American/European AIDS diseases, that exceed their normal background, result from recreational and anti-HIV drugs. African AIDS is proposed to result from protein malnutrition, poor sanitation and subsequent parasitic infections. This hypothesis resolves all paradoxes of the virus-AIDS hypothesis. It is epidemiologically and experimentally testable and provides a rational basis for AIDS control.

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Note Added in Proof

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References

“It’s too late to correct,” said the Red Queen. “When you’ve once said a thing, that fixes it, and you must take the consequences.”

—Lewis Carroll, *Through the Looking Glass*

1. Virus-AIDS Hypothesis Fails to Predict Epidemiology and Pathology of AIDS

At a press conference in April 1984, the American Secretary of Health and Human Services announced that the Acquired Immunodeficiency Syndrome (AIDS) was an infectious disease, caused by a sexually and parenterally transmitted retrovirus, now termed human immunodeficiency virus (HIV). The announcement predicted an antiviral vaccine within two years (Connor, 1987; Adams, 1989; Farber, 1992; Hodgkinson, 1992).

However, the hypothesis has been a complete failure in terms of public health benefits. Despite unprecedented efforts in research and health care, the hypothesis has failed to generate the promised vaccine, and it has failed to develop a cure (Thompson, 1990; Savitz, 1991; Duesberg, 1992b; Waldholz, 1992). The U.S. Government alone spends annually about \$1 billion for AIDS research and about \$3 billion for AIDS-related health care (National Center for Health Statistics, 1992). The situation has become so desperate that the director for AIDS research at the National Institutes of Health (NIH) promotes via press release, eight years after HIV was declared the cause of AIDS, an as yet unedited paper, which has no more to offer than a renewed effort at causing AIDS in monkeys: “The best possible situation would be to have a human virus [HIV] that infects monkeys” (Steinbrook, 1992). This is said nine years after the NIH first started infecting chimpanzees with HIV—over 150 so far at a cost of \$40,000–50,000 apiece—all of which are still healthy (Hilts, 1992; Steinbrook, 1992) (Section 3.3 and Jorg Eichberg, personal communication).

Moreover, the virus-AIDS hypothesis has failed completely to predict the course of the epidemic (Institute of Medicine, 1988; Duesberg, 1989c, 1991a; Duesberg and Ellison, 1990; Thompson, 1990; Savitz, 1991). For example, the NIH and others have predicted that AIDS would “explode” into the general population (Shorter, 1987; Anderson and May, 1992) and the Global AIDS Policy Coalition from Harvard’s International AIDS Center declared in June 1992, “The pandemic is dynamic, volatile and unstable. . . . An explosion of HIV has recently occurred in Southeast Asia, in Thailand . . .” (Mann and the Global AIDS Policy Coalition, 1992). But despite widespread alarm the “general population” has been spared from AIDS, although there is a general increase in unwanted pregnancies and conventional venereal diseases (Institute of Medicine, 1988; Aral and Holmes, 1991). Instead, American and European AIDS has spread, during the last 10 years, steadily but almost exclusively among intravenous drug users and male homosexuals who were heavy users of sexual stimulants and had hundreds of sexual partners (Sections 2.1.3, 3.3.4 and 4.3.2).

The hypothesis even fails to predict the AIDS diseases an infected person may develop and whether and when an HIV-infected person is to develop either diarrhea or dementia, Kaposi’s sarcoma or pneumonia (Grimshaw, 1987; Albonico, 1991a,b). In addition the hypothesis fails to explain why the annual AIDS risks differ over 100-fold between different HIV-infected risk groups, i.e. recipients of transfusions, babies born to drug-addicted mothers, American/European homosexuals, intravenous drug users, hemophiliacs and Africans (Section 3.4.4).

Clearly a correct medical hypothesis might not produce a cure or the prevention of a disease, as for example theories on cancer or sickle-cell anemia. However, a correct medical hypothesis must be able to (1) identify those at risk for a disease, (2) predict the kind of disease a person infected or affected by its putative cause will get, (3) predict how soon disease will follow its putative cause and (4) lead to a determination of how the putative agent causes the disease. Since this is not true for the virus-AIDS hypothesis, this hypothesis must be fundamentally flawed. Further, it seems particularly odd that an AIDS vaccine cannot be developed, since HIV induces highly effective virus-neutralizing

antibodies within weeks after infection (Clark *et al.*, 1991; Daar *et al.*, 1991). These are the same antibodies that are detected by the widely used “AIDS-test” (Institute of Medicine, 1986; Duesberg, 1989c; Rubinstein, 1990).

In view of this, AIDS is subjected here to a critical analysis aimed at identifying a cause that can correctly predict its epidemiology, pathology and progression.

2. Definition of AIDS

2.1. AIDS: 2 Epidemics, Sub-Epidemics and 25 Epidemic-Specific Diseases

AIDS includes 25 previously known diseases and two clinically and epidemiologically very different AIDS epidemics, one in America and Europe, the other in Africa (Table 1) (Centers for Disease Control, 1987; Institute of Medicine, 1988; World Health Organization, 1992a). The American/European epidemic falls into four sub-epidemics: the male homosexual, the intravenous drug user, the hemophilia and the transfusion recipient epidemics (Table 1).

2.1.1. The Epidemics by Case Numbers, Gender and Age

The American/European AIDS epidemics of homosexuals and intravenous drug users are new, starting with drug-using homosexual AIDS patients in Los Angeles and New York in 1981 (Centers for Disease Control, 1981; Gottlieb *et al.*, 1981; Jaffe *et al.*, 1983a). By December 1991, 206,392 AIDS cases had been recorded in the U.S. and 65,979 in Europe (Table 1) (World Health Organization, 1992a; Centers for Disease Control, 1992b). The U.S. has reported about 30,000—40,000 new cases annually since 1987, and Europe reports about 12,000—16,000 cases annually (World Health Organization, 1992a; Centers for Disease Control, 1992b).

Remarkably for a presumably infectious disease, 90% of all American and 86% of all European AIDS patients are males. Nearly all American (98%) and European (96%) AIDS patients are over 20 years old; the remaining 2% and 4%, respectively, are mostly infants (Table 1) (World Health Organization, 1992a; Centers for Disease Control, 1992b).

Table 1. AIDS Statistics*

Epidemics	American	European	African
AIDS total 1985–1991	206,000	66,000	129,000
AIDS annual since 1990	30–40,000	12–16,000	~20,000
HIV carriers since 1985	1 million	500,000	6 million
Annual AIDS per HIV carrier	3–4%	3%	about 0.3%
AIDS by sex	90% male	86% male	50% male
AIDS by age, over 20 years	98%	96%	?
AIDS by risk group			
male homosexual	62%	48%	
intravenous drugs	32%	33%	
transfusions	2%	3%	
hemophiliacs	1%	3%	
general population	3%	13%	100%
AIDS by Disease:			
Microbial	50% <i>Pneumocystis</i> pneumonia 17% candidiasis 11% mycobacterial disease including 3% tuberculosis 5% toxoplasmosis 8% cytomegalovirus 4% herpes virus	75% opportunistic infections	fever diarrhea tuberculosis slim disease
Microbial total	62% (sum > 62% due to overlap)	75%	about 90%
Non-Microbial	19% wasting 10% Kaposi's 6% dementia 3% lymphoma	5% wasting 12% Kaposi's 5% dementia 3% lymphoma	
Non-microbial total	38%	25%	

*Data from references cited in Section 2. There are small ($\pm 1\%$) discrepancies between some numbers cited here and the most recent surveys cited in the text, because some calculations are based on previous surveys.

There is very little AIDS among teenagers, as only 789 American teenagers have developed AIDS over the last 10 years, including 160 in 1991 and 170 in 1990 (Centers for Disease Control, 1992b).

Since 1985, 129,066 AIDS cases have been recorded in Africa (World Health Organization, 1992b), mainly from the people of Central Africa (Blattner, 1991). Unlike the American and European cases, the African cases are distributed equally between the sexes (Quinn *et al.*, 1986; Blattner *et al.*, 1988; Institute of Medicine, 1988; Piot *et al.*, 1988; Goodgame, 1990) and range “in age from 8 to 85 years” (Widy-Wirski *et al.*, 1988).

An AIDS crisis that was reported to “loom” in Thailand as of 1990 (Anderson, 1990; Smith, 1990) and that was predicted to “explode” now (Mann and the Global AIDS Policy Coalition, 1992) has generated only 123 AIDS patients from 1984 until June 1991 (Weniger *et al.*, 1991).

2.1.2. *AIDS Diseases*

The majority of American (62%) and European (75%) AIDS patients have microbial diseases or opportunistic infections that result from a previously acquired immunodeficiency (World Health Organization, 1992a; Centers for Disease Control, 1992b). In America these include *Pneumocystis pneumonia* (50%), candidiasis (17%) and mycobacterial infections such as tuberculosis (11%), toxoplasmosis (5%), cytomegalovirus (8%) and herpes virus disease (4%) (Table 1) (Centers for Disease Control, 1992b). *Pneumocystis pneumonia* is often described and perceived as an AIDS-specific pneumonia. However, *Pneumocystis carinii* is a ubiquitous fungal parasite that is present in all humans and may become active upon immune deficiency like many others (Freeman, 1979; Pifer, 1984; Williford Pifer *et al.*, 1988; Root-Bernstein, 1990a). Since bacterial opportunists of immune deficiency, like tuberculosis bacillus or pneumococcus, are readily defeated with antibiotics, fungal and viral pneumonias predominate in countries where antibiotics are readily available. This is particularly true for risk groups that use antibiotics chronically as AIDS prophylaxis (Callen, 1990; Bardach, 1992). Indeed, young rats treated for several weeks simultaneously with antibiotics and immunosuppressive cortisone all developed *Pneumocystis pneumonia* spontaneously (Weller, 1955).

Contrary to its name, AIDS of many American (38%) and European (25%) patients does not result from immunodeficiency and microbes (Section 3.5.8). Instead, these patients suffer dementia (6%/5%), wasting disease (19%/5%), Kaposi's sarcoma (10%/12%) and lymphoma (3%/3%) (Table 1) (World Health Organization, 1992a; Centers for Disease Control, 1992b).

The African epidemic includes diseases that have been long established in Africa, such as fever, diarrhea, tuberculosis and "slim disease" (Table 1) (Colebunders *et al.*, 1987; Konotey-Ahulu, 1987; Pallangyo *et al.*, 1987; Berkley *et al.*, 1989; Evans, 1989a; Goodgame, 1990; De Cock *et al.*, 1991; Gilks, 1991). Only about 1% are Kaposi's sarcomas (Widy-Wirski *et al.*, 1988). The African AIDS definition is based primarily on these Africa-specific diseases (Widy-Wirski *et al.*, 1988) "because of limited facilities for diagnosing HIV infection" (De Cock *et al.*, 1991).

2.1.3. AIDS Risk Groups and Risk-group Specific AIDS Diseases

Almost all American (97%) and European (87%) AIDS patients come from abnormal health risk groups whose health had been severely compromised prior to the onset of AIDS: 62% of American (47% of European) AIDS patients are male homosexuals who have frequently used oral aphrodisiac drugs (Section 4), 32% (33%) are intravenous drug users, 2% (3%) are critically ill recipients of transfusions and 1% (3%) are hemophiliacs (Institute of Medicine, 1988; Brenner *et al.*, 1990; Centers for Disease Control, 1992b; World Health Organization, 1992a). About 38% of the American teenage AIDS cases are hemophiliacs and recipients of transfusions, 25% are intravenous drug users or sexual partners of intravenous drug users and 25% are male homosexuals (Centers for Disease Control, 1992b). Approximately 70% of the American babies with AIDS are born to drug-addicted mothers ("crack babies") and 13% are born with congenital deficiencies like hemophilia (Centers for Disease Control, 1992b). Only 3% of the American and 13% of the European AIDS patients are from "undetermined exposure categories," i.e. from the general population (Table 1) (World Health Organization, 1992a; Centers for Disease Control, 1992b). Some of the differences between European and American statistics may reflect dif-

ferences in national AIDS standards between different European countries and the U.S. and differences in reporting between the World Health Organization (WHO) and the American Centers for Disease Control (CDC) (World Health Organization, 1992a). By contrast to the American and European AIDS epidemics, African AIDS does not claim its victims from sexual, behavioral or clinical risk groups.

The AIDS epidemics of different risk groups present highly characteristic, country-specific and sub-epidemic-specific AIDS diseases (Table 1 and Table 2):

1. About 90% of the AIDS diseases from Africa are old African diseases that are very different from those of the American/European epidemic (Section 2.1.2, Table 1). The African diseases do not include *Pneumocystis* pneumonia and candidiasis (Goodgame, 1990), although *Pneumocystis* and *Candida* are ubiquitous microbes in all humans including Africans (Freeman, 1979; Pifer, 1984).
2. The American/European epidemic falls into several sub-epidemics based on sub-epidemic-specific diseases:
 - a) American homosexuals have Kaposi's sarcoma 20 times more often than all other American AIDS patients (Selik *et al.*, 1987; Beral *et al.*, 1990).
 - b) Intravenous drug users have a proclivity for tuberculosis (Sections 4.5 and 4.6).
 - c) "Crack" (cocaine) smokers exhibit pneumonia and tuberculosis (Sections 3.4.5 and 4.6).
 - d) Ninety-nine percent of all hemophiliacs with AIDS have opportunistic infections, of which about 70% are fungal and viral pneumonias, but less than 1% have Kaposi's sarcoma (Evatt *et al.*, 1984; Centers for Disease Control, 1986; Selik *et al.*, 1987; Koerper, 1989).
 - e) Nearly all recipients of transfusions have pneumonia (Curran *et al.*, 1984; Selik *et al.*, 1987).

- f) HIV-positive wives of hemophiliacs exhibit only pneumonia and a few other AIDS-defining opportunistic infections (Section 3.4.4.5).
 - g) American babies exclusively have bacterial diseases (18%) and a high rate of dementia (14%) compared to adults (6%) (Table 1) (Centers for Disease Control, 1992b).
 - h) Users of the cytotoxic DNA chain terminator AZT, prescribed to inhibit HIV, develop anemia, leukopenia and nausea (Section 4.6.2).
3. The Thai mini-epidemic of 123 is made up of intravenous drug users (20%), heterosexual male and female “sex workers” (50%) and male homosexuals (30%) (Weniger *et al.*, 1991). Among the Thais 24% have tuberculosis, 22% have pneumonia and other opportunistic infections common in Thailand and 10% have had septicemia, which is indicative of intravenous drug consumption (Weniger *et al.*, 1991).

2.2. The HIV-AIDS Hypothesis, or the Definition of AIDS

Based on epidemiological data collected between 1981 and 1983, AIDS researchers from the CDC (Centers for Disease Control, 1986) “found in gay culture—particularly in its perceived “extreme” and “non-normative” aspects (that is “promiscuity” and recreational drugs)—the crucial clue to the cause of the new syndrome” (Oppenheimer, 1992). Accordingly the CDC had initially favored a “lifestyle” hypothesis for AIDS.

However, by 1983 immunodeficiency was also recorded in hemophiliacs, some women and intravenous drug users. Therefore, the CDC adopted the “hepatitis B analogy” (Oppenheimer, 1992) and re-interpreted AIDS as a new viral disease, transmitted sexually and parenterally by blood products and needles shared for the injection of intravenous drugs (Francis *et al.*, 1983; Jaffe *et al.*, 1983b; Centers for Disease Control, 1986; Oppenheimer, 1992). In April 1984 the American Secretary of Health and Human Services and the virus researcher Robert Gallo announced at a press conference that the new AIDS virus was found.

The announcement was made, and a test for antibody against the virus—termed the “AIDS test”—was registered for a patent, before even one American study had been published on this virus (Connor, 1987; Adams, 1989; Crewdson, 1989; Culliton, 1990; Rubinstein, 1990). Since then most medical scientists have believed that AIDS is infectious, spread by the transmission of HIV.

According to the virus-AIDS hypothesis the 25 different AIDS diseases and the very different AIDS epidemics and sub-epidemics are all held together by a single common cause, HIV. There are two strains of HIV that are 50% related, HIV-1 and HIV-2. But as yet only one American-born AIDS patient has been infected by HIV-2 (O’Brien *et al.*, 1992). Since nearly all HIV-positive AIDS cases recorded to date are infected by HIV-1, this strain will be referred to as HIV in this article. The HIV-AIDS hypothesis proposes: (a) that HIV is a sexually, parenterally and perinatally transmitted virus, (b) that it causes immunodeficiency by killing T-cells, but on average only 10 years after infection in adults and two years after infection in infants—a period that is described as the “latent period of HIV” because the virus is assumed to become reactivated in AIDS—and (c) that all AIDS diseases are consequences of this immunodeficiency (Coffin *et al.*, 1986; Institute of Medicine, 1986, 1988; Gallo, 1987; Blattner *et al.*, 1988; Gallo and Montagnier, 1988; Lemp *et al.*, 1990; Weiss and Jaffe, 1990; Blattner, 1991; Goudsmit, 1992).

Because of this belief, 25 previously known, and in part entirely unrelated diseases have been redefined as AIDS, provided they occur in the presence of HIV. HIV is in practice only detectable indirectly via antiviral antibodies, because of its chronic inactivity even in AIDS patients (Section 3.3). These antibodies are identified with disrupted HIV, a procedure that is termed the “AIDS test” (Institute of Medicine, 1986; Rubinstein, 1990). Virus isolation is a very inefficient and expensive procedure, designed to activate dormant virus from leukocytes. It depends on the activation of a single, latent HIV from about 5 million leukocytes from an antibody-positive person. For this purpose the cells must be propagated *in vitro* away from the virus-suppressing immune system of the host. Virus may then be detected weeks later in the culture medium (Weiss *et al.*, 1988; Duesberg, 1989c).

Antibodies against HIV were originally claimed to be present in most (88%) AIDS patients (Sarngadharan *et al.*, 1984), but have since been confirmed in no more than about 50% of the American AIDS patients (Institute of Medicine, 1988; Selik *et al.*, 1990). The rest are presumptively diagnosed based on disease criteria outlined by the CDC (Centers for Disease Control, 1987; Institute of Medicine, 1988). Because of confidentiality laws more tests are probably done than are reported to the CDC.

Since the "AIDS test" became available in 1985, over 20 million tests have been performed annually in the U.S. alone on blood donors, servicemen and applicants to the Army, AIDS patients and many others, and millions more are performed in Europe, Russia, Africa and other countries (Section 3.6). On the basis of such widespread testing, clearly the most comprehensive in the history of virology, about 1 million, or 0.4% of mostly healthy Americans (Curran *et al.*, 1985; Institute of Medicine, 1988; Duesberg, 1991a; Vermund, 1991; Centers for Disease Control, 1992a), 0.5 million, or 0.2% of Western Europeans (Mann *et al.*, 1988; Blattner, 1991; World Health Organization, 1992a), 6 million, or 10% of mostly healthy Central Africans (Curran *et al.*, 1985; Institute of Medicine, 1988; Piot *et al.*, 1988; Goodgame, 1990; Blattner, 1991; Anderson and May, 1992) and 300,000 or 0.5% of healthy Thais (Weniger *et al.*, 1991) are estimated to carry antibodies to HIV (Table 1). According to the CDC the incidence of HIV-2 is "relatively high" in Western Africa with a record of 9% in one community, but "exceedingly low" in the U.S. where not even one infection was detected among 31,630 blood donors (O'Brien *et al.*, 1992).

2.3. Alternative Infectious Theories of AIDS

In view of the heterogeneity of the AIDS diseases and the difficulties in reducing them to a common, active microbe, several investigators have proposed that AIDS is caused by a multiplicity of infectious agents such as viruses and microbes, or combinations of HIV with other microbes (Sonnabend *et al.*, 1983; Konotey-Ahulu, 1987, 1989; Stewart, 1989; Cotton, 1990; Goldsmith, 1990; Lemaitre *et al.*, 1990; Root-Bernstein, 1990a,c; Balter, 1991; Lo *et al.*, 1991).

However, the proponents of infectious AIDS who reject HIV as the

sole cause or see it as one of several causes of AIDS have failed to establish a consistent alternative to or cofactor for HIV. Instead, they typically blame AIDS on viruses and microbes that are widespread and either harmless or not life-threatening to a normal immune system, such as *Pneumocystis*, cytomegalovirus, herpes virus, hepatitis virus, tuberculosis bacillus, *Candida*, mycoplasma, treponema, gonococci, toxoplasma and cryptosporidiae (Section 3.5.7) (Freeman, 1979; Mims and White, 1984; Pifer, 1984; Evans, 1989c; Mills and Masur, 1990; Bardach, 1992). Since such microbes are more commonly active in AIDS patients than in others, they argue that either chronic or repeated infections by such microbes would generate fatal AIDS (Sonnabend *et al.*, 1983; Stewart, 1989; Mills and Masur, 1990; Root-Bernstein, 1990a,c).

Yet all of these microbes also infect people with normal immune systems either chronically or repeatedly without causing AIDS (Freeman, 1979; Mims and White, 1984; Evans, 1989c; Mills and Masur, 1990). It follows that pathogenicity by these microbes in AIDS patients is a consequence of immunodeficiency acquired by other causes (Duesberg, 1990c, 1991a). This is why most of these infections are termed opportunistic.

3. Discrepancies Between AIDS and Infectious Disease

3.1. Criteria of Infectious and Noninfectious Disease

The correct hypothesis explaining the cause of AIDS must predict the fundamental differences between the two main AIDS epidemics and the bewildering heterogeneity of the 25 AIDS diseases. In addition, the cause of American/European AIDS should make clear why—in an era of ever-improving health parameters, population growth and decreasing mortality (The Software Toolworks World Atlas™, 1992; Anderson and May, 1992)—suddenly a subgroup of mostly 20- to 45-year-old males would die from diverse microbial and nonmicrobial diseases. The mortality from all infectious diseases combined has been reduced to less than 1% in the Western World (Cairns, 1978) through advanced sanitation and nutrition (Section 6) (McKeown, 1979; Moberg and Cohn, 1991; Oppenheimer, 1992). Further, 20- to 45-year-olds are the

least likely to die from any disease (Mims and White, 1984). Their relative immunity to all diseases is why they are recruited as soldiers. The correct AIDS hypothesis would also have to explain why only a small group of about 20,000 Africans have developed AIDS diseases annually since 1985 (Table 1), during a time in which Central Africa enjoyed the fastest population growth in the world, i.e. 3% (The Software Toolworks World Atlas™, 1992).

The sudden appearance of AIDS could signal a new microbe, i.e. infectious AIDS. Yet the suddenness of AIDS could just as well signal one or several new toxins, such as the many new psychoactive drugs that have become popular in America and Europe since the Vietnam War (Section 4).

Based on common characteristics of all orthodox infectious diseases, infectious AIDS would be predicted to:

(1) Spread randomly between the sexes. This is just as true for venereal as for other infectious diseases (Judson *et al.*, 1980; Haverkos, 1990).

(2) Cause primary disease within weeks or months after infection, because infectious agents multiply exponentially in susceptible hosts until stopped by immunity. They are self-replicating, and thus fast acting toxins. (Although “slow” viruses are thought to be pathogenic long after neutralization by antiviral immunity (Evans, 1989c), slow pathogenicity by a neutralized virus has never been experimentally proven (Section 6.1).)

(3) Coincide with a common, active and abundant microbe in all cases of the same disease. (Inactive microbes or microbes at low concentrations are harmless passengers, e.g. lysogenic bacteriophages, endogenous and latent retroviruses (Weiss *et al.*, 1985), latent herpes virus or latent ubiquitous *Pneumocystis* and *Candida* infections (Freeman, 1979; Pifer, 1984; Williford Pifer *et al.*, 1988). Hibernation is a proven microbial strategy of survival, which allows indefinite coexistence with the host without pathogenicity.)

(4) Coincides with a microbe that lyses or renders nonfunctional more cells than the host can spare or regenerate.

(5) Generate a predictable pattern of symptoms.

By contrast non-infectious AIDS, caused by toxins, would be predicted to:

(1) Spread nonrandomly, according to exposure to toxins. For example, lung cancer and emphysema were observed much more frequently in men than in women 20 years ago, because men consumed much more tobacco than women 30–40 years ago (Cairns, 1978).

(2) Follow intoxication after variable intervals as determined by life-time dosage and personal thresholds for disease. These intervals would be considerably longer than those between microbes and disease, because microbes are self-replicating toxins. For example, lung cancer and emphysema are “acquired” only after 10–20 years of smoking, and liver cirrhosis is “acquired” only after 10–20 years of alcoholism.

(3) Manifest toxin-specific and intoxication site-specific diseases, e.g. cigarettes causing lung cancer and alcohol causing liver cirrhosis.

3.2. AIDS Not Compatible with Infectious Disease

All direct parameters of AIDS are incompatible with classical criteria of infectious disease:

(1) Unlike conventional infectious diseases, including venereal diseases (Judson *et al.*, 1980), American/European AIDS is nonrandomly (90%) restricted to males, although no AIDS disease is male-specific (Table 1).

(2) The long and unpredictable intervals between infection and “acquiring” primary AIDS symptoms—averaging two years in infants and 10 years in adults, and termed “latent periods of HIV”—stand in sharp contrast to the short intervals of days or weeks between infection and primary disease observed with all classical viruses, including retroviruses (Duesberg, 1987; Duesberg and Schwartz, 1992). These short intervals reflect the time periods, that all exponentially growing microbes with generation times of half-hours, and viruses including HIV (Clark *et al.*, 1991; Daar *et al.*, 1991) with generation times of 8–48 hr need to reach immunogenic and thus potentially pathogenic concentrations (Fenner *et al.*, 1974; Freeman, 1979; Mims and White, 1984). Once stopped by immunity, conventional viruses and microbes are no longer

pathogenic. Thus long latent periods between immunity against a microbe and a given disease are incompatible with conventional microbial causes, including HIV (Section 3.5.14). The discrepancy of eight years between the hypothetical “latent periods of HIV” in infants and adults presents a secondary paradox.

Nevertheless, HIV could possibly play a role in AIDS if it were consistently reactivated by an “acquired immunodeficiency”—10 years after it was neutralized by antibodies (Section 3.4.2)—just as *Candida*, *Pneumocystis* and cytomegalovirus play roles in AIDS if they are activated by “acquired immunodeficiency.” However, HIV is nearly always inactive even during acquired immunodeficiency (Sections 3.3.1 and 3.5.6). In the absence of HIV reactivation during AIDS, long hypothetical latent periods are simply statistical artifacts. They are conceived to link HIV with AIDS and to buy time for the real causes of AIDS to generate AIDS-defining diseases.

(3) There is no active microbe common to all AIDS patients, and no common group of target cells are lysed or rendered nonfunctional (Sections 3.3 and 3.5.10).

(4) There is no common, predictable pattern of AIDS symptoms in patients of different risk groups. Instead, different risk groups have characteristic AIDS diseases (Sections 2.1.3, 3.4.4 and 3.4.5).

Thus AIDS does not meet even one of the classical criteria of infectious disease. In a recent response to these arguments, Goudsmit, a proponent of the HIV-AIDS hypothesis, confirmed that “AIDS does not have the characteristics of an ordinary infectious disease. This view is incontrovertible” (Goudsmit, 1992). Likewise, the epidemiologists Eggers and Weyer conclude that “the spread of AIDS does not behave like the spread of a disease that is caused by a single sexually transmitted agent” (Eggers and Weyer, 1991) and hence have “simulated a cofactor [that] cannot be identified with any known infectious agent” (Weyer and Eggers, 1990). Anderson and May (1992) had to invent “assortative scenarios” for different AIDS risk groups to reconcile AIDS with infectious disease. Indeed, AIDS would never have been accepted as infectious without the numerous unique assumptions that have been made to accommodate HIV as its cause (Sections 3.5 and 6.1).

3.3. No Proof for the Virus-AIDS Hypothesis

Despite research efforts that exceed those on all other viruses combined and have generated over 60,000 papers on HIV (Christensen, 1991), it has not been possible to prove that HIV causes AIDS. These staggering statistics illustrate that the virus-AIDS hypothesis is either not provable or is very difficult to prove.

Proof for pathogenicity of a virus depends either on (1) meeting Koch's classical postulates, (2) preventing pathogenicity through vaccination, (3) curing disease with antiviral drugs or (4) preventing disease by preventing infection. However, the HIV-hypothesis fails all of these criteria.

3.3.1. Virus Hypothesis Fails to Meet Koch's Postulates

Koch's postulates may be summarized as follows: (i) the agent occurs in each case of a disease and in amounts sufficient to cause pathological effects; (ii) the agent is not found in other diseases; and (iii) after isolation and propagation in culture, the agent can induce the disease anew (Merriam-Webster, 1965; Weiss and Jaffe, 1990).

But:

(i) HIV is certainly not present in all AIDS patients, and even antibody against HIV is not found in all patients who have AIDS-defining diseases. HIV is not even present in all persons who die from multiple indicator-diseases plus general immune system failure—the paradigm AIDS cases (Sections 3.4 and 4.5). In addition, HIV is never present “in amounts sufficient to cause pathological effects” based on the following evidence:

(1) On average only 1 in 500 to 3000 T-cells, or 1 in 1500 to 8000 leukocytes of AIDS patients are infected by HIV (Schnittman *et al.*, 1989; Simmonds *et al.*, 1990). (About 35% of leukocytes are T-cells (Walton *et al.*, 1986).) A recent study, relying on in situ amplification of a proviral HIV DNA fragment with the polymerase chain reaction, detects HIV DNA in 1 of 10 to 1 of 1000 leukocytes of AIDS patients. However, the authors acknowledge that the *in situ* method cannot distinguish between intact and defective proviruses and may include false-

positives, because it does not characterize the amplified DNA products (Bagasra *et al.*, 1992). Indeed the presence of 1 provirus per 10 or even 100 cells is exceptional in AIDS patients. This is why direct hybridization with viral DNA, a technique that is capable of seeing 1 provirus per 10 to 100 cells, typically fails to detect HIV DNA in AIDS patients (Duesberg, 1989c). According to one study, "The most striking feature . . . is the extremely low level of HIV provirus present in circulating PBMCs (peripheral blood mononuclear cells) in most cases" (Simmonds *et al.*, 1990).

Since on average only 0.1% (1 out of 500 to 3000) of T-cells are ever infected by HIV in AIDS patients, but at least 3% of all T-cells are regenerated (Sprent, 1977; Guyton, 1987) during the two days it takes a retrovirus to infect a cell (Duesberg, 1989c), HIV could never kill enough T-cells to cause immunodeficiency. Thus even if HIV killed every infected T-cell (Section 3.5.10), it could deplete T-cells only at 1/30 of their normal rate of regeneration, let alone activated regeneration. The odds of HIV causing T-cell deficiency would be the same as those of a bicycle rider trying to catch up with a jet airplane.

(2) It is also inconsistent with a common pathogenic mechanism that the fraction of HIV-infected leukocytes in patients with the same AIDS diseases varies 30- to 100-fold. One study reports that the fraction of infected cells ranges from 1 in 900 to 1 in 30,000 (Simmonds *et al.*, 1990), and another reports that it ranges from 1 in 10 to 1 in 1000 (Bagasra *et al.*, 1992). In all conventional viral diseases the degree of pathogenicity is directly proportional to the number of infected cells.

(3) It is entirely inconsistent with HIV-mediated pathogenicity that there are over 40-times more HIV-infected leukocytes in many healthy HIV carriers than in AIDS patients with fatal AIDS (Simmonds *et al.*, 1990; Bagasra *et al.*, 1992). Simmonds *et al.* report that there are from 1 in 700 to 1 in 83,000 HIV-infected leukocytes in healthy HIV carriers and from 1 in 900 to 1 in 30,000 in AIDS patients. Bagasra *et al.* report that there are from 1 in 30 to 1 in 1000 infected leukocytes in healthy carriers and from 1 in 10 to 1 in 1000 in patients with fatal AIDS. Thus there are healthy persons with 43 times (30,000:700) and 33 times (1000:30) more HIV-infected cells than in AIDS patients.

(4) In terms of HIV's biological function, it is even more important

that the levels of HIV RNA synthesis in AIDS are either extremely low or even nonexistent. Only 1 in 10,000 to 100,000 leukocytes express viral RNA in 50% of AIDS patients. In the remaining 50% no HIV expression is detectable (Duesberg, 1989c; Simmonds *et al.*, 1990). The very fact that amplification by the polymerase chain reaction must be used to detect HIV DNA or RNA (Semple *et al.*, 1991) in AIDS patients indicates that not enough viral RNA can be made or is made in AIDS patients to explain any, much less fatal, pathogenicity based on conventional precedents (Duesberg and Schwartz, 1992). The amplification method is designed to detect a needle in a haystack, but a needle in a haystack is not sufficient to cause a fatal disease, even if it consists of plutonium or cyanide.

(5) In several AIDS diseases, that are not caused by immunodeficiency (Section 3.5.8), HIV is not even present in the diseased tissues, e.g. there is no trace of HIV in any Kaposi's sarcomas (Salahuddin *et al.*, 1988) and there is no HIV in neurons of patients with dementia, because of the generic inability of retroviruses to infect nondividing cells like neurons (Sections 3.5.8 and 3.5.10) (Duesberg, 1989c).

As a result, there is typically no free HIV in AIDS patients (Section 3.5.6). Indeed, the scarcity of infectious HIV in typical AIDS patients is the reason that neutralizing antibodies, rather than virus, have become the diagnostic basis of AIDS. It is also the reason that on average 5 million leukocytes of HIV-positives must be cultured to activate ("isolate") HIV from AIDS patients. Even under these conditions it may take up to 15 different isolation efforts (!) to get just one infectious virus out of an HIV carrier (Weiss *et al.*, 1988). The scarcity of HIV and HIV-infected cells in AIDS patients is also the very reason for the notorious difficulties experienced by leading American (Hamilton, 1991; Palca, 1991a; Crewdson, 1992) and British (Connor, 1991, 1992; Weiss, 1991) AIDS researchers in isolating, and in attributing credit for isolating HIV from AIDS patients.

(ii) HIV does not meet Koch's second postulate, because it is found not just in one, but in 25 distinct diseases, many as unrelated to each other as dementia and diarrhea, or Kaposi's sarcoma and pneumonia (Table 1, Section 2.1.2).

(iii) HIV also fails Koch's third postulate, because it fails to cause AIDS when experimentally inoculated into chimpanzees which make antibodies against HIV just like their human cousins (Blattner *et al.*, 1988; Institute of Medicine, 1988; Evans, 1989b; Weiss and Jaffe, 1990). Up to 150 chimpanzees have been inoculated since 1983 and all are still healthy (Duesberg, 1989c) (Jorg Eichberg, personal communication, see Section 1). HIV also fails to cause AIDS when accidentally introduced into humans (Duesberg, 1989c, 1991a).

There is, however, a legitimate limitation of Koch's postulates, namely that most microbial pathogens are only conditionally pathogenic (Stewart, 1968; McKeown, 1979; Moberg and Cohn, 1991). They are pathogenic only if the immune system is low, allowing infection or intoxication of the large numbers of cells that must be killed or altered for pathogenicity. This is true for tuberculosis bacillus, cholera, influenza virus, polio virus and many others (Freeman, 1979; Mims and White, 1984; Evans, 1989c).

However, even with such limitations HIV fails the third postulate. The scientific literature has yet to prove that even one health care worker has contracted AIDS from the over 206,000 American AIDS patients during the last 10 years, and that even one of thousands of scientists has developed AIDS from HIV, which they propagate in their laboratories and companies (Section 3.5.16) (Duesberg, 1989c, 1991a). AIDS is likewise not contagious to family members living with AIDS patients for at least 100 days in the same household (Friedland *et al.*, 1986; Sande, 1986; Hearst and Hulley, 1988; Peterman *et al.*, 1988). However the CDC has recently claimed that seven health care workers have developed AIDS from occupational infection (Centers for Disease Control, 1992c). But the CDC has failed to provide any evidence against nonoccupational causation, such as drug addiction (see Section 4). Indeed thousands of health care workers, e.g. 2586 by 1988 (Centers for Disease Control, 1988), have developed AIDS from nonprofessional causes. In addition the CDC has failed to report the AIDS diseases of the seven patients and those of their putative donors, has failed to report their sex (see next paragraph) and whether these patients developed AIDS only after AZT treatment (see Section 4) (Centers for Disease Control, 1992c).

The failure of HIV to meet the third postulate is all the more definitive since there is no antiviral drug or vaccine. Imagine what would happen if there were 206,000 polio or viral hepatitis patients in our hospitals and no health care workers were vaccinated!

Contrary to expectations that health care workers would be the first to be affected by infectious AIDS, the AIDS risk of those health care workers that have treated the 206,000 American AIDS patients is in fact lower than that of the general population, based on the following data. The CDC reports that about 75% of the American health care workers are females, but that 92% of the AIDS patients among health care workers are males (Centers for Disease Control, 1988). Thus the AIDS risk of male health care workers is 35 times higher than that of females, indicating nonprofessional AIDS causes.

Moreover, the CDC reports that the incidence of AIDS among health care workers is percentagewise the same as that in the general population, i.e. by 1988, 2586 out of 5 million health care workers, or 1/2000 had developed AIDS (Centers for Disease Control, 1988), by the same time 110,000 out of the 250 million Americans, or 1/2250, had developed AIDS (Centers for Disease Control, 1992b). Since health care workers are nearly all over 20 years old and since there is virtually no AIDS in those under 20 (Table 1), but those under 20 make up about 1/3 of the general population, it can be estimated that the AIDS risk of health care workers is actually 1/3 lower (1/3 times 1/2000) than that of the general population—hardly an argument for infectious AIDS.

In view of this, leading AIDS researchers have acknowledged that HIV fails Koch's postulates as the cause of AIDS (Blattner *et al.*, 1988; Evans, 1989a,b; Weiss and Jaffe, 1990; Gallo, 1991). Nevertheless, they have argued that the failure of HIV to meet Koch's postulates invalidates these postulates rather than HIV as the cause of AIDS (Section 6.1) (Evans, 1989b, 1992; Weiss and Jaffe, 1990; Gallo, 1991). But the failure of a suspected pathogen to meet Koch's postulates neither invalidates the timeless logic of Koch's postulates nor any claim that a suspect causes a disease (Duesberg, 1989b). It only means that the suspected pathogen cannot be proven responsible for a disease by Koch's postulates—but perhaps by new laws of causation (Section 6).

3.3.2. *Anti-HIV Immunity Does Not Protect Against AIDS*

Natural antiviral antibodies, or vaccination, against HIV—which completely neutralize HIV to virtually undetectable levels—are consistently diagnosed in AIDS patients with the “AIDS test.” Yet these antibodies consistently fail to protect against AIDS diseases (Section 3.5.11) (Duesberg, 1989b,c, 1991a; Evans, 1989a,b). According to Evans, “The dilemma in HIV is that antibody is not protective” (Evans, 1989a).

By contrast, all other viral diseases are prevented or cured by antiviral immunity. Indeed, since Jennerian vaccination in the late 18th century, antiviral immunity has been the only protection against viral disease. In view of this HIV researchers have argued that antibodies do not neutralize this virus (Section 3.5.11) instead of considering that HIV may not be the cause of AIDS.

3.3.3. *Antiviral Drugs Do Not Protect Against AIDS*

All anti-HIV drugs fail to prevent or cure AIDS diseases (Section 4).

3.3.4. *All AIDS-defining Diseases Occur in the Absence of HIV*

The absence of HIV does not prevent AIDS-defining diseases from occurring in all AIDS risk groups, it only prevents their diagnosis as AIDS (Sections 3.4.4, 4.5 and 4.7).

Thus, there is no proof for the virus-AIDS hypothesis—not even that AIDS is contagious. Instead, the virus-AIDS hypothesis is based only on circumstantial evidence, including epidemiological correlations and anecdotal cases (Sections 3.4 and 3.5).

3.4. Noncorrelations Between HIV and AIDS

Leading AIDS researchers acknowledge that correlations are the only support for the virus-AIDS hypothesis. For example, Blattner *et al.* state, “. . . overwhelming seroepidemiologic evidence (is) pointing toward HIV as the cause of AIDS . . . Better methods . . . show that HIV infection is present in essentially all AIDS patients” (Blattner *et al.*, 1988). According to an editorial in *Science*, Baltimore deduces from studies reporting an 88% correlation between antibodies to HIV and AIDS: “This was the kind of evidence we are looking for. It distinguishes between a virus that was a passenger and one that was the cause”

(Booth, 1988). The studies Baltimore relied on are those published by Gallo *et al.* in *Science* in 1984 that are the basis for the virus-AIDS hypothesis (Gallo *et al.*, 1984; Sarngadharan *et al.*, 1984), but their authenticity has since been questioned on several counts (Beardsley, 1986; Schupach, 1986; Connor, 1987; Crewdson, 1989; Hamilton, 1991; Palca, 1991a; Crewdson, 1992). Weiss and Jaffe concur that “the evidence that HIV causes AIDS is epidemiological . . .” (Weiss and Jaffe, 1990), although Gallo concedes that epidemiology is just “one hell of a good beginning” (Gallo, 1991). In view of correlations it is argued that “persons infected with HIV will develop AIDS and those not so infected will not” (Evans, 1989a), or that “HIV . . . is the *sine qua non* for the epidemic” (Gallo, 1991).

But correlations are only circumstantial evidence for a hypothesis. According to Sherlock Holmes, “Circumstantial evidence is a very tricky thing. It may seem to point very straight to one thing, but if you shift your point of view a little, you may find it pointing in an equally uncompromising manner to something entirely different” (Doyle, 1928). The risk in epidemiological studies is that the cause may be difficult to distinguish from noncausal associations. For example, yellow fingers are noncausally and smoking is causally associated with lung cancer. “In epidemiological parlance, the issue at stake is that of confounding” (Smith and Phillips, 1992). This is true for the “overwhelming sero-epidemiologic evidence” claimed to support the virus-AIDS hypothesis on the following grounds.

3.4.1. *Only about Half of American AIDS is Confirmed HIV-antibody-positive*

In the U.S. antibodies against HIV are only confirmed in about 50% of all AIDS diagnoses; the remainder are presumptively diagnosed (Institute of Medicine, 1988; Selik *et al.*, 1990). Several studies indicate that the natural coincidence between antibodies against HIV and AIDS diseases is not perfect, because all AIDS defining diseases occur in all AIDS risk groups in the absence of HIV (Section 4). Ironically, the CDC never records the incidence of HIV in its *HIV/AIDS Surveillance Reports* (Centers for Disease Control, 1992b).

It follows that the reportedly perfect correlation between HIV and

AIDS is in reality an artifact of the definition of AIDS and of allowances for presumptive diagnoses (Centers for Disease Control, 1987; Institute of Medicine, 1988). Since AIDS has been defined exclusively as diseases occurring in the presence of antibody to HIV (Section 2.2), the diagnosis of AIDS is biased by its definition toward a 100% correlation with HIV. That is why “persons infected by HIV will develop AIDS and . . . those not so infected will not” (Evans, 1989a), and why HIV is the *“sine qua non”* of AIDS (Gallo, 1991).

3.4.2. *Antibody-positive, but Virus-negative AIDS*

The correlations between AIDS and HIV are in fact not correlations with HIV, but with antibodies against HIV (Sarngadharan *et al.*, 1984; Blattner *et al.*, 1988; Duesberg, 1989c). But antibodies signal immunity against viruses, signal neutralization of viruses, and thus protection against viral disease—not a prognosis for a future disease as is claimed for antibodies against HIV. For example, antibody-positive against polio virus and measles virus means virus-negative, and thus protection against the corresponding viral diseases. The same is true for antibodies against HIV: antibody-positive means very much virus-negative. Residual virus or viral molecules are almost undetectable in most antibody-positive persons (Sections 3.3 and 3.5.6). Thus antibodies against HIV are not evidence for a future or current HIV disease unless additional assumptions are made (Section 3.5.11).

3.4.3. *HIV: Just One of Many Harmless Microbial Markers of Behavioral and Clinical AIDS Risks*

In addition to antibodies against HIV, there are antibodies against many other passenger viruses and microbes in AIDS risk groups and AIDS patients (Sections 2.3 and 4.3.2). These include cytomegalovirus, hepatitis virus, Epstein-Barr virus, Human T-cell Leukemia Virus-I (HTLV-I), herpes virus, gonorrhea, syphilis, mycoplasma, amoebae, tuberculosis, toxoplasma and many others (Gallo *et al.*, 1983; Sonnabend *et al.*, 1983; Blattner *et al.*, 1985; Mathur-Wagh *et al.*, 1985; Darrow *et al.*, 1987; Quinn *et al.*, 1987; Messiah *et al.*, 1988; Stewart, 1989; Goldsmith, 1990; Mills and Masur, 1990; Root-Bernstein, 1990a,c; Duesberg, 1991a; Buimovici-Klein *et al.*, 1988). In addition, there are between 100 and

150 chronically latent retroviruses in the human germ line (Martin *et al.*, 1981; Nakamura *et al.*, 1991). These human retroviruses are in every cell, not just in a few like HIV, and have the same genetic structure and complexity as HIV and all other retroviruses (Duesberg, 1989c). According to Quinn *et al.*, “Common to African patients with AIDS and outpatient controls and American patients with AIDS and homosexual men was the finding of extremely high prevalence rates of antibody to CMV (range, 92–100%), HSV (range, 90–100%), hepatitis B virus (range, 78–82%), hepatitis A virus (range, 82–95%), EBV capsid antigen (100%), syphilis (11–23%), and *T. gondii* (51–74%). In contrast, the prevalence of antibody to each of these infectious agents was significantly lower among the 100 American heterosexual men . . .” (Quinn *et al.*, 1987). Thus, the incidence of many human parasites, both rare and common, is high in typical AIDS patients and in typical AIDS risk groups (Sections 2.3 and 5). However, none of these microbes are fatal and nearly all are harmless to a normal immune system (Section 2.3).

Most of these parasites, including HIV, have been accumulated by AIDS risk behavior and by clinical AIDS risks (Blattner *et al.*, 1985; Institute of Medicine, 1988; Stewart, 1989). Such behavior includes the long-term injection of unsterile, recreational “street” drugs and large numbers of sexual contacts promoted by oral and injected aphrodisiac drugs (Section 4) (Dismukes *et al.*, 1968; Darrow *et al.*, 1987; Des Jarlais *et al.*, 1987; Espinoza *et al.*, 1987; Moss, 1987; Moss *et al.*, 1987; van Griensven *et al.*, 1987; Des Jarlais *et al.*, 1988; Messiah *et al.*, 1988; Chaisson *et al.*, 1989; Weiss, S.H., 1989; Deininger *et al.*, 1990; McKegney *et al.*, 1990; Stark *et al.*, 1990; Luca-Moretti, 1992; Seage *et al.*, 1992). Clinical risk groups, such as hemophiliacs, accumulate such viruses and microbes from occasionally contaminated transfusions (Section 3.4.4).

It follows that a high correlation between AIDS and antibodies against one particular virus, such as HIV, does not “distinguish between a virus that was a passenger and one that was a cause” (Baltimore, see above) (Booth, 1988). It is an expected consequence or marker of behavioral and clinical AIDS risks, particularly in countries where the percentage of HIV carriers is low (Duesberg, 1991a). In addition to HIV, many other microbes and viruses which are rare and inactive, or just inactive, in the general population, such as hepatitis virus, are “spe-

cific” for AIDS patients, and thus markers for AIDS risks (Sections 2.2, 2.3 and 4.3.2). For example, 100% of AIDS patients within certain cohorts, not just 50% as with HIV (Section 2.2), were shown to have antibodies against, or acute infections of, cytomegalovirus (Gottlieb *et al.*, 1981; Francis, 1983; van Griensven *et al.*, 1987; Buimovici-Klein *et al.*, 1988). A comparison of 481 HIV-positive with 1499 HIV-negative homosexual men in Berlin found that the HIV-positives were “significantly more often carriers of antibodies against hepatitis A virus, hepatitis B virus, cytomegalovirus, Epstein-Barr virus and syphilis” (Deininger *et al.*, 1990). And the frequent occurrence of antibodies against hepatitis B virus in cohorts of homosexual AIDS patients, termed “hepatitis cohorts,” was a precedent, that helped to convince the CDC to drop the “lifestyle” hypothesis of AIDS in favor of the “hepatitis analogy” (Francis *et al.*, 1983; Centers for Disease Control, 1986; Oppenheimer, 1992) (Section 2.2).

The higher the consumption of unsterile, injected drugs, the more sexual contacts mediated by aphrodisiac drugs and the more transfusions received, the more accidentally contaminating microbes will be accumulated (Sections 3.4.4.5, 4.3.2 and 4.5). In Africa antibodies against HIV and hepatitis virus are poor markers for AIDS risks, because millions carry antibodies against these viruses (Table 1) (Quinn *et al.*, 1987; Evans, 1989c; Blattner, 1991). Thus it is arbitrary to consider HIV the AIDS “driver” rather than just one of the many innocent microbial passengers of AIDS patients (Francis, 1983), because it is neither distinguished by its unique presence nor by its unique biochemical activity.

3.4.4. Annual AIDS Risks of Different HIV-infected Risk Groups, Including Babies, Homosexuals, Drug Addicts, Hemophiliacs and Africans, Differ over 100-fold

If HIV were the cause of AIDS the annual AIDS risks of all infected persons should be similar, particularly if they are from the same country. Failure of HIV to meet this prediction would indicate that HIV is not a sufficient cause of AIDS. The occurrence of the same AIDS-defining diseases in HIV-free controls would indicate that HIV is not even necessary for AIDS.

3.4.4.1. *Critically ill recipients of transfusions.* The annual AIDS risk of HIV-infected American recipients of transfusions (other than hemophiliacs) is about 50%, as half of all recipients die within one year after receiving a transfusion (Table 2) (Ward *et al.*, 1989).

Table 2. Annual AIDS Risks of HIV-infected Groups*

HIV-infected group	Annual AIDS in percent	Group-specific diseases
American recipients of transfusions	50	pneumonia, opportunistic infections
American babies	25	dementia, bacterial
Male homosexuals using sexual stimulants	4–6	Kaposi's sarcoma
Intravenous drug users	4–6	tuberculosis, wasting
American hemophiliacs	2	pneumonia, opportunistic infections
German hemophiliacs	1	pneumonia, opportunistic infections
American teenagers	0.16–1.7	hemophilia-related
American general population	0. 1–1	opportunistic infections
Africans	0.3	fever, diarrhea, tuberculosis
Thais	0.05	tuberculosis

*Based on controlled studies, it is proposed that the health risks of all HIV-infected AIDS risk groups are the same as those of matched HIV-free controls (Sections 3.4.4, 4 and 5). The virus hypothesis simply claims the specific morbidity of each of these groups for HIV.

Since the AIDS risk of transfusion recipients is much higher than the national 3–4% average, nonviral factors must play a role (Table 1). Indeed, about 50% of American recipients of transfusions without HIV also die within 1 year after receiving a transfusion (Hardy *et al.*, 1985; Ward *et al.*, 1989), and over 60% within 3 years (Bove *et al.*, 1987). Moreover, the AIDS risk of transfusion recipients increases 3–6 times faster with the

volume of blood received than their risk of infection by HIV (Hardy *et al.*, 1985; Ward *et al.*, 1989). This indicates that the illnesses that necessitated the transfusions are responsible for the mortality of the transfusion recipients. Yet the virus hypothesis claims the relatively high mortality of American transfusion patients for HIV without considering HIV-free controls. The hypothesis also fails to consider that the effects of HIV on transfusion mortality should be practically undetectable in the face of the high mortality of transfusion recipients and its postulate that HIV causes AIDS on average only 10 years after infection.

3.4.4.2. *HIV-infected babies.* The second highest annual AIDS risk is reported for perinatally infected American babies, whose health has been compromised by maternal drug addiction or by congenital diseases like hemophilia (Section 2.1.3). They develop AIDS diseases on average two years after birth (Anderson and May, 1988; Blattner *et al.*, 1988; Institute of Medicine, 1988; Blattner, 1991). This corresponds to an annual AIDS risk of 25% (Table 2).

Since the AIDS risk of babies is much higher than the national average of 3–4% (Table 1), nonviral factors must play a role in pediatric AIDS. Based on correlations and controlled studies documenting AIDS-defining diseases in HIV-free babies, it is proposed below that maternal drug consumption (Section 4) and congenital diseases, like hemophilia (Section 3.4.4.5), are the causes of pediatric AIDS. Indeed, before AIDS surfaced, many studies had shown that maternal drug addiction was sufficient to cause AIDS-defining diseases in newborns (Section 4.6.1). In accord with this proposal it is shown that HIV is naturally a perinatally transmitted retrovirus—and thus harmless (Section 3.5.2).

3.4.4.3. *HIV-positive homosexuals.* The annual AIDS risk of HIV-infected male homosexuals with hundreds of sex partners, who frequently use aphrodisiac drugs (Section 4), was originally estimated at about 6% (Mathur-Wagh *et al.*, 1985; Anderson and May, 1988; Institute of Medicine, 1988; Lui *et al.*, 1988; Moss *et al.*, 1988; Turner *et al.*, 1989; Lemp *et al.*, 1990; van Griensven *et al.*, 1990; Blattner, 1991). As more HIV-positives became identified, lower estimates of about 4% were reported

(Table 2) (Rezza *et al.*, 1990; Biggar and the International Registry of Seroconverters, 1990; Munoz *et al.*, 1992).

Since the annual AIDS risk of such homosexual men is higher than the national average, group-specific factors must be necessary for their specific AIDS diseases. Based on correlations with drug consumption and studies of HIV-free homosexuals, it is proposed here that the cumulative consumption of sexual stimulants and psychoactive drugs determines the annual AIDS risk of homosexuals (Sections 4.4 and 4.5). Indeed, all AIDS-defining diseases were observed in male homosexuals from behavioral risk groups before HIV was discovered and have since been observed in HIV-free homosexuals from AIDS risk groups (Sections 4.5 and 4.7).

In the spirit of the virus-AIDS hypothesis, many of these HIV-free homosexual AIDS cases have been blamed on various retrovirus-like particles, papilloma viruses, other viruses and microbes by researchers who have not investigated drug use, particularly not oral drug use. These cases include 153 immunodeficient HIV-free homosexuals with T4/T8-cell ratios below 1 (Drew *et al.*, 1985; Weber *et al.*, 1986; Novick *et al.*, 1986; Collier *et al.*, 1987; Bartholomew *et al.*, 1987; Buimovici-Klein *et al.*, 1988) and 23 HIV-free Kaposi's sarcomas (Afrasiabi *et al.*, 1986; Ho *et al.*, 1989b; Bowden *et al.*, 1991; Safai *et al.*, 1991; Castro *et al.*, 1992; Huang *et al.*, 1992) (see also Note added in proof).

3.4.4.4. *HIV-positive intravenous drug users.* Application of the annual AIDS risk of male homosexual risk groups led to valid predictions for the annual AIDS risk of intravenous drug users (Lemp *et al.*, 1990). Therefore the annual AIDS risk of HIV-infected intravenous drug users was originally estimated to be 6% (Table 2) (Lemp *et al.*, 1990; Blattner, 1991; Goudsmit, 1992). More recent studies have concluded that the annual AIDS risk of intravenous drug users is about 4% (Table 2) (Rezza *et al.*, 1990; Munoz *et al.*, 1992).

These findings argue against a sexually transmitted cause, because sexual transmission predicts a much higher AIDS risk for homosexuals with hundreds of sexual partners than for intravenous drug users (Section 4) (Weyer and Eggers, 1990; Eggers and Weyer, 1991). Indeed, numerous controlled studies have indicated that the morbidity and mor-

tality of intravenous drug users is independent of HIV (Sections 4.4, 4.5 and 4.7). On the basis of such studies it is proposed that the lifetime dose of drug consumption determines the annual AIDS risk of intravenous drug users (Section 4).

3.4.4.5. *HIV-positive hemophiliacs.* The hemophiliacs provide the most accessible group to test the virus hypothesis, because the time of infection can be estimated and because the role of other health risks can be controlled by studying HIV-free hemophiliacs.

About 15,000, or 75% of the 20,000 American hemophiliacs have HIV from transfusions received before the "AIDS test" was developed in 1984 (Tsoukas *et al.*, 1984; Hardy *et al.*, 1985; Institute of Medicine, 1986, 1988; Stehr-Green *et al.*, 1988; Goedert *et al.*, 1989; Koerper, 1989). Based on limited data and antibodies against selected viral antigens, it is generally estimated that most of these infections occurred between 1978 and 1984 (Evatt *et al.*, 1985; Johnson *et al.*, 1985; McGrady *et al.*, 1987; Goedert *et al.*, 1989). This high rate of infection reflects the practice, developed in the 1960s and 1970s, of preparing factor VIII from blood pools collected from large numbers of donors (Johnson *et al.*, 1985; Aronson, 1988; Koerper, 1989). Since only about 300 of the 15,000 HIV-infected American hemophiliacs have developed AIDS annually over the last 5 years (Morgan *et al.*, 1990; Centers for Disease Control, 1992a,b), the annual AIDS risk of HIV-infected American hemophiliacs is about 2% (Table 2). Data from Germany extend these results: about 50% of the 6000 German hemophiliacs are HIV-positive (Koerper, 1989), and only 37 (1%) of these developed AIDS-defining diseases during 1991 and 303 (1% annually) from 1982 until 1991 (Bundesgesundheitsamt (Germany), 1991; Leonhard, 1992). An international study estimated the annual AIDS risk of adult hemophiliacs at 3% and that of children at 1% over a 5-year period of HIV-infection (Biggar and the International Registry of Seroconverters, 1990).

According to the virus-AIDS hypothesis, one would have expected that by now (about one 10-year-HIV-latent-period after infection) at least 50% of the 15,000 HIV-positive American hemophiliacs would have developed AIDS or died from AIDS. But the 2% annual AIDS risk indicates that the average HIV-positive hemophiliac would have to

wait for 25 years to develop AIDS diseases from HIV, which is the same as their current median age. The median age of American hemophiliacs has increased from 11 years in 1972, to 20 years in 1982 and to over 25 years in 1986, despite the infiltration of HIV in 75% (Johnson *et al.*, 1985; Institute of Medicine, 1986; Koerper, 1989). Thus, one could make a logical argument that HIV, instead of decreasing the lifespan of hemophiliacs, has in fact increased it.

Considering the compromised health of many hemophiliacs compared to the general population, it is also surprising, that the 1–2% annual AIDS risk of HIV-infected hemophiliacs is lower than the 3–4% risk of the average HIV-infected, nonhemophilic European or American (Table 1). There is even a bigger discrepancy between the annual AIDS risks of hemophiliacs and those of intravenous drug users and male homosexuals, which are both about 4–6% (Table 2). In an effort to reconcile the relatively low annual AIDS risks of hemophiliacs with that of homosexuals, the hematologists Sullivan *et al.* (1986) noted “The reasons for this difference remain unclear.” And Biggar and colleagues (1990) noted that “AIDS incubation . . . was significantly faster” for drug users and homosexuals than for hemophiliacs.

In view of the many claims that HIV causes AIDS in hemophiliacs, it is even more surprising that there is not even one controlled study from any country showing that the morbidity or mortality of HIV-positive hemophiliacs is higher than that of HIV-negative controls.

Instead, controlled studies show that immunodeficiency in hemophiliacs is independent of HIV, and that the lifetime dosage of transfusions is the cause of AIDS-defining diseases of hemophiliacs. Studies describing immunodeficiency in HIV-free hemophiliacs are summarized in Table 3 (Tsoukas *et al.*, 1984; AIDS Hemophilia French Study Group, 1985; Ludlam *et al.*, 1985; Gill *et al.*, 1986; Kreiss *et al.*, 1986; Madhok *et al.*, 1986; Sullivan *et al.*, 1986; Sharp *et al.*, 1987; Matheson *et al.*, 1987; Antonaci *et al.*, 1988; Mahir *et al.*, 1988; Aledort, 1988; Jin *et al.*, 1989; Jason *et al.*, 1990; Lang, *et al.*, 1989; Becherer *et al.*, 1990). One of these studies even documents an AIDS-defining disease in an HIV-free hemophiliac (Kreiss *et al.*, 1986). Immunodeficiency in these studies is typically defined by a T4 to T8-cell ratio of about 1 or less than 1, compared to a normal ratio of 2.

Most of the studies listed in Table 3 and additional ones conducted before HIV had been discovered have concluded or noted that immunodeficiency is directly proportional to the number of transfusions received over a lifetime (Menitove *et al.*, 1983; Kreiss *et al.*, 1984; Johnson *et al.*, 1985; Hardy *et al.*, 1985; Pollack *et al.*, 1985; Prince, 1992; Ludlum *et al.*, 1985; Gill *et al.*, 1986). According to the hematologists Pollack *et al.* (1985) "derangement of immune function in hemophiliacs results from transfusion of foreign proteins or a ubiquitous virus rather than contracting AIDS infectious agent." The "ubiquitous virus" was a reference to the virus-AIDS hypothesis but a rejection of HIV, because in 1985 HIV was extremely rare in blood concentrates outside the U.S., but immunodeficiency was observed in Israeli, Scottish and American hemophiliacs (Pollack *et al.*, 1985). Madhok *et al.* also arrived at the conclusion that "clotting factor concentrate impairs the cell mediated immune response to a new antigen in the absence of infection with HIV" (Madhok *et al.*, 1986). Aledort observed that "chronic recipients . . . of factor VIII, factor IX and pooled products . . . demonstrated significant T-cell abnormalities regardless of the presence of HIV antibody" (Aledort, 1988). Even those who claim that clotting factor does not cause immunodeficiency show that immunodeficiency in hemophiliacs increases with both the age and the cumulative dose of clotting factor received during a lifetime (Becherer *et al.*, 1990).

One controlled study showed directly that protein impurities of commercial factor VIII, rather than factor VIII or HIV, were immunosuppressive among factor VIII-treated, HIV-positive hemophiliacs. Over a period of two years the T-cells of HIV-positive hemophiliacs treated with commercial factor VIII declined two-fold, while those of matched HIV-positive controls treated with purified factor VIII remained unchanged (Table 3) (de Biasi *et al.*, 1991).

Before AIDS, a multicenter study investigating the immune systems of 1551 hemophiliacs treated with factor VIII from 1975 to 1979 documented lymphocytopenia in 9.3% and thrombocytopenia in 5% (Eyster *et al.*, 1985). Accordingly, AIDS-defining opportunistic infections, including 60% pneumonias and 20% tuberculosis, have been recorded in hemophiliacs between 1968 and 1979 (Johnson *et al.*, 1985). These transfusion-acquired immunodeficiencies could more than account for

Table 3. Immunosuppression in HIV-negative and -positive Hemophiliacs

Study	Immunosuppression (T4/T8 about or less than 1)	
	HIV-negative	HIV-positive
1. Tsoukas <i>et al.</i> (1984)	6/14	9/15
2. Ludlam <i>et al.</i> (1985)	15	—
3. French Study Group (1985)	33	55
4. Sullivan <i>et al.</i> (1986)	28	83
5. Madhok <i>et al.</i> (1986)	9	10
6. Kreiss <i>et al.</i> (1986)	6/17	22/24
7. Gill <i>et al.</i> (1986)	8/24	30/32
8. Sharp <i>et al.</i> (1987)	5/12	—
9. Matheson <i>et al.</i> (1987)	5	3
10. Mahir <i>et al.</i> (1988)	6	5
11. Antonaci <i>et al.</i> (1988)	15	10
12. Aledort (1988)	57	167
13. Jin <i>et al.</i> (1989)	12	7
14. Lang <i>et al.</i> (1989)	24	172
15. Becherer <i>et al.</i> (1990)	74	136
16. Jason <i>et al.</i> (1990)	31	—
17. de Biasi <i>et al.</i> (1991)	—	10/20

*In a normal immune system, the T4 to T8 T-cell ratio is about 2, in immunodeficient persons and in many AIDS patients it is about 1 or below 1. Studies which list the fraction of immunodeficient hemophiliacs in HIV-positive and HIV-negative groups indicate, that HIV-positives are more likely to be immunodeficient. This is because HIV is a marker for the number of transfusions received and transfusion of foreign proteins causes immune deficiency. The study by de Biasi *et al.* (1991) showed that among 20 HIV-positive hemophiliacs only those 10 who received commercially purified factor VIII, but not those who received further purified factor VIII developed immunodeficiency over a period of two years. See text for references.

the 2% annual incidence of AIDS-defining diseases in HIV-positive hemophiliacs recorded now (Centers for Disease Control, 1992b). An American hematologist who recorded opportunistic infections in hemophiliacs occurring between 1968 and 1979, including 2 candidiasis and 66 pneumonia deaths, commented in 1983 "... it seems possible that many of the unspecified pneumonias in hemophiliacs in the past would be classified today as AIDS" (Aronson, 1983).

It follows that long-term transfusion of foreign proteins causes immunodeficiency in hemophiliacs with or without HIV. The virus hypothesis has simply claimed normal morbidity and mortality of hemophiliacs for HIV, by ignoring HIV-free controls.

Nevertheless several investigators comparing HIV-negative to HIV-positive hemophiliacs have noted that immunodeficiency is more often associated with HIV-positives (Table 3), and have observed that HIV correlates with the number of transfusions received (Tsoukas *et al.*, 1984; Kreiss *et al.*, 1986; Sullivan *et al.*, 1986; Koerper, 1989; Becherer *et al.*, 1990). According to Kreiss *et al.* "seropositive hemophiliac subjects, on average, had been exposed to twice as much concentrate ... as seronegative[s]" (Kreiss *et al.*, 1986). And according to Goedert *et al.* "the prevalence of HIV-1 antibodies was directly associated with the degree of severity (of hemophilia)" (Goedert *et al.*, 1989). Thus HIV appears just to be a marker of the multiplicity of transfusions, rather than a cause of immunodeficiency.

The conclusion that long-term transfusion of foreign proteins causes immunodeficiency makes three testable predictions:

(1) It predicts that hemophiliacs with "AIDS" would be older than average hemophiliacs. Indeed, the median age of hemophiliacs with AIDS in the U.S. (Evatt *et al.*, 1984; Koerper, 1989; Stehr-Green *et al.*, 1989), England (Darby *et al.*, 1989) and other countries (Biggar and the International Registry of Seroconverters, 1990; Blattner, 1991) is significantly higher (about 34 years in the U.S.; Johnson *et al.*, 1985; Koerper, 1989; Becherer *et al.*, 1990) than the average age of hemophiliacs (20–25 years in the U.S., see above). Goedert *et al.* reported that the annual AIDS risk of 1- to 17-year-old hemophiliacs was 1.5%, that of 18- to 34-year-old hemophiliacs was 3% and that of 64-year-old hemo-

philiacs was 5% (Goedert *et al.*, 1989). This confirms that the cumulative dose of transfusions received is the cause of AIDS-defining diseases among hemophiliacs. According to the hematologist Koerper, "this may reflect lifetime exposure to a greater number of units of concentrate, . . ." and to Evatt *et al.*, "This age bias may be due to differences in duration of exposure to blood products . . ." (Evatt *et al.*, 1984; Koerper, 1989).

By contrast, AIDS caused by an autonomous infectious pathogen would be largely independent of the age of the recipient. Even if HIV were that pathogen, the hemophiliac population with AIDS should have the same age distribution as the hemophiliac population over 10 years, because HIV is thought to take 10 years to cause AIDS and nearly all hemophiliacs were infected about 10 years ago (Johnson *et al.*, 1985; McGrady *et al.*, 1987; Koerper, 1989).

(2) Foreign protein-mediated immunodeficiency further predicts that all AIDS diseases of hemophiliacs are opportunistic infections. If hemophilia AIDS were due to HIV only 62% of their AIDS diseases would be opportunistic infections, because 38% of all American AIDS patients have diseases, that are not dependent on, and not consistently associated with, immunodeficiency (Table 1, Section 3.5.8). These include wasting disease (19%), Kaposi's sarcoma (10%), dementia (6%) and lymphoma (3%) (Table 1).

The AIDS pathology of hemophiliacs confirms the prediction of the foreign protein-hypothesis exactly. In America 99% of the hemophiliacs with AIDS have opportunistic infections, of which about 70% are fungal and viral pneumonias, and less than 1% have Kaposi's sarcoma (Evatt *et al.*, 1984; Selik *et al.*, 1987; Stehr-Green *et al.*, 1988; Goedert *et al.*, 1989; Koerper, 1989; Becherer *et al.*, 1990). The small percentage of Kaposi's sarcoma is due to the nitrite inhalants used by male homosexual hemophiliacs as sexual stimulants (Section 4). There are no reports of wasting disease and dementia in hemophiliacs.

(3) If hemophilia AIDS is due to transfusion of foreign proteins, the wives of hemophiliacs should not contract AIDS from their mates. But if it were due to a parenterally or sexually transmitted virus, hemophilia AIDS would be sexually transmissible. Indeed, AIDS researchers claim that the wives of hemophiliacs develop AIDS from sexual transmission

of HIV (Lawrence *et al.*, 1990; Weiss and Jaffe, 1990; Centers for Disease Control, 1992b). For example AIDS researcher Fauci asks: “How about the 60-year-old wife of a hemophiliac who gets infected? Is she cruising, too?” (Booth, 1988).

However, (a) statistical scrutiny and (b) a controlled study unconfirm the hypothesis that hemophilia AIDS is sexually transmissible: (a) The CDC reports that 94 wives of hemophiliacs have been diagnosed with unnamed AIDS diseases since 1985 (Centers for Disease Control, 1992b). If one considers that there have been 15,000 HIV-positive hemophiliacs in the U.S. since 1985 and assumes that a third are married, then there are 5000 wives of HIV-positive hemophiliacs. About 13 of these women have developed AIDS annually during the 7 years (94:7) from 1985 to 1991 (Centers for Disease Control, 1992b). By contrast, at least 80 of these women would be expected to die per year, considering the human lifespan of about 80 years and that on average at least 1.6% of all those over 20 years of age die annually. Thus, until controls show that among 5000 HIV-negative wives of hemophiliacs only 67 (80–13) die annually, the claim that wives of hemophiliacs die from sexual transmission of HIV is unfounded speculation.

Moreover, it has been pointed out that all AIDS-defining diseases of the wives of hemophiliacs are typically age-related opportunistic infections, including 81% pneumonia (Lawrence *et al.*, 1990). Kaposi’s sarcoma, dementia, lymphoma and wasting syndrome are not observed in wives of hemophiliacs (Lawrence *et al.*, 1990). Thus the virus-AIDS hypothesis seems to claim, once more, normal morbidity and mortality of the wives of hemophiliacs for HIV.

(b) To test the hypothesis that immunodeficiency of hemophiliacs is sexually transmissible the T4 to T8 cell-ratio of 41 spouses and female sexual partners of immunodeficient hemophiliacs were analyzed (Kreiss *et al.*, 1984). Twenty-two of the females had relationships with hemophiliacs with T-cell ratios below 1 and 19 with hemophiliacs with ratios of 1 and greater. The mean duration of relationships was 10 years, the mean number of sexual contacts was 111 during the previous year, and only 12% had used condoms (Kreiss *et al.*, 1984). Since the T-cell ratios of all spouses were normal, averaging 1.68—exactly like those of 57 normal controls, the authors concluded that “there is no evidence to

date for heterosexual or household-contact transmission of T-cell subset abnormalities from hemophiliacs to their spouses . . .” (Kreiss *et al.*, 1984).

It follows that the foreign protein-hypothesis, but not the HIV-hypothesis, correctly predicts (1) the pathology, (2) the age bias, (3) the non-contagiousness of hemophilia AIDS and (4) HIV-free immunodeficiency in hemophiliacs. It also explains the discrepancies between the annual AIDS risks of hemophiliacs and other risk groups (Table 2).

Since the virus hypothesis has become totally dominant in 1988, no new studies have described HIV-free immunodeficient hemophiliacs (Table 3) and the question whether HIV-free immunodeficient hemophiliacs ever developed AIDS-defining diseases became a taboo. The study by Jason *et al.* described data collected in the mid 1980s, the studies by Jin *et al.* and Becherer *et al.* collected data before 1988 and the one by de Biasi *et al.* compared the effects of purified to unpurified factor VIII only in HIV-positive hemophiliacs (Table 3).

In response to the argument that hemophiliacs only began to develop AIDS diseases when HIV appeared (Centers for Disease Control, 1986; Oppenheimer, 1992), it is proposed that “new” AIDS-defining diseases among hemophiliacs are an indirect consequence of extending their life with factor VIII treatment. Long-term treatment with factor VIII has prolonged the median life of hemophiliacs from 11 in 1972 to 25 in 1986. But contaminating foreign proteins received over periods of 10 years of treatment have also caused immunodeficiencies, and various viral and microbial contaminants have caused infections in some, and HIV infection in 75%. HIV has been a marker for the number of transfusions and factor VIII treatments received, just like hepatitis virus infection was a marker of the number of transfusions received until it was eliminated from the blood supplies (Anonymous, 1984; Koerper, 1989). Prior to factor VIII therapy most hemophiliacs died as adolescents from internal bleeding (Koerper, 1989).

3.4.4.6. HIV-positive teenagers. The annual AIDS risk of HIV-infected American teenagers can be calculated as follows: There are about 30 million American teenagers, of which 0.03% (10,000) (Burke *et al.*, 1990) to 0.3% (100,000) (St Louis *et al.*, 1991) are HIV-positive. Since only

160 developed AIDS in 1991 and only 170 in 1990 (Centers for Disease Control, 1992b), their annual AIDS risk is between 0.16% and 1.7% (Table 2).

Thus the AIDS risk of teenagers with HIV is less than the national average of 3–4%. There are no statistics to indicate that the annual risk for AIDS-defining diseases of the HIV-infected teenage population is higher than that of HIV-free controls (Section 3.5.2). Since most American teenagers with AIDS are either hemophiliacs (38%), intravenous drug users (25%) or male homosexuals (25%) (Section 2.1.3), it is proposed that the associated risk factors, rather than HIV, are the cause of teenage AIDS (Sections 3.4.4.5 and 4).

3.4.4.7. *HIV-positive general U.S. population.* The CDC reports that 3% of all American AIDS cases are from the general population, corresponding to 900–1200 of the 30,000–40,000 annual AIDS cases (Table 1) (Centers for Disease Control, 1992b). Since at least 0.03% to 0.3%, or 80,000 to 800,000, of the general American population of 250 million are infected (Section 3.5.2) (U.S. Department of Health and Human Services, 1990; Burke *et al.*, 1990; Morgan *et al.*, 1990; St Louis *et al.*, 1991), the annual AIDS risk of the general population must be between 0.1% and 1% (Table 2). Thus the annual AIDS risk of HIV-infected Americans of the general population is similar to that of teenagers.

There are no statistics to indicate that the annual AIDS risk of the general HIV-infected population is higher than the annual risk for AIDS-defining diseases in HIV-free controls. Because the incidence of AIDS in the general population is exceedingly low, it is proposed again that it reflects the normal, low incidence of AIDS-defining diseases, rather than HIV-mediated diseases.

3.4.4.8. *HIV-positive Africans.* The annual AIDS risks of HIV-infected Africans is only 0.3% (Tables 1 and 2), because 6 million HIV carriers generated 129,000 AIDS cases from 1985 to the end of 1991 (Table 1). There are no controlled studies indicating that the risk for AIDS-defining diseases of HIV-infected Africans differs from that of HIV-negative controls.

Since the annual AIDS risk of HIV-infected Africans is (1) 10-times

lower than the average American and European risk, (2) up to 100-fold less than that of American/European risk groups, (3) the same for both sexes unlike that in America and Europe and (4) very low considering that the annual mortality in Africa is around 2% and that AIDS includes the most common African diseases, it is proposed that African AIDS is just a new name for indigenous African diseases (Section 2.1.2).

Instead of a new virus, malnutrition, parasitic infections and poor sanitary conditions have all been proposed as causes of African AIDS-defining diseases (Editorial, 1987; KonoteyAhulu, 1987, 1989; Rapoport, 1988; Adams, 1989). Further, it has been proposed that the incidence of tuberculosis, diarrhea, fever and other African AIDS-defining diseases may be the same in Africans with and without HIV (Editorial, 1987). And prior to the discovery of HIV, protein malnutrition was identified by the AIDS researchers Fauci *et al.* as the world's leading cause of immunodeficiency, particularly in underdeveloped countries (Seligmann *et al.*, 1984).

Indeed, recent studies document that only 2168 out of 4383 (49.5%) African AIDS patients with slim disease, tuberculosis and other Africa-specific diseases, who all met the WHO definition of AIDS, were infected by HIV. These patients were from Abidjan, Ivory Coast (De Cock *et al.*, 1991; Taelman *et al.*, 1991), Lusaka, Zambia and Kinshasa, Zaire (Taelman *et al.*, 1991). Another study reports 135 (59%) HIV-free AIDS patients from Ghana out of 227 diagnosed by clinical criteria of the WHO. These patients suffered from weight loss, diarrhea, chronic fever, tuberculosis and neurological diseases (Hishida *et al.*, 1992). An earlier study documents 116 HIV-negatives among 424 African patients that meet the WHO definition of AIDS (Widy-Wirski *et al.*, 1988). According to an African AIDS doctor, "Today, because of AIDS, it seems that Africans are not allowed to die from these conditions any longer" (Konotey-Ahulu, 1987). Another asks "What use is a clinical case definition for AIDS in Africa?" (Gilks, 1991).

The 10-fold difference between the average annual AIDS risks of Africans and Americans/ Europeans (Table 1) can thus be resolved as follows: (1) The high AIDS risk of HIV-positive Americans and Europeans is the product of the low absolute numbers of HIV carriers in the U.S. and Europe compared to Africa (Table 1) and of the concentra-

tion of HIV in AIDS risks groups, e.g. consumers of recreational drugs and the antiviral drug AZT (Section 4) and recipients of transfusions (Section 3.4.3). (2) The low AIDS risk of Africans is a product of large absolute numbers of HIV carriers and their relatively low, spontaneous and malnutrition-mediated AIDS risks.

3.4.4.9. *HIV-positive Thais*. Given that there have been only 123 Thai AIDS cases in the last 1–2 years and an estimated 300,000 HIV carriers in Thailand (Weniger *et al.*, 1991), the annual AIDS risk of HIV-infected Thais can be calculated to be less than 0.05% (Table 2). Since most of these 123 were either intravenous drug users or “sex workers” (Section 2.1.3), it is proposed that these specific health risks are their cause of AIDS (Section 4), rather than the HIV that they share, unspecifically, with 300,000 healthy Thais.

The over 100-fold range in the annual AIDS risks of different AIDS risks groups, summarized in Table 2, clearly indicates that HIV is not sufficient to cause AIDS. It confirms and extends an earlier CDC conclusion: “The magnitude of some of the differences in rates is so great that even gross errors in denominator estimates can be overcome” (Hardy *et al.*, 1985). Moreover, analysis of the specific health risks of each risk group has identified nonviral health risks that are necessary and sufficient causes of AIDS (Table 3 and Section 4.5).

3.4.5. *Specific AIDS Diseases Predetermined by Prior Health Risks*

If HIV were the cause of AIDS, every AIDS case should have the same risk of having one or more of the 25 AIDS diseases. However, the data listed above (Section 2.1) and in Table 2 indicate that per AIDS case different risk groups have very specific AIDS diseases:

(1) Male homosexuals have 20 times more Kaposi’s sarcoma than all other American and European AIDS risk groups.

(2) Hemophiliacs and other recipients of transfusions have fungal and viral pneumonia and other opportunistic infections, and practically no Kaposi’s sarcoma or dementia.

(3) The AIDS diseases of the “general population” are either spontaneous, hemophilia- or age-related opportunistic infections. Typical

examples are cited below (Section 3.5.16).

(4) Babies exclusively have bacterial infections (18%) and a high rate of dementia (14%), compared to adults (6%) (Table I).

(5) Africans develop Africa-specific AIDS diseases 10 times more and Kaposi's sarcoma 10 times less often than Americans or Europeans.

The epidemiological data summarized in Section 3.4 indicate that HIV is sufficient to determine neither the annual AIDS risk, nor the type of AIDS disease an infected person may develop. Instead, prior health risks including drug consumption, malnutrition and congenital diseases like hemophilia and their treatments and even the country of residence, predetermine AIDS diseases. The correlations between HIV and AIDS that are claimed to support the virus-AIDS hypothesis are not direct, not complete, not distinctive and, above all, not controlled. Controlled studies indicate that the incidence of AIDS-defining diseases in intravenous drug users, male homosexuals practicing risk behavior and hemophiliacs is independent of HIV.

Therefore, it is proposed that various group-specific health risk factors, including recreational and antiviral drugs (Section 4) and malnutrition, are necessary and sufficient causes of AIDS. The existence of risk group-specific AIDS-defining diseases in the absence of HIV confirms this conclusion (Sections 3.4.4 and 4.5).

3.5. Assumptions and Anecdotal Cases that Appear to Support the Virus-AIDS Hypothesis

The following assumptions and anecdotal cases are frequently claimed to prove the virus-AIDS hypothesis. Despite the popularity of these claims they are either uncontrolled for alternative explanations or they are natural coincidences between HIV infection and naturally-occurring diseases.

3.5.1. HIV is Presumed New Because AIDS is New

HIV is presumed new in all countries with AIDS, because AIDS is new (Blattner *et al.*, 1988; Gallo and Montagnier, 1988; Weiss and Jaffe, 1990). The presumed newness of HIV is used as a primary argument for the virus-AIDS hypothesis: . . . 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” (Blattner *et al.*, 1988) or: “In every country and city where AIDS has appeared, HIV infection preceded it just by a few years” (Weiss and Jaffe, 1990).

However, according to Farr’s law, the age of a microbe in a population is determined by changes in its incidence over time (Bregman and Langmuir, 1990). If a microbe is spreading from a low to a high incidence it is new; however, if its incidence in a population is constant, it is old (Fig. 1) (Freeman, 1979; Duesberg, 1991a). Figure 1 shows the incidences of long established microbes in the U.S. population, i.e. *Candida* and *Pneumocystis* each at about 100% (Freeman, 1979; Pifer, 1984;

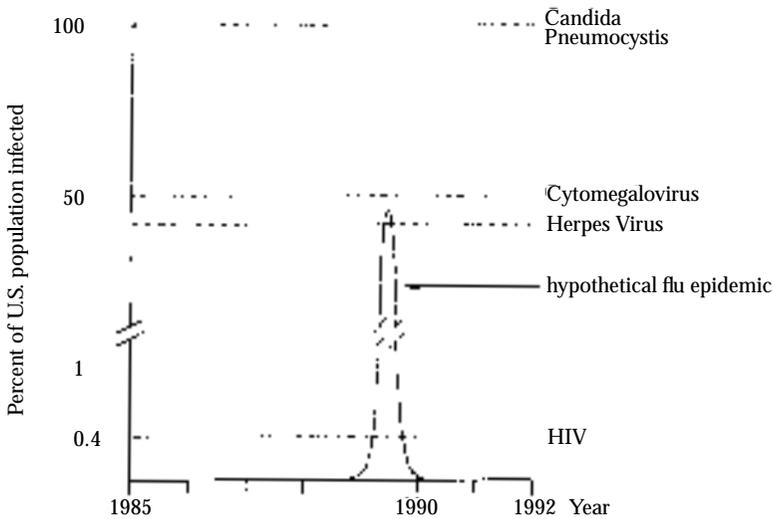


Figure 1. Determination of the age of a microbe in a population based on Farr’s law. Farr’s law holds that a microbe entering a population spreads exponentially until a susceptible pool is saturated. Subsequently those microbes that are incompatible with long term survival of the host are eliminated exponentially, to generate a bell-shaped curve. The rise and fall of a hypothetical flu epidemic caused by a new strain of influenza virus is an example. But microbes that can coexist with their host become established. Examples are *Candida*, *Pneumocystis* (Freeman, 1979), cytomegalovirus, herpes virus (Evans, 1989c) and HIV (see text), these are shown at the percentages at which they are established in the American population.

Williford Pifer *et al.*, 1988), and cytomegalovirus and herpes virus at about 50% and 40%, respectively (Evans, 1989c). In addition, it shows the typical exponential rise and subsequent fall of a hypothetical epidemic by a new influenza virus strain (Freeman, 1979).

Ever since antibodies against HIV were first detected by the “AIDS test” in 1985, the number of antibody-positive Americans has been fixed at a constant population of 1 million, or 0.4% (Section 2.2. and Table 1). The U.S. Army also reports that from 1985 to 1990 an unchanging 0.03% of male and female applicants have been HIV-positive (Burke *et al.*, 1990). This is the predicted distribution of a long established virus (Fig. 1). Since there are over 250 million uninfected Americans, and since there is no antiviral vaccine or drug to stop the spread of HIV, the non-spread of HIV in the U.S. in the last 7 years is an infallible indication that the American “HIV epidemic” is old. The Central African HIV epidemic has also remained fixed at about 10% of the population since 1985 (Section 2.2). Likewise, HIV has remained fixed at 500,000 Europeans since 1988 (World Health Organization, 1992a). The non-spread of HIV confirms exactly the conclusion reached below that HIV behaves in a population as a quasi-genetic marker (Section 3.5.2). Hence, the assumption that HIV is new in the U.S. or in Africa is erroneous.

Indeed HIV existed in the U.S. long before its fictitious origin in Africa (Gallo, 1987; Gallo and Montagnier, 1988; Anderson and May, 1992) and its fictitious entry into this country in the 1970s (Shilts, 1987). For example, in the U.S. in 1968 an HIV-positive, male homosexual prostitute died from Kaposi’s sarcoma and immunodeficiency (Garry *et al.*, 1988), and 45 out of 1129 American intravenous drug users were found to be HIV-positive in 1971 and 1972 (Moore *et al.*, 1986).

The putative novelty of HIV is an anthropocentric interpretation of new technology that made it possible to discover HIV and many other latent retroviruses like HTLV-I (Duesberg and Schwartz, 1992). Indeed, the technology to detect a latent virus like HIV only became available around the time AIDS appeared. Given a new virus-scope, the assertion that HIV is new is just like claiming the appearance of “new” stars with a new telescope. Thus the claims that “. . . first HIV is introduced, then AIDS appears” (Blattner *et al.*, 1988) and that “HIV . . . preceded it (AIDS)” (Weiss and Jaffe, 1990) are ironically more true than the

proponents of the virus hypothesis had anticipated. HIV preceded AIDS by many, perhaps millions, of years.

3.5.2. *HIV—Assumed to be Sexually Transmitted—Depends on Perinatal Transmission for Survival*

AIDS is said to be a sexually transmitted disease, because HIV is thought to be a sexually transmitted virus (Section 2.2). However, HIV is not by nature a sexually transmitted virus. Sexual transmission of HIV is extremely inefficient. Based on studies measuring heterosexual and homosexual transmission, it depends on an average of 1000 heterosexual contacts and 100–500 homosexual contacts with antibody-positive people (Rosenberg and Weiner, 1988; Lawrence *et al.*, 1990; Blattner, 1991; Hearst and Hulley, 1988; Peterman *et al.*, 1988). According to Rosenberg and Weiner, “HIV infection in non-drug using prostitutes tends to be low or absent, implying that sexual activity alone does not place them at high risk” (Rosenberg and Weiner, 1988). Moreover, unwanted pregnancies and venereal diseases, but not HIV infections, have increased significantly in the U.S. since HIV has been known (Institute of Medicine, 1988; Aral and Holmes, 1991). This argues directly against sexual transmission of HIV.

Sexual transmission is so inefficient because there is no free, non-neutralized HIV anywhere in antibody-positive persons, particularly not in semen (Section 3.3). In a group of 25 antibody-positive men, only one single provirus of HIV could be found in over 1 million cells of semen in one of the men and no HIV at all was found in the semen of the other 24 (Van Voorhis *et al.*, 1991). Likewise, HIV could only be isolated or reactivated from ejaculates of 9 out of 95 antibody-positive men by cocultivation with 2 million phytohemagglutinin-activated leukocytes (Anderson *et al.*, 1992). No virus or microbe could survive if it depended on a transmission strategy that is as inefficient as 1 in 1000 contacts.

Indeed, HIV depends on perinatal, instead of sexual, transmission for survival—just like other animal and human retroviruses. Therefore, the efficiency of perinatal transmission must be high. This appears to be the case. Based on HIV-tracking via the “AIDS test,” perinatal transmission from the mother is estimated to be 13–50% efficient (Blattner

et al., 1988; Blattner, 1991; Duesberg, 1991a; Institute of Medicine, 1988; European Collaborative Study, 1991). This number does not include paternal HIV transmission to the baby via semen, for which there are currently no data. The real efficiency of perinatal transmission must be higher than the antibody-tests suggest, because in a fraction of recipients HIV only becomes immunogenic when its hosts are of an advanced age (Quinn *et al.*, 1986; St Louis *et al.*, 1991). During the antibody-negative phase, latent HIV can be detected by the polymerase chain reaction (Rogers *et al.*, 1989, European Collaborative Study, 1991). This is also true for other perinatally transmitted human (Blattner, 1990; Duesberg, 1991a) and animal retroviruses (Rowe, 1973; Duesberg, 1987).

HIV survival via perinatal transmission leads to two predictions: (1) HIV cannot be inherently pathogenic—just like all other perinatally transmitted viruses and microbes (Freeman, 1979; Mims and White, 1984). No microbe-host system could survive if the microbe were perinatally transmitted and at once fatal. (2) HIV must function as a quasi-genetic marker, because it is quasi-nontransmissible by sex, or other natural horizontal modes of transmission, just like known murine retrovirus prototypes (Rowe, 1973; Duesberg, 1987). Both predictions are confirmed:

(1) Overwhelming statistical evidence from the U.S. and Africa documents that the risk for AIDS-defining diseases for HIV-positive babies, in the absence of other risk factors (Sections 3.4.4 and 4), is the same as that of HIV-free controls:

(a) “AIDS tests” from applicants to the U.S. Army and the U.S. Job Corps indicate that between 0.03% (Burke *et al.*, 1990) and 0.3% (St Louis *et al.*, 1991) of the 17- to 19-year-old applicants are HIV-infected but healthy. Since there are about 90 million Americans under the age of 20, there must be between 27,000 and 270,000 (0.03%–0.3% of 90 million) HIV carriers. In Central Africa there are even more, since 1–2% of healthy children are HIV-positive (Quinn *et al.*, 1986).

Most, if not all, of these adolescents must have acquired HIV from perinatal infection for the following reasons: sexual transmission of HIV depends on an average of 1000 sexual contacts, and only 1 in 250 Americans carries HIV (Table 1). Thus, all positive teenagers would

have had to achieve an absurd 1000 contacts with a positive partner, or an even more absurd 250,000 sexual contacts with random Americans to acquire HIV by sexual transmission. It follows that probably all of the healthy adolescent HIV carriers were perinatally infected, as for example the 22-year-old Kimberly Bergalis (Section 3.5.16).

The AIDS risk of perinatally infected babies of the general population can be estimated as follows. Between 27,000 and 270,000 Americans under the age of 20 carry HIV. But only about 4260 AIDS cases have been recorded in this age group in the last 10 years (Centers for Disease Control, 1992b). Therefore, between 85% and 98% of HIV-infected youths do not develop AIDS up to 20 years after perinatal infection (Section 2.1). Since the above number includes the AIDS babies from drug-addicted mothers (Sections 3.4.2 and 4), the AIDS risk of HIV-infected babies from mothers that don't use drugs probably reflects normal infant mortality.

(b) A controlled study from Africa compared 218 newborns from HIV-positive mothers to 218 from HIV-negative mothers, and the "rates of prematurity, low birth weight, congenital malformations and neonatal mortality were comparable in the two groups" (Lepage *et al.*, 1991). The mothers were matched for age and parity and the "frequency of signs and symptoms was not statistically different in the two groups."

(2) The incidence of HIV in American teenagers of different ethnic backgrounds is predictable on genetic grounds. It is about 10-fold higher in blacks than in whites, i.e. 0.3% compared to 0.03% (U.S. Department of Health and Human Services, 1990; Burke *et al.*, 1990; Blattner, 1991; Palca, 1991b; St Louis *et al.*, 1991; Vermund, 1991). HIV was even 50-fold more common in black mothers in inner-city hospitals in New York (36%) than in whites (0.7%) (Landesmann *et al.*, 1987). This reflects the 25- to 50-fold higher incidence of HIV in the blacks' African ancestors (10%) compared to the whites' European ancestors (0.2 to 0.4%) (Section 2.2, Table 1). Likewise, the different ethnic groups of the Caribbean reflect the distinct HTLV-I incidences of their ancestors in Africa, Europe and Japan, despite generations of coexistence on the Caribbean islands (Blattner, 1990). The unchanging incidence of HIV in the American population (Fig. 1) also confirms the view that HIV is a quasi-genetic marker. Since there is virtually no horizontal transmis-

sion of retroviruses, murine retroviruses have functioned as classical genetic markers of mice that could only be distinguished from cellular genes by fastidious genetic crosses (Rowe, 1973).

Thus the assumption that AIDS is sexually transmitted by HIV is not consistent with the natural perinatal mode of HIV transmission. If natural transmission of HIV caused a disease, AIDS would be a pediatric disease. Instead, HIV is merely a marker of either an average of 1000 sexual contacts and thus of many other possible AIDS risks associated with very high sexual activity or of long-term intravenous drug use (Sections 3.4.3 and 5).

3.5.3. AIDS Assumed to be Proportional to HIV Infection

The incidence of AIDS is assumed to be proportional to the incidence of HIV via a constant factor. For example, a 10-fold higher incidence of AIDS in American and European males compared to females is assumed to reflect a 10-fold higher incidence of HIV in men (Blattner *et al.*, 1988; Blattner, 1991; Goudsmit, 1992).

However, there is no evidence that the incidence of HIV is 10 times higher in males than in females of the general American and European population, although this is the case for AIDS (Table I). Indeed, the most recent claim for a 90% bias of HIV for males of the general population (Blattner, 1991) is only supported by a reference to an editorial (Palca, 1991b), which itself provides nothing more than an unreferenced cartoon showing global patterns of HIV infection. According to a CDC epidemiologist, estimates of how HIV is distributed between the sexes of the general population are “approximations” based on the distribution of AIDS (Tim Dondero, CDC, personal communication; see also Anderson and May, 1992)—a tautology.

Proportionality between HIV and AIDS via a constant is also incompatible with the following statistics. The U.S. Army (Burke *et al.*, 1990) and the U.S. Job Corps (St Louis *et al.*, 1991) report, based on millions of tests, that HIV has been equally distributed between the sexes among 17- to 21-year-olds of the general population over the last five years for which data were available (Sections 3.5.1 and 3.5.2). Since testing 17- to 19-year-olds annually for 5 years is equivalent to testing 17- to 24-year-olds, the U.S. Army data predict that among 17- to 24-year-olds,

AIDS risks should be distributed equally between the sexes. However, the CDC documents that 85% of the AIDS cases among 17- to 24-year-olds were males (Centers for Disease Control, 1992b).

In response to this, some proponents of the virus-AIDS hypothesis have speculated that teenage homosexuals exclude themselves from the Army. However, Randy Shilts, a homosexual writer, reports that just the opposite is true (Shilts, 1991). Moreover, most teenagers are not as yet aware of a definite homosexual persuasion and are not likely to understand the implications nor to fear the consequences of a positive "AIDS test."

The over 100-fold discrepancies between the AIDS risks of different HIV-infected risk groups also disprove the claim that the incidence of AIDS is proportional via a constant to the incidence of HIV (Table 2). The proportionality between HIV and AIDS only holds if the analysis is restricted to groups with the same AIDS risks. In groups with the same percentage of HIV but with different AIDS risks, AIDS segregates specifically with nonviral AIDS risks, e.g. illicit recreational drugs, the antiviral drug AZT (Section 4) and frequent transfusions (Section 3.4.4).

3.5.4. *AIDS Assumed to be Homosexually Transmitted in the U.S. and Europe*

In view of a sexually transmitted AIDS virus, it is paradoxical that AIDS is 90% male in America and 86% male in Europe (Sections 3.1 and 3.2). Therefore it is assumed that "the virus first got its footing in the U.S." in male homosexuals (Booth, 1988) and has remained with homosexuals because it is transmitted preferentially by anal intercourse and because homosexuals have no sex with heterosexuals (Centers for Disease Control, 1986; Shilts, 1987; Blatter *et al.*, 1988; Institute of Medicine, 1988; Blattner, 1991; Bardach, 1992; Project Inform, 1992).

However, this assumption is inconsistent with the fact that about 10% of all males and females prefer anal intercourse (Bolling and Voeller, 1987; Turner *et al.*, 1989) and that American and European heterosexuals have sufficient access to HIV. The females would be infected by HIV-positive, heterosexual intravenous drug users, hemophiliacs, and bisexual males. Thus, if HIV were transmitted by anal intercourse, about the same percentage of women as men should develop AIDS, particu-

larly since the efficiencies of transmission of anal and vaginal intercourse are approximately the same, e.g. between 1 to 100 and 1 to 500 for anal and 1 to 1000 for vaginal intercourse (Blattner, 1991) (see also Section 3.5.2). Yet, despite widespread alarm, this has not occurred in the last 10 years in the U.S. (Table 1), although the first women with AIDS had been diagnosed as early as in 1981 (Centers for Disease Control, 1986; Guinan and Hardy, 1987). The risk of women for both HIV infection and AIDS is the same for those who practice anal intercourse as for those who practice other types of intercourse (Guinan and Hardy, 1987).

The preferred anal-transmission hypothesis is also incompatible with the sexually equal distribution of HIV and AIDS in Africa. Since it is postulated that HIV appeared in America and Africa at about the same time 10–20 years ago (Institute of Medicine, 1986; Blattner *et al.*, 1988; Gallo and Montagnier, 1988), HIV should have reached the same equilibria between the sexes in all countries.

Instead it is shown below that the male bias for AIDS in America and Europe reflects male-specific behavior, including the facts that over 75% of all intravenous drug users are males and that long-term consumption of sexual stimulants, like amylnitrite and ethylchloride inhalants, is almost entirely restricted to male homosexuals (Section 4). HIV is just a marker of the many sexual stimulants used to achieve 500–1000 sexual contacts (Section 4). The difference between the AIDS risks of men in America and Europe, namely drugs, and those of Africans, namely country-specific, but not sex-specific, risk factors (Section 3.4.4.8) resolves the paradox between the different sexual distributions of AIDS in these countries.

3.5.5. AIDS Assumed to be Heterosexually Transmitted by African "Life-style"

AIDS in Africa is assumed to affect both genders equally, because HIV is distributed equally between the sexes by "prostitution" (Institute of Medicine, 1988), lack of "circumcision" (Klein, 1988; Marx, 1989; Blattner, 1991), African "lifestyle" (Quinn *et al.*, 1987; Blattner *et al.*, 1988; Goodgame, 1990) and "voodoo rituals" (Gallo, 1991). These assumptions are compatible with the sexually equal distributions of HIV and AIDS in Africa.

However, AIDS in Africa is hard to reconcile with the known efficiency of sexual transmission of HIV. Since it takes 1000 HIV-positive sexual contacts to transmit HIV and about 10% of all Central Africans, or 6 million, are HIV-positive (Section 2.2), 6 million Africans would have had to achieve on average at least 10,000 sexual contacts with random Africans to pick up HIV. Since this is highly improbable, it is also highly improbable that sexual transmission of HIV is the cause of AIDS in Africa. The true reason for the sexually equal distribution of HIV in Africa is perinatal transmission of HIV (Section 3.5.2). Nonsexual, country-specific risk factors are the reason for the “sexually” equal distribution of AIDS in Africa (Section 3.4.4.8).

3.5.6. *HIV Claimed to be Abundant in AIDS Cases*

HIV is said to be abundant or viremic in AIDS patients (Baltimore and Feinberg, 1989; Coombs *et al.*, 1989; Ho *et al.*, 1989a; Semple *et al.*, 1991) and thus compatible with orthodox viruses which cause disease only at high titers (Duesberg and Schwartz, 1992). In other words HIV is assumed to meet Koch’s first postulate (Section 3.3). The assumption is based on two papers which reported HIV titers of 10^2 to 10^3 infectious units per mL of blood in 75% of AIDS patients and in 25–50% of asymptomatic HIV carriers (Coombs *et al.*, 1989; Ho *et al.*, 1989a). The authors and an accompanying editorial, *HIV Revealed, Toward a Natural History of the Infection* (Baltimore and Feinberg, 1989), concluded that these findings established HIV viremia as an orthodox criterion of viral pathogenicity. Viremia of similar titers was recently also implied in some AIDS patients and asymptomatic carriers based on an indirect assay that amplifies HIV RNA *in vitro* (Semple *et al.*, 1991).

However, several arguments cast doubt on the claim that HIV viremia is relevant for AIDS:

(1) Since viremia was observed in 25–50% of asymptomatic HIV carriers (Coombs *et al.*, 1989; Ho *et al.*, 1989a; Semple *et al.*, 1991), it cannot be sufficient for AIDS.

(2) Since no viremia was observed in 25% of the AIDS cases studied by two groups (Coombs *et al.*, 1989; Ho *et al.*, 1989a), it is not necessary for AIDS.

(3) Viremia initiated from a previously suppressed virus and observed

years after infection is a classical consequence, rather than the cause of immunodeficiency. Indeed, many normally latent parasites become activated and may cause chronic “opportunistic infections” in immunodeficient persons, as for example *Candida*, *Pneumocystis*, herpes virus, cytomegalovirus, hepatitis virus, tuberculosis bacillus, toxoplasma (Sections 2.3 and 3.4.3)—and sometimes even HIV. It is consistent with this view that HIV viremia is observed more often in AIDS patients than in asymptomatic carriers (Duesberg, 1990c).

(4) The HIVs that make up the “viremias” are apparently not infectious *in vivo*, because only a negligible fraction of leukocytes, on average only 1 in 1500 to 8000, of AIDS patients are infected (Section 3.3). The probable reason is that the “viremias” consist of viruses that are neutralized by the antiviral antibodies of “seropositive” AIDS patients (Duesberg, 1992d). Since viruses, as obligatory cellular parasites, can only be pathogenic by infecting cells, these noninfectious viremias cannot be relevant to the cause of AIDS. If assayed *in vitro*, in the absence of free antiviral antibodies, antibodies may dissociate from neutralized viruses and thus render the virus infectious for cells in culture. This explains the discrepancy between the noninfectious “viremias” *in vivo* and the relatively high infectivity recorded *in vitro* (Coombs *et al.*, 1989; Ho *et al.*, 1989a).

Thus HIV viremia is a rare, predictable consequence of immunodeficiency rather than its cause.

3.5.7. *HIV to Depend on Cofactors for AIDS*

Conceding that HIV is not sufficient to cause AIDS, it is assumed to depend on cofactors. Montagnier (Goldsmith, 1990; Lemaitre *et al.*, 1990; Balter, 1991) and Lo (Lo *et al.*, 1991) have proposed mycoplasmas that were discovered in their laboratories; Gallo has proposed two viruses, herpes virus-6 and HTLV-I, which were both discovered in his laboratory (Cotton, 1990; Gallo, 1990, 1991; Lusso *et al.*, 1991). Others have proposed cytomegalovirus, Epstein-Barr virus (Quinn *et al.*, 1987; Evans, 1989a; Root-Bernstein, 1990c), “age” (Evans, 1989a; Goedert *et al.*, 1989; Weiss and Jaffe, 1990; Biggar and the International Registry of Seroconverters, 1990), unidentified “coagents” (Weyer and Eggers, 1990; Eggers and Weyer, 1991), “clinical illness promotion fac-

tors” (Evans, 1989b, 1992) and even “pre-existing immune abnormalities” (Ludlam *et al.*, 1985; Marion *et al.*, 1989; Ludlam, 1992) as cofactors of HIV.

However, cofactor hypotheses only replace HIV-specific AIDS problems with the following HIV-plus-cofactor-specific AIDS problems:

(1) Since HIV is extremely rare and dormant in most antibody-positive AIDS patients (Sections 2.2 and 3.3), it is hard to imagine how its various AIDS-allies could benefit from their dormant “cofactor” HIV.

(2) Since HTLV-I is just as dormant and unable to kill cells as HIV (Duesberg, 1987; Blattner, 1990; Duesberg and Schwartz, 1992), it is even harder to imagine how one dormant virus could help another dormant virus to generate the biochemical activity that would be necessary to cause a fatal disease.

(3) Since mycoplasma (Freeman, 1979; Cotton, 1990; Goldsmith, 1990; Balter, 1991), herpes virus-6 (Cotton, 1990; Lusso *et al.*, 1991), cytomegalovirus and Epstein-Barr virus (Mims and White, 1984; Evans, 1989c) are each very common, if not ubiquitous, parasites (Freeman, 1979; Froesner, 1991), AIDS should develop in most people as soon as they are infected by HIV. Likewise, “aged” people should develop AIDS as soon as they are infected by HIV. Yet not more than 3–4% of HIV-antibody-positive Americans or Europeans and 0.3% of antibody-positive Africans develop AIDS per year (Tables 1 and 2).

Moreover, if infectious cofactors helped HIV to cause AIDS, the AIDS risk of Africans would be expected to be higher than that of Americans. This is because the incidence of hypothetical, microbial cofactors in Africans without AIDS was found to be the same as in those with AIDS, while the incidence of microbial cofactors in Americans without AIDS risks was significantly lower than in those with AIDS (Section 3.4.3) (Quinn *et al.*, 1987). Even the cofactor HIV was present in 6% of African AIDS-free controls (Quinn *et al.*, 1987). Yet the annual AIDS risk of HIV-infected Africans is 10-times lower than that of Americans (Table 1).

(4) Contrary to the claims that “age” is an AIDS cofactor of HIV, the virus-AIDS hypothesis postulates that the latent period for HIV is longer in adults (10 years) than in children (2 years) (Section 2.2). How-

ever, the proposal that “age” is a cofactor for HIV becomes more compelling the more the hypothetical “latent period” of HIV grows. Clearly, if a 70-year-old will be infected by a virus with a “latent period” of 10 years, “age” will be a predictable cofactor (see, for example, hemophiliacs, Section 3.4.4.5 and Paul Gann, Section 3.5.16).

(5) The claims that HIV depends on “clinical illness promotion factors” (Evans, 1992) or on a “pre-existing immune abnormality” (Marion *et al.*, 1989; Ludlam, 1992) for AIDS are euphemisms for saying that HIV cannot cause AIDS until something else does (Duesberg, 1989b). The additional hypothesis that a “pre-existing immune abnormality” (Ludlam, 1992) or a “prior immune dysfunction” (Marion *et al.*, 1989) makes a subject more susceptible to HIV is erroneous, because a pre-existing immune deficiency only affects the progression of an infection, but not the risk of infection.

In view of this, I share Gallo’s concerns about cofactors of HIV, which he expresses with a quotation from Lewis Thomas: “Multifactorial is multi-ignorance. Most factors go away when we learn the real cause of a disease” (Gallo, 1991). The “cofactor” HIV may be no exception. Until any one of these hypothetical cofactors is actually shown to depend on HIV to cause AIDS, HIV must be considered just one of many innocent bystanders found in AIDS patients (Section 3.4.3).

3.5.8. *All AIDS Diseases to Result from Immunodeficiency*

All AIDS diseases are said to reflect a primary immunodeficiency (Cofin *et al.*, 1986; Institute of Medicine, 1986; Blattner *et al.*, 1988).

However, immunodeficiency is not a common denominator of all AIDS diseases. About 38% of all AIDS diseases, i.e. dementia, wasting disease, Kaposi’s sarcoma and lymphoma (Table 1), are neither caused by, nor necessarily associated with, immunodeficiency. Cancer is not a consequence of immunodeficiency (Stutman, 1975; Duesberg, 1989c). Indeed, Kaposi’s sarcoma frequently has been diagnosed in male homosexuals in the absence of immunodeficiency. For example, the immune systems of 20 out of 37 HIV-positive homosexuals with Kaposi’s sarcoma were normal when their disease was first diagnosed (Spornraft *et al.*, 1988). Another study also describes 19 male homosexual Kaposi’s sarcoma patients with normal immune systems (Mur-

ray *et al.*, 1988). Likewise, Kaposi's sarcomas have been diagnosed in HIV-free male homosexuals with normal immune systems (Afrasiabi *et al.*, 1986; Archer *et al.*, 1989; Friedman-Kien *et al.*, 1990; Marquart *et al.*, 1991).

Dementia and wasting disease also are not consequences of immunodeficiency (Duesberg, 1989c, 1991a). Thus, the assumption that all AIDS diseases are caused by immunodeficiency is erroneous.

3.5.9. *HIV to Induce AIDS via Autoimmunity and Apoptosis*

In view of the extremely low number of HIV-infected cells in AIDS patients (Section 3.3), HIV has recently been proposed to cause AIDS by inducing autoimmunity (Hoffmann, 1990; Maddox, 1991a; Mathé, 1992) or apoptosis (Laurent-Crawford *et al.*, 1991; Goudsmit, 1992). According to these new ideas HIV is assumed either to confuse the immune system into attacking itself or to persuade the immune cells to commit suicide, termed apoptosis. The autoimmune hypothesis postulates homology between HIV and human cells, and currently relies only on mouse and monkey models (Hoffmann, 1990; Maddox, 1991a), and on precedents for autoimmunity induced in humans as a consequence of graft rejection and blood transfusions (Root-Bernstein, 1990a,b; Mathe, 1992). One autoimmunologist claims that "each of Duesberg's paradoxes might be understood in the context of the model without sacrificing the idea that HIV is usually involved in pathogenesis" (Hoffmann, 1990). This strategy of crediting me rather than the virus-AIDS hypothesis for its paradoxes shifts the discussion from a problem with science to a problem with a scientist (Booth, 1988; Weiss and Jaffe, 1990).

However, both the autoimmune and the apoptosis hypotheses are incompatible with human AIDS on several grounds:

(1) Autoimmunity or apoptosis cannot account for all those AIDS diseases that are not caused by immunodeficiency, e.g. Kaposi's sarcoma, dementia, wasting disease and lymphoma (Section 3.5.8).

(2) Autoimmunity or apoptosis fail to explain risk group-specific AIDS diseases (Section 2.1.3, Tables 1 and 2).

(3) Autoimmunity and apoptosis fail to explain the long average intervals, "latent periods," from conventional immunity against HIV,

detected by the “AIDS test,” to hypothetical autoimmunity 10 years later (Section 3.2).

(4) Autoimmunity and apoptosis fail to explain the over 100-fold discrepancies between the annual AIDS risks of different HIV-infected groups (Table 2).

(5) HIV-induced autoimmunity or apoptosis fail to explain the consistent 90% bias of American/European AIDS for males (Section 2.1, Table 1).

(6) In view of the autoimmunity or apoptosis hypothesis, it is paradoxical that 80% of antibody-positive Americans (1 million minus the 206,000 who have developed AIDS) and 98% of antibody-positive Africans (6 million minus the 129,000 who have developed AIDS) have not developed AIDS since 1984 (Table 1). Obviously, these figures are not even corrected for the normal and drug-induced incidence of AIDS-defining diseases in those groups (Section 3.4.4, Table 2).

(7) There is no sequence homology between HIV and human DNA detectable by hybridization to predict autoimmunity (Shaw *et al.*, 1984). Therefore, autoimmunologists argue that antibodies against those antibodies, which are directed at the viral proteins that bind to cellular receptors, would also react with cellular receptors and thus cause AIDS (Hoffmann, 1990). However, if this were true, all viruses should cause AIDS.

Thus the HIV-autoimmunity and apoptosis hypotheses of AIDS are (a) not compatible with essential parameters of human AIDS and (b) arbitrary, because they are not based on an autoimmunogenic or apoptogenic property of HIV that is distinct from all other viruses.

3.5.10. *HIV Assumed to Kill T-cells*

Based on an early observation by Gallo *et al.* HIV is assumed to cause immunodeficiency by specifically killing T-cells (Gallo *et al.*, 1984; Weiss and Jaffe, 1990). Gallo’s observation was restricted to primary T-cells (Gallo *et al.*, 1984) but not to established T-cell lines (Rubinstein, 1990). However, according to Montagnier, the discoverer of HIV, “In a search for a direct cytopathic effect of the virus on (primary) T-lymphocytes, no gross changes could be seen in virus-producing cultures, with regard to cell lysis or impairment of cell growth” (Montagnier *et al.*, 1984).

Others have confirmed that HIV does not kill infected, primary T-cells *in vitro* (Hoxie *et al.*, 1985; Anand *et al.*, 1987; Langhoff *et al.*, 1989; Duesberg, 1989c). Moreover HIV-infected primary T-cells are considered the natural “reservoir” of HIV *in vivo* (Schnittman *et al.*, 1989).

Thus Gallo’s controversial observation probably reflects the notorious difficulties experienced by his laboratory in maintaining primary blood cells alive in culture instead of a genuine cytotoxic function of HIV (Crewdson, 1989; Culliton, 1990; Rubinstein, 1990; Hamilton, 1991). Gallo showed in a later study from his laboratory that about 50% of uninfected T-cells died within 12 days in culture (Gallo, 1990).

Indeed, the assumption that HIV is cytotoxic is incompatible with generic properties of retroviruses and with specific properties of HIV:

(1) The hallmark of retrovirus replication is to convert the viral RNA into DNA and to deliberately integrate this DNA as a parasitic gene into the cellular DNA (Weiss *et al.*, 1985). This process of integration depends on mitosis to succeed, rather than on cell death (Rubin and Temin, 1958; Duesberg, 1989c). The resulting genetic parasite can then be either active or passive, just like other cellular genes (Duesberg, 1987). Transcription of viral RNA from chromosomally integrated proviral DNA also only works if the cell survives infection, because dying cells are not transcriptionally active. Thus, this strategy of replication depends entirely on the survival of the infected cell.

Noncytotoxic replication is the reason that retroviruses were all considered potential carcinogens before AIDS (Weiss *et al.*, 1985; Duesberg, 1987). For example, Gallo’s first candidate for an AIDS virus is called Human T-cell Leukemia Virus-I (Gallo *et al.*, 1983), and Gallo’s second candidate for an AIDS virus was originally described at a press conference in April 1984 by Gallo and the Secretary of Health and Human Services as “a variant of a known human cancer virus called HTLV III” (Crewdson, 1989; Rubinstein, 1990). It used to be called Human T-cell Leukemia Virus-III by Gallo (Gallo *et al.*, 1984; Shaw *et al.*, 1984) before it was renamed HIV in 1986 (Coffin *et al.*, 1986).

(2) Limited cytotoxicity of HIV has been observed soon after infection of cells *in vitro* (Duesberg, 1989c; Bergeron and Sodroski, 1992). Therefore, it has been proposed that multiple copies of unintegrated

proviral DNA, generated by multiple infections before all cellular receptors are blocked by newly replicated viruses, could kill T-cells (Bergeron and Sodroski, 1992). However, cells infected by every retrovirus, including HIV (Bergeron and Sodroski, 1992), survive multiple unintegrated proviral DNAs during the early phase of the infection (Weiss *et al.*, 1985). Rare cell death during this phase of infection is a consequence of cell fusion, which is mediated by viruses on the surface of infected cells binding to receptors of uninfected cells. In some conditions retrovirus-mediated fusion occurs so reliably that it has been used to quantitate retroviruses in tissue culture. However, virus-mediated fusion is blocked by antiviral antibodies and thus not relevant to the loss of T-cells in persons with antibodies against HIV (Duesberg, 1989c).

Alternatively, it has been proposed that HIV proteins are directly toxic because of structural similarities with scorpion and snake poisons (Gallo, 1991; Garry *et al.*, 1991; Garry and Koch, 1992). However, no such toxicity is observed in millions of asymptomatic HIV carriers, and there is no reason that it should occur, if it did, only after latent periods of 10 years.

(3) The propagation of HIV in indefinitely growing human T-cells for the "AIDS test" was patented by Gallo *et al.* in 1984 (Rubinstein, 1990) and was recently confirmed by Montagnier (Lemaître *et al.*, 1990). It is totally incompatible with Gallo's claim that HIV kills T-cells. Such HIV-producing T-cells have been growing in many laboratories and companies since 1984 producing virus at titers of up to 10^6 virus particles per mL, which is many orders of magnitude more than is ever observed in humans with or without AIDS (Duesberg, 1989c, 1991a).

In view of this, Gallo postulates that T-cell lines in culture have all acquired resistance to HIV killing (Gallo, 1991). However, there is no precedent for this *ad hoc* hypothesis, as no other cytotoxic virus has ever been observed that is cytotoxic *in vivo* and in primary cells *in vitro*, but is noncytotoxic in cell lines in culture. It is also implausible that a potentially life-saving cellular mutation, such as resistance to the hypothetical "AIDS virus," would be restricted just to cells in culture, particularly if these mutations occur so readily that they are found in all T-cell lines. There is not even one T-cell line that is consistently killed by HIV.

(4) HIV, like all other retroviruses, does not specifically infect T-cells.

It also infects monocytes, epithelial cells, B-cells, glial cells and macrophages, etc. and none of these are killed by HIV (Levy, 1988; Duesberg, 1991a). Most other retroviruses also infect T-cells, which is why so many of them are suspected “T-cell leukemia” viruses (Weiss *et al.*, 1985; Duesberg, 1987; Blattner, 1990).

Thus, the assumption that HIV causes AIDS by killing T-cells is not tenable.

3.5.11. *Antibodies Assumed not to Neutralize HIV*

Antibodies against HIV, detected by a positive “AIDS test,” are claimed not to protect against AIDS because they do not neutralize HIV (Institute of Medicine, 1988; Evans, 1989a; Weiss and Jaffe, 1990; Gallo, 1991). “It is a test for anti-HIV antibodies and not, as Duesberg states, ‘neutralizing antibodies’” (Baltimore and Feinberg, 1990).

However, antiviral immunity completely neutralizes HIV and restricts it to undetectable levels in healthy HIV-carriers as well as in AIDS patients (Section 3.3.1) (Duesberg, 1989b,c). Indeed, two recent studies have just confirmed that HIV activity is “rapidly and effectively limited” by antiviral immunity (Clark *et al.*, 1991; Daar *et al.*, 1991) to less than 1 in 1000 T-cells (Section 3.3). By contrast, HIV replicates in the absence of antiviral immunity in human T-cells in culture to titers of 10^6 virus particles per mL (Section 3.5.10). Thus, the assumption that HIV causes AIDS because of inadequate antiviral immunity is unconfirmed. Baltimore’s, Feinberg’s and Evans’ paradox “that antibody is not protective” (Evans, 1989a) is their failure to recognize the non-role of HIV in AIDS (Section 3.3.2).

3.5.12. *HIV Claimed to Cause AIDS in 50% Within 10 Years*

All HIV-infected persons are said to die from AIDS after a medium latent period of 10 years (Anderson and May, 1988; Institute of Medicine, 1988; Moss *et al.*, 1988; Lemp *et al.*, 1990; Blattner, 1991; Duesberg, 1991a).

However, according to statistics from the CDC, only about 30,000–40,000, or 3–4%, of a reservoir of 1 million HIV-infected Americans develop AIDS annually (Table 1). Likewise, 3% of infected Europeans develop AIDS per year (Table 1). Accordingly, 50% of

HIV-infected Americans and Europeans would have to wait 12–16 years and 100%, 24–33 years to develop AIDS. During this time, many would die from other causes. Since only 0.3% of infected Africans develop AIDS diseases annually (Tables 1 and 2), 50% of Africans would have to wait about 150 years and 100% would have to wait 300 years to develop AIDS.

Thus, it is presumptuous to claim that HIV causes AIDS in 50% of infected persons after median latent periods of 10 years, particularly since the virus has only been known for nine years.

3.5.13. *HIV Said to Derive Pathogenicity from Constant Mutation*

During its long latent periods, HIV is claimed to acquire pathogenicity by mutation, for example by generating variants that escape immunity (Hahn *et al.*, 1986; Levy, 1988; Eigen, 1989; Gallo, 1990; Weiss and Jaffe, 1990; Anonymous, 1992; Anderson and May, 1992) or by generating defective variants (Eigen, 1989; Haas, 1989; Weiss, R.A., 1989).

However, a recent study just demonstrated that the replicative and functional properties of HIVs from AIDS patients are the same as those from asymptomatic carriers (Lu and Andrieu, 1992). Indeed, most essential structural and replicative proteins of a virus cannot be mutated without eliminating its viability. Functionally relevant mutations of any virus are also severely restricted by the necessity to remain compatible with the host (Duesberg, 1990b). Moreover, there is no precedent for an immune system that has been able to neutralize a virus completely and is then unable to catch up with an occasional subsequent mutation. If viruses in general could evade the immune system by mutation, the immune system would be a useless burden to the host.

Likewise, the proposals that defective HIVs could generate pathogenicity is untenable. Defective viruses are only viable in the presence of nondefective helper viruses and thus unlikely to survive in natural transmission from host to host at low multiplicity of infection, particularly with helper viruses that never achieve high titers like HIV (Duesberg, 1989a).

There are, however, examples of new antigenic variants of retroviruses (Beemon *et al.*, 1974) or influenza viruses (Duesberg, 1968), that have arisen upon rare double infection by two antigenically distinct

virus strains via genetic recombination. Yet antigenically new variants of HIV have never been observed in American and European AIDS patients, as all HIV strains diagnosed to date crossreact with the very same standard HIV-1 strain that is patented in America and Europe for the “AIDS test” (Connor, 1991, 1992; Palca, 1991a; Weiss, 1991).

Moreover, if recombination or spontaneous mutation could generate pathogenic HIV mutants from nonpathogenic strains, one would expect all those who are infected by HIV from AIDS patients to develop AIDS within weeks after infection. Such HIV mutants should be pathogenic just as soon as conventional, nonpathogenic HIV strains are immunogenic. But this is not observed.

Thus, the assumption that HIV acquires pathogenicity by mutation during the course of the infection is not tenable.

3.5.14. *HIV Assumed to Cause AIDS with Genes Unique Among Retroviruses*

AIDS researchers assert that HIV causes AIDS with unique genetic information that all other animal and human retroviruses lack and that these unique genes would regulate HIV down during the “latent period” and up during AIDS (Gallo and Montagnier, 1988; Haseltine and Wong-Staal, 1988; Institute of Medicine, 1988; Eigen, 1989; Temin, 1990; Fauci, 1991; Gallo, 1991). Further, it is claimed that HIV-infected cells export factors encoded by these genes that promote neoplastic growth of uninfected cells to cause, for example, Kaposi’s sarcoma (Salahuddin *et al.*, 1988; Ensoli *et al.*, 1990; Gallo, 1990); at the same time such genes are said to export “scorpion poison”-related toxins that kill uninfected neurons to cause dementia (Gallo, 1991; Garry *et al.*, 1991; Garry and Koch, 1992). By contrast, all other known bacterial, animal and human viruses, including retroviruses, are only able to kill or alter those cells they infect, because viruses are manufactured inside cells and would not benefit from proteins released to uninfected cells.

However, the claims of unique retroviral HIV genes with unique control functions raises several unresolvable problems:

(1) Despite its presumed unique properties HIV has the same genetic complexity, i.e. 9000 nucleotides, and the same genetic structure as all

other retroviruses (Beemon *et al.*, 1974; Wang *et al.*, 1976; Institute of Medicine, 1988). It shares with other retroviruses the three major genes *gag-pol-env*, which are linked in this order in all animal and human retroviruses (Wang *et al.*, 1976). Although “novel” genes that overlap with the major retroviral genes have been discovered in HIV by computerized sequence analysis, and by new protein detection technology (Varmus, 1988), such genes have also been found with the same technology in other retroviruses that do not cause AIDS, such as HTLV-I, other human retroviruses, bovine retroviruses, simian retroviruses and sheep retroviruses (Varmus, 1988; Weiss, 1988; Duesberg, 1989c; Palca, 1990). Thus there is no unique genetic material and no uncommon genetic structure in HIV RNA that could indicate where this unique AIDS-specific information of HIV is hiding.

(2) Since all retroviral genes share just one common promoter, it would be impossible to differentially activate one HIV gene while the others are latent. Thus the idea that different viral genes would regulate latency and virulence, as with lambda phage, is not compatible with HIV (Haseltine and Wong-Staal, 1988; Eigen, 1989; Temin, 1990; Fauci, 1991). Since all HIV genes share the same promoter, latent HIV can only be activated by the host—just like all other latent retroviruses. In addition HIV cannot make specific AIDS factors, while its major genes are dormant. Since viral RNA synthesis *in vivo* is only detectable in 1 out of 10,000 to 100,000 leukocytes and then only in half of all AIDS patients (Section 3.3), HIV cannot make Kaposi’s sarcomagenic and neurotoxic factors in amounts sufficient to cause fatal tumors and dementias. This is why such factors have not been detectable *in vivo* (Weiss and Jaffe, 1990; Gallo, 1991).

Thus, based on the structure, information and function of its RNA, HIV is a profoundly conventional retrovirus. It does not contain unique genes that distinguish it from other retroviruses, nor can its genes be differentially regulated at the transcriptional level.

3.5.15. *Simian Retroviruses to Prove that HIV Causes AIDS*

Animal retroviruses may cause diseases in experimental animals that overlap with the wide spectrum of AIDS diseases. Such systems are now studied for analogies to gain experimental support for the virus-

AIDS hypothesis (Blattner *et al.*, 1988; Weiss and Jaffe, 1990; Goudsmit, 1992). For example, a retrovirus isolated from macaques (Fultz *et al.*, 1990), termed simian immunodeficiency virus (SIV), that is 40% related to HIV, is said to cause AIDS-like diseases in rhesus monkeys (Kestler *et al.*, 1990; Temin, 1990). According to an editorial in *Science*, “if SIV infection is all that is needed to cause simian AIDS, that’s one more indication that HIV is all that is needed to cause human AIDS” (Palca, 1990).

However, the presumed role of SIV in the diseases of infected monkeys is very different from that of HIV in human AIDS:

(1) According to one study, about half of the infected monkeys developed diseases within several months to one year after infection (Kestler *et al.*, 1990). By contrast only 3–4% of HIV-infected Americans or Europeans and 0.3% of infected Africans develop AIDS annually (Table 1).

(2) In the same study, the absence of antiviral antibodies predicted the incidence of diseases in monkeys, while the opposite is claimed for humans infected with HIV. Another study has confirmed that the monkey’s risk of disease is directly proportional to the titer of SIV (Fultz *et al.*, 1990).

(3) The simian retroviruses barely reduce the T-cell levels of ill monkeys (Kestler *et al.*, 1991), while HIV is claimed to deplete T-cells in humans.

(4) The spectrum of diseases observed in the SIV-infected monkeys is different from AIDS, including bacteremia and lacking, among others, Kaposi’s sarcoma and dementia (Kestler *et al.*, 1990; Fultz *et al.*, 1990).

(5) In follow-up studies, SIV failed to cause disease in rhesus and mangabey monkeys despite extensive sequence variation which is thought to enhance pathogenicity of the virus (Fultz *et al.*, 1990; Burns and Desrosiers, 1991; Villinger *et al.*, 1991).

(6) Since SIV has never caused any disease in wild monkeys, although about 50% are naturally infected (Duesberg, 1987, 1989c; Blattner *et al.*, 1988; Fultz *et al.*, 1990; Burns and Desrosiers, 1991; Villinger *et al.*, 1991), SIV is not an appropriate model for the hypothesis that HIV causes AIDS in naturally infected humans.

It would appear that SIV causes disease in monkeys like all viruses

cause disease soon after infection and in the absence of effective immunity. This is not a model for the hypothesis that HIV causes AIDS 10 years after it is neutralized by antibodies. Indeed, in the vast literature on retroviruses there is not even one proven example of a latent retrovirus that, in the presence of antiviral immunity, has ever caused a disease in any animal, including chickens, mice, cattle, and monkeys (Weiss *et al.*, 1985; Duesberg, 1987, 1989c).

Moreover, the observation that a retrovirus that is 60% unrelated to HIV causes disease in monkeys cannot prove that HIV causes AIDS in humans, even if all parameters of infection were completely analogous. It can only prove that under analogous conditions other retroviruses may also cause disease, which has been demonstrated with numerous avian and murine retroviruses long ago (Weiss *et al.*, 1985).

3.5.16. *Anecdotal AIDS Cases from the General Population*

Rare AIDS cases occurring outside the major risk groups are claimed to prove that HIV alone is sufficient to cause AIDS in persons with no other AIDS risks (Blattner *et al.*, 1988; Booth, 1988; Baltimore and Feinberg, 1989; Weiss and Jaffe, 1990). Four examples illustrate this point:

(1) Ryan White, an 18-year-old hemophiliac, was said to have died from AIDS in April 1990. However, information from the National Hemophilia Foundation revealed that White had died from unstoppable internal bleeding and had also been treated for an extended period with the cytotoxic DNA chain terminator AZT prior to his death (Duesberg and Ellison, 1990). It appears that hemophilia and AZT (Section 4) would each be sufficient causes of death, and certainly a combination of both would be more than adequate to explain the death of Ryan White. Thus there is no convincing evidence that White died from HIV.

To prove that HIV played a role in White's death, it would be necessary to compare mortality of matched hemophiliacs with and without HIV. To prove that AZT contributed to his death, matched HIV-positive hemophiliacs with and without AZT must be compared. Without such evidence the HIV-death of White is just a hypothesis. Yet White was generally described as an innocent victim of HIV (practicing no risk behavior), which is why the U.S. Senate approved the Ryan

White Comprehensive AIDS Resources Act for over \$550 million in aid to hospitals for AIDS emergencies and treatment of children (Anonymous, 1990).

(2) In 1989 the California tax-reformer Paul Gann was reported to have died from AIDS at the age of 77 after receiving HIV from a blood transfusion. However, a close examination of Gann's case reveals that he had 5-bypass heart surgery for blocked arteries in 1982, when he may have received the blood transfusion with HIV. In 1983 he needed further bypass surgery for blocked intestinal arteries. In 1989, at the age of 77, he was hospitalized again for a broken hip. While recovering from the hip fracture, Gann was immobilized for weeks and developed a pneumonia from which he died (Folkart, 1989). This is a rather typical death for a 77-year-old man in poor health.

To determine whether HIV played any role at all in his death, a controlled study would be necessary showing that the mortality of HIV-positive 77-year-old bypass patients with broken hips is higher than that of HIV-negative counterparts. No such study exists.

(3) Kimberly Bergalis, a 22-year-old woman, developed candidiasis and a transient pneumonia 17 and 24 months, respectively, after the extraction of two molars (Centers for Disease Control, 1990). After her dentist had publicly disclosed that "he had AIDS," she was tested for HIV, although Bergalis was a virgin and did not belong to an AIDS risk group (Breo and Bergalis, 1990). Since she was HIV-antibody-positive the CDC concluded that she had contracted AIDS from her dentist (Centers for Disease Control, 1990), who was a homosexual with Kaposi's sarcoma (Ou *et al.*, 1992).

Clearly prior to the virus-AIDS hypothesis, the story of a doctor transmitting his Kaposi's sarcoma in the form of a yeast infection to his client via a common infectious cause would have hardly made *The New York Times* and certainly not the scientific literature (Lambert, 1991). But since the two entirely unrelated diseases are both labeled AIDS and because of the tremendous popularity of the virus-AIDS hypothesis, the paradoxical story became a case célèbre for AIDS in the general population.

Once diagnosed for AIDS Bergalis was treated with the cytotoxic DNA chain terminator AZT, which is prescribed to inhibit HIV, until

she died in December 1991 with weight loss (15 kg), hair loss, uncontrollable candidiasis, anemia and muscle atrophy (requiring a wheelchair) (Breo and Bergalis, 1990; Anonymous, 1991; Lauritsen, 1991)—the symptoms of chronic AZT toxicity (Section 4). It is not clear whether her AZT therapy started before or after her pneumonia, since it was only mentioned in an edited interview conducted for the American Medical Association (Breo and Bergalis, 1990) and in some newspapers (Anonymous, 1991), but not in a single one of several scientific reports (Centers for Disease Control, 1990; Witte and Wilcox, 1991; Ou *et al.*, 1992; Palca, 1992a,b) and not in *The New York Times* (Lambert, 1991). Since her fatal condition was attributed to HIV, she received \$1 million in compensation from her dentist's, rather than from her AZT doctor's (Section 4), malpractice insurance (Palca, 1992a).

In view of the celebrity of the case and the fear it inspired among patients, 1100 further patients of the dentist came forward to be tested for HIV (Ou *et al.*, 1992; Palca, 1992a). Seven of these, including Bergalis, tested positive. Four or 5 of these, including Bergalis and another woman, did not belong to an AIDS risk group, but 2 or 3 did. At least three of those who did not belong to a risk group received \$1 million settlements from the dentist's malpractice insurance (Palca, 1992b). However, a plausible mechanism of HIV transmission from the dentist to his 4–5 positive clients without AIDS risks was never identified, and there is no consensus as to whether the viruses of the three carriers studied by the CDC and the insurance companies were sufficiently related to claim a common source (Palca, 1992a,b).

Statistically, it can be shown that the incidence of HIV-infections among the dentist's clients reflects, almost to the decimal point, the national incidence of the virus in the U.S. The national incidence of HIV-positives among all Americans is 0.4% (1 out of 250) (Table 1), the incidence of HIV-positives among 1100 patients of the Florida dentist was 0.4% (4 to 5 out of 1100) and the incidence among 15,795 patients from 32 HIV-positive doctors, determined by the CDC for the Bergalis case, was 0.5% (84 out of 15,795). Thus the incidence of HIV in patients from HIV-positive doctors reflects the national incidence of HIV. This suggests noniatrogenic and, most likely, perinatal infection as the source of HIV in these patients, particularly in the case of the virgin Bergalis

(Section 3.5.2). In addition, it identifies a rich source of insurance income for 0.4% of American patients of HIV-positive doctors!

To determine whether HIV had contributed to Bergalis' death, a controlled study would be necessary comparing the mortality of women with yeast infections, with and without antibodies against HIV, and with and without AZT therapy. Since such a study is not available, the assumption that Bergalis died from HIV is pure speculation.

(4) A doctor, presumably infected with HIV from a needle stick in 1983 (Aoun, 1992), described himself in a letter to the *New England Journal of Medicine* as an AIDS patient (Aoun, 1989). He was diagnosed HIV-positive in 1986 (Aoun, 1992). His only AIDS symptom at that time was a weight loss of 4.5 kg (Aoun, 1989). In 1991, then 8 years after the presumed date of the infection, the doctor described his case again in a speech "From the eye of the storm . . ." published in the *Annals of Internal Medicine* (Aoun, 1992). The speech did not describe any current AIDS symptoms. This case has been cited as an example that HIV is sufficient to cause AIDS (Baltimore and Feinberg, 1989).

However, the weight loss diagnosed in 1986 could have been the result of the anxiety that HIV infection causes in believers of the HIV-AIDS hypothesis, rather than the work of HIV. This interpretation is consistent with the fact that since 1985 at least 800,000 Americans (1 million minus the 206,000 AIDS cases recorded by the end of 1991; see Table 1) have not lost weight or developed other AIDS diseases (Duesberg, 1991a). Likewise, 6 million Central Africans (minus the 129,000 with AIDS) have been healthy HIV-carriers since at least 1985 (Table 1).

Thus, there are no convincing anecdotal cases to prove that HIV causes AIDS in persons outside the major risk groups. The use of the above assumptions and anecdotal cases as proof for the virus-AIDS hypothesis is misleading, although they may provide valuable clues for future research.

3.6. Consequences of the Virus-AIDS Hypothesis

Despite the lack of proof and numerous discrepancies with orthodox criteria of infectious disease, the virus-AIDS hypothesis has remained since 1984 the only basis for all efforts in predicting, preventing, investigating and even treating AIDS. AIDS prevention is based entirely on

preventing the spread of HIV. This includes promotion of safe sex (Booth, 1988; Institute of Medicine, 1988; Weiss and Jaffe, 1990; Mann and the Global AIDS Policy Coalition, 1992; Anderson and May, 1992), clean injection equipment for intravenous drugs (National Commission on AIDS, 1991) and the exclusion of HIV antibody-positive blood donations from transfusions (Vermund, 1991; Duesberg and Schwartz, 1992).

The Food and Drug Administration mandated in 1985 that the 12 million plus annual blood donations in the U.S. (Williams *et al.*, 1990) are tested for HIV-1, and as of 1992 also for HIV-2, although there is as yet only one single American AIDS patient infected by HIV-2 (O'Brien *et al.*, 1992). Since 1985 over 2 million tests have also been performed annually by the U.S. Army (Burke *et al.*, 1990). By 1986 already over 20 million "AIDS tests" were performed in the U.S. (Institute of Medicine, 1986), at a minimum cost to the client of \$12 to \$70 (Irwin Memorial Blood Bank, San Francisco, personal communication) or \$45 (U.S. Immigration Service). The former U.S.S.R. conducted 20.2 million "AIDS tests" in 1990 and 29.4 million in 1991 to detect 112 and 66 antibody-positives, respectively (Voevodin, 1992).

The detection of antibodies in healthy persons is interpreted as a 50% certain prognosis for AIDS within 10 years (Section 3.5.12). Therefore, a positive "AIDS test" is psychologically toxic (Grimshaw, 1987; Albonico, 1991b) and often the basis for the physiologically toxic antiviral therapy with AZT (Section 4) (Duesberg, 1992b,d). A negative test for HIV is a condition for admission to the U.S. Army (Burke *et al.*, 1990), for admission to health insurance programs, for residence in many countries and even for travel into the U.S. and China. Currently, over 50 countries restrict one or more classes of entrants based on positive antibody-tests for HIV (Duckett and Orkin, 1989). Antibody-positive Americans who had sex with antibody-negatives have been convicted of "assault with a deadly weapon" (Duesberg, 1991c; McKee, 1992). In communist Cuba about 600 antibody-positive persons are quarantined in the name of the virus-AIDS hypothesis (Scheper-Hughes and Herrick, 1992; Treichler, 1992).

Based on the assumption that HIV had either originated recently or spread recently from isolation to its current levels, at the same rates as AIDS had spread in the risk groups in the U.S. and Europe, and on the

assumption that AIDS would follow the presumed spread of HIV with a hiatus of 10 years, epidemiologists have made apocalyptic predictions about an AIDS epidemic that has raised fears and funding to unprecedented levels (Heyward and Curran, 1988; Mann *et al.*, 1988; Mann and the Global AIDS Policy Coalition, 1992; Anderson and May, 1992).

Above all, over 180,000 antibody-positives, with and without AIDS, are currently treated indefinitely with the cytotoxic DNA chain terminator AZT in an effort to inhibit HIV (Section 4.4).

4. The Drug-AIDS Hypothesis

After the global acceptance of the virus-AIDS hypothesis, several investigators have recently revived the original hypothesis that AIDS is not infectious (Section 2.2). In view of (1) the almost complete restriction (97%) of American AIDS to groups with severely compromised health, (2) the predetermination for certain AIDS diseases by prior health risks, and (3) the many links between AIDS and drug consumption (Sections 2.1.3 and 3.4, Table 2), it has been proposed that recreational drugs and AZT may cause AIDS (Lauritsen and Wilson, 1986; Haverkos, 1988a, 1990; Holub, 1988; Papadopoulos-Eleopoulos, 1988; Rappoport, 1988; Duesberg, 1990a, 1991a, 1992c,f; Lauritsen, 1990; Albonico, 1991a,b; Pillai *et al.*, 1991; Cramer, 1992; Leonhard, 1992). Here the hypothesis is investigated that all American and European AIDS diseases, above the normal background of hemophilia and transfusion-related diseases, are the result of the long-term consumption of recreational and anti-HIV drugs.

4.1. Chronological Coincidence Between the Drug and AIDS Epidemics

The appearance of AIDS in America in 1981 followed a massive escalation in the consumption of psychoactive drugs that started after the Vietnam War (Newell *et al.*, 1985b; Kozel and Adams, 1986; National Institute on Drug Abuse, 1987; Bureau of Justice Statistics, 1988; Haverkos, 1988b; Office of National Drug Control Policy, 1988; Flanagan and Maguire, 1989; Lerner, 1989; Shanon *et al.*, 1990). The Bureau of Justice Statistics reports that the number of drug arrests in the U.S.

has increased from about 450,000 in 1980 to 1.4 million in 1989 (Bureau of Justice Statistics, 1988; Shannon *et al.*, 1990). About 500 kg of cocaine were confiscated by the Drug Enforcement Administration in 1980, about 9000 kg in 1983, 80,000 kg in 1989, and 100,000 kg in 1990 (Bureau of Justice Statistics, 1988, 1991; Flanagan and Maguire, 1989). In 1974, 5.4 million Americans had used cocaine at some point in their lives and in 1985 that number had gone up to 22.2 million (Kozel and Adams, 1986). Currently about 8 million Americans are estimated to use cocaine regularly (Weiss, S.H., 1989; Finnegan *et al.*, 1992). The number of dosage units of domestic stimulants confiscated, such as amphetamines, increased from 2 million in 1981 to 97 million in 1989 (Flanagan and Maguire, 1989).

Several arguments indicate that these increases reflect increased drug consumption rather than just improved drug control, as has been suggested (Maddox, 1992a):

(1) The Bureau of Justice Statistics estimates that at most 20% of the cocaine smuggled into the U.S. was confiscated each year (Anderson, 1987).

(2) The National Institute on Drug Abuse reports that between 1981 and 1990 cocaine-related hospital emergencies increased 24-fold from 3296 to 80,355 and deaths from 195 to 2483 (Kozel and Adams, 1986; National Institute on Drug Abuse, 1990a,b). Thus cocaine-related hospital emergencies had increased 24-fold during 9 of the 10 years in which cocaine seizures had increased 100-fold.

(3) It is highly improbable that, before the jet-age, the U.S. would have imported annually as much cocaine as it did in 1990 plus the 100,000 kg that were confiscated in that year.

Further, the recreational use of psychoactive and aphrodisiac nitrite inhalants began in the 1960s and reached epidemic proportions in the mid-1970s, a few years before AIDS appeared (Newell *et al.*, 1985b, 1988). The National Institute on Drug Abuse reports that in 1979–1980 over 5 million people used nitrite inhalants in the U.S. at least once a week (Newell *et al.*, 1988), a total of 250 million doses per year (Wood, 1988). In 1976 the sales of nitrite inhalants in one American city alone amounted to \$50 million annually (Newell *et al.*, 1985b, 1988) at \$5 per 12 mL dose (Schwartz, 1988).

Since 1987 the cytotoxic DNA chain terminator AZT has been prescribed as an anti-HIV drug to AIDS patients (Kolata, 1987; Yarchoan and Broder, 1987b) and since 1990 to asymptomatic carriers of HIV (Editorial, 1990). Currently about 120,000 Americans and 180,000 HIV-positive persons worldwide, with and without AIDS, take AZT in efforts to inhibit HIV. This estimate is based on the annual AZT sales of \$364 million and a wholesale price of \$2000 per year for a daily dose of 500 mg AZT per person (Burroughs Wellcome Public Relations, 3 April 1992). In addition, an unknown number take other DNA chain terminators like ddI and ddC (Smothers, 1991; Yarchoan *et al.*, 1991).

4.2. Overlap Between Drug-Use and AIDS Statistics

Drugs and AIDS appear to claim their victims from the same risk groups. For instance, the CDC reports that the annual mortality of 25- to 44-year-old American males increased from 0.21% in 1983 to 0.23% in 1987, corresponding to about 10,000 deaths among about 50 million in this group (Buehler *et al.*, 1990). Since the annual AIDS deaths had also reached 10,000 by 1987, HIV was assumed to be the cause (Institute of Medicine, 1986; Centers for Disease Control, 1987, 1992b). Further, HIV infection was blamed for a new epidemic of immunological and neurological deficiencies, including mental retardation, in American children (Blattner *et al.*, 1988; Institute of Medicine, 1988; Centers for Disease Control, 1992b).

However, mortality in 25- to 44-year-old males from septicemia, considered an indicator of intravenous drug use, rose almost 4-fold from 0.46 per 100,000 in 1980 to 1.65 in 1987, and direct mortality from drug use doubled (National Center for Health Statistics, 1989; Buehler *et al.*, 1990), indicating that drugs played a significant role in the increased mortality of this group (Buehler *et al.*, 1990). In addition, deaths from AIDS diseases and nonAIDS pneumonia and septicemia per 1000 intravenous drug users in New York increased at exactly the same rates, from 3.6 in 1984 to 14.7 and 13.6, respectively, in 1987 (Selwyn *et al.*, 1989). Indeed, the cocaine-related hospital emergencies alone could more than account for the 32% of American AIDS patients that are intravenous drug users (Section 2.1.3). The emergencies had increased from "a negligible number of people" in 1973 to 9946 non-fatal and 580

fatal cases in 1985 (Kozel and Adams, 1986), when a total of 10,489 AIDS cases were recorded and to 80,355 nonfatal and 2483 fatal cases in 1990 (National Institute on Drug Abuse, 1990a,b), when a total of 41,416 AIDS cases were recorded by the CDC (Centers for Disease Control, 1992a). Moreover 82% of the cocaine-related and 75% of the morphine-related hospital emergencies were 20–39 years old (National Institute on Drug Abuse, 1990a), the age distribution typical of AIDS patients (Section 2.1.1).

Another striking coincidence is that over 72% of all American AIDS patients (Centers for Disease Control, 1992b) and about 75% of all Americans who consume “hard” psychoactive drugs such as cocaine, amphetamines and inhalants (National Institute on Drug Abuse, 1987, 1990a,b; Ginzburg, 1988) or get arrested for possession of such drugs (Bureau of Justice Statistics, 1988) or are treated for such drugs (National Institute on Drug Abuse, 1990a) are 20- to 44-year-old males. Thus there is substantial epidemiological overlap between the two epidemics (Lerner, 1989), reported as “The twin epidemics of substance use and HIV” by the National AIDS Commission (National Commission on AIDS, 1991).

Moreover, maternal drug consumption was blamed by some for the new epidemic of immunological and neurological deficiencies, including dementias, of American children (Toufexis, 1991). In view of this, the CDC acknowledges, “We cannot discern, however, to what extent the upward trend in death rates from drug abuse reflects trends in illicit drug use independent of the HIV epidemic” (Buehler *et al.*, 1990).

4.3. Drug Use in AIDS Risk Groups

4.3.1. Intravenous Drug Users Generate a Third of All AIDS Patients

Currently 32% of the American (National Commission on AIDS, 1991; Centers for Disease Control, 1992b) and 33% of the European (Brenner *et al.*, 1990; World Health Organization, 1992a) AIDS patients are intravenous or intrauterine users of heroin, cocaine, and other drugs (Section 2.1.3). These include:

(1) 75% of all heterosexual AIDS cases in America and about 70% of those in Europe,

(2) 71% of the American and 57% of the European females with AIDS,

(3) over 10% of the American and 5% of the European male homosexuals,

(4) 10% of the American hemophiliacs with AIDS,

(5) 70% of American children with AIDS including 50% born to mothers who are confirmed intravenous drug users and another 20% to mothers who had “sex with intravenous drug users” and are thus likely users themselves (Amaro *et al.*, 1989),

(6) 80–85% of the European children with AIDS who were born to drug-addicted mothers (Mok *et al.*, 1987; European Collaborative Study, 1991).

In an article entitled “AIDS and intravenous drug use: the real heterosexual epidemic” the AIDS researcher Moss points out that “90% of infected prostitutes reported in Florida, Seattle, New York and San Francisco have been intravenous drug users . . . Drug use is also the source of most neonatal AIDS, with 70% of cases occurring in children of intravenous drug users . . .” (Moss, 1987). Indeed, all studies of American and European prostitutes indicate that HIV infection is almost exclusively restricted to drug users (Rosenberg and Weiner, 1988), although all prostitutes should have the same risks of HIV infection, if HIV were sexually transmitted. Surprisingly, all of these studies only mention the incidence of HIV, rather than of AIDS, in prostitutes.

4.3.2. Homosexual Users of Aphrodisiac Drugs Generate about 60% of AIDS Patients

Approximately 60% of American AIDS patients are male homosexuals over the age of 20 (Table 1). They are generated by risk groups that have sex with large numbers of partners (Centers for Disease Control, 1982; Jaffe *et al.*, 1983b; Darrow *et al.*, 1987; Oppenheimer, 1992) that often average over 100 per year and have exceeded 1000 over a period of several years (Mathur-Wagh *et al.*, 1984; Newell *et al.*, 1985a; Turner *et al.*, 1989; Callen, 1990). The following evidence indicates that these sexual activities and the corresponding conventional venereal diseases are directly proportional to the consumption of toxic sexual stimulants, which include nitrite- and ethylchloride inhalants, cocaine, ampheta-

mines, methaqualone, lysergic acid, phenylcyclidine, and more (Blattner *et al.*, 1985; Shilts, 1987; Lauritsen and Wilson, 1986; Darrow *et al.*, 1987; Haverkos, 1988a; Rappoport, 1988; Raymond, 1988; Adams, 1989; Turner *et al.*, 1989; Weiss, S.H., 1989; Ostrow *et al.*, 1990; Lesbian and Gay Substance Abuse Planning Group, 1991a).

An early CDC study of 420 homosexual men attending clinics for sexually transmitted diseases in New York, Atlanta and San Francisco reported that 86.4% had frequently used amyl- and butylnitrites as sexual stimulants. The frequency of nitrite use was proportional to the number of sexual partners (Centers for Disease Control, 1982).

In 1983 Jaffe *et al.* investigated AIDS risk factors of 170 male homosexuals from sexual disease clinics, including 50 with Kaposi's sarcoma and pneumonia and 120 without AIDS. In this group, 96% were regular users of nitrite inhalants and 35–50% of ethylchloride inhalants. In addition, 50–60% had used cocaine, 50–70% amphetamines, 40% phenylcyclidine, 40–50% lysergic acid, 40–60% methaqualone, 25% barbiturates, 90% marijuana and 10% heroin (Jaffe *et al.*, 1983b). Over 50% had also used prescription drugs. About 80% of these men had past or current gonorrhoea, 40–70% had syphilis, 15% mononucleosis, 50% hepatitis and 30% parasitic diarrhoea. Those with Kaposi's sarcoma had a median of 61 sex partners per year and those without AIDS about 26. The study points out that "lifetime exposure to nitrites . . . (and) use of various 'street' drugs . . . was greater for cases than controls." The lifetime drug dose of "cases" was reported to be two times higher than of asymptomatic HIV carriers (Jaffe *et al.*, 1983b).

A study of a group of 359 homosexual men in San Francisco reported in 1987 that 84% had used cocaine, 82% alkyl nitrites, 64% amphetamines, 51% methaqualone, 41% barbiturates, 20% injected drugs and 13% shared needles (Darrow *et al.*, 1987). About 74% had past or current infection by gonococcus, 73% by hepatitis B virus, 67% by HIV, 30% by amoebae and 20% by treponema (Darrow *et al.*, 1987). This group had been randomly selected from a list of homosexuals who had volunteered to be investigated for hepatitis B virus infection and to donate antisera to hepatitis B virus between 1978 and 1980. For the same group the 50% "progression rate" from HIV to AIDS was calculated to be 8–11 years (Table 2) (Moss *et al.*, 1988; Lemp *et al.*, 1990)—

and reported to be relevant for “the (HIV-infected) population as a whole” (Moss *et al.*, 1988)!

A study investigating AIDS risk factors among French homosexuals reported that 31% of those with AIDS, but only 12% of those without AIDS, had achieved “over 100 nitrite inhalations” (Messiah *et al.*, 1988). The study included 53, or 45%, of all homosexual AIDS patients recorded in France by 1987.

The staggering oral drug use among male homosexuals at risk for AIDS was confirmed in 1990 by the largest survey of its kind. It reports that 83% of 3916 self-identified American homosexual men had used one, and about 60% two or more drugs with sexual activities during the previous six months (Ostrow *et al.*, 1990). Similar drug use has been reported for European homosexuals at risk for AIDS (van Griensven *et al.*, 1987).

A survey of homosexual men from Boston, conducted between 1985 and 1988, documented that among 206 HIV-positives 92% had used nitrite inhalants, 73% cocaine, 39% amphetamines, 29% lysergic acid in addition to six other psychoactive drugs as sexual stimulants; among 275 HIV-negative controls 71% had used nitrites, 57% cocaine, 21% amphetamines, 17% lysergic acid again in addition to six other psychoactive drugs (Seage *et al.*, 1992). A similar survey of 364 HIV-positive homosexual men in Berlin conducted between 1983 and 1987 stated that 194 (53.3%) had used nitrite inhalants (Deininger *et al.*, 1990).

According to Newell *et al.* (1985b), volatile nitrites had penetrated “every corner of gay life” by 1976. Surveys studying the use of nitrite inhalants found that in San Francisco 58% of homosexual men were users in 1984 and 27% in 1991, compared to less than 1% of heterosexuals and lesbians of the same age group (Lesbian and Gay Substance Abuse Planning Group, 1991b).

Several investigators have pointed out that nitrite inhalants, and possibly other drugs, are preferred by male homosexuals as aphrodisiacs because they facilitate anal intercourse by relaxing smooth muscles (Section 4.4.1) (Mirvish and Haverkos, 1987; Newell *et al.*, 1985b; Ostrow *et al.*, 1990; Lesbian and Gay Substance Abuse Planning Group, 1991a; Seage *et al.*, 1992). “Nitrites were used primarily for heightened sexual stimulation during sexual activity by reducing social and sexual inhi-

bitions, prolonging duration, heightening sexual arousal, relaxing the anal sphincter during anal intercourse, and prolonging orgasm” (Newell *et al.*, 1985b).

4.3.3. Asymptomatic AZT Users Generate an Unknown Percentage of AIDS Patients

The DNA chain terminator AZT has been licensed in the U.S. since 1987 as a treatment for AIDS patients (Chernov, 1986; Kolata, 1987; Lauritsen, 1990; Yarchoan *et al.*, 1991) based on a placebo controlled study sponsored by Burroughs Wellcome, the manufacturer of AZT (Section 4.4.2) (Fischl *et al.*, 1987; Richman *et al.*, 1987). In 1990 AZT was also licensed as AIDS prophylaxis for healthy HIV carriers (Section 4.4.2) (Volberding *et al.*, 1990; Yarchoan *et al.*, 1991).

The choice of this drug as anti-AIDS treatment is based entirely on the virus-AIDS hypothesis. According to Broder *et al.*, “The rationale for anti-retroviral therapy for AIDS is . . . that HIV is the etiologic agent of AIDS” and that HIV RNA-dependent DNA synthesis is inhibited by AZT (Yarchoan *et al.*, 1991). In view of this and their faith in the virus-AIDS hypothesis, about 120,000 American HIV carriers, with and without AIDS, and 180,000 worldwide currently take AZT every day (Section 4.1). It follows that probably a high percentage of the 40,000 Americans and 15,000 Europeans that currently develop AIDS per year (Table 1) have used AZT and other DNA chain terminators prior to AIDS.

The drug is now recommended as AIDS prophylaxis for all AIDS-free persons with less than 500 T-cells per microliter by the director of AIDS research at the NIH (Kolata, 1992) and with some reservations also by the National Hemophilia Association of New York (personal communication), despite recent doubts about its usefulness (Kolata, 1992). For instance, AZT has been used indefinitely by over 1200 AIDS-free, but presumably HIV-infected, homosexual men from the Multi-center AIDS Cohort Study referenced above (Ostrow *et al.*, 1990), including 7% of 3670 with over 500 T-cells per microliter, 16% of 1921 with 350–499 T-cells, 26% of 1374 with 200–349 T-cells and 51% of 685 with fewer than 200 T-cells (Graham *et al.*, 1991). Yet the large study acknowledges finding “. . . no effects (of AZT) on rates of progression

to lower CD4+ lymphocyte counts in any of the transition intervals” (Graham *et al.*, 1991). In San Francisco 3.3% of 151 AIDS-free male homosexuals with over 500 T-cells, 11% of 128 with 200–500 T-cells and 36% of 42 with less than 200 T-cells were on AZT in 1989 (Lang *et al.*, 1991). Another study reports that, in 1989, 26 out of 322 HIV-positive but AIDS-free homosexuals from San Francisco, Chicago and Denver had taken AZT for less than 6 months and 101 for over 6 months (Holmberg *et al.*, 1992).

To distinguish between HIV and drugs as causes of AIDS, it is necessary to identify either HIV-carriers that develop AIDS only when they use drugs (Section 4.4) or to identify HIV-free drug users that develop AIDS indicator diseases (Section 4.5) and to demonstrate drug toxicity (Section 4.6).

4.4. Drug Use Necessary for AIDS in HIV-Positives

Studies demonstrating that drugs are necessary for AIDS among HIV-positives fall into two subgroups: (1) those demonstrating that AIDS among HIV-positives depends on the long-term use of recreational drugs and (2) those demonstrating that HIV-positive AIDS-free persons and AIDS patients on the antiviral drug AZT develop new AIDS diseases or AZT-specific diseases. Since the health of AIDS-free persons selected for AZT prophylaxis is compromised by prior AIDS risks, e.g. less than 500 T-cells, and since nearly all American and European AIDS patients have used recreational drugs or have been immunosuppressed by long-term transfusions, evaluating the role of AZT in the progression of AIDS is complicated by these confounding risk factors (Sections 3.4.4 and 4.3.3).

4.4.1. AIDS From Recreational Drugs

(1) A study of 65 HIV-infected drug users from New York showed that their T-cell count dropped over nine months in proportion with drug injection, on average 35%, compared to controls who had stopped (Des Jarlais *et al.*, 1987).

(2) The incidence of AIDS diseases and death among HIV-positive, asymptomatic intravenous drug users over 16 months were 19% (23/124)

among those who persisted in injecting psychoactive drugs, 5% (5/93) among those who had stopped injecting drugs and 6% (5/80) among those on methadone treatment (Weber *et al.*, 1990).

(3) Among male homosexuals, receptive anal intercourse carries a 2.75 times (Warren Winkelstein, personal communication) to 4.4 times (Haverkos, 1988b) higher AIDS risk than insertive intercourse, presumably reflecting a higher risk of infection by HIV (Moss *et al.*, 1987; van Griensven *et al.*, 1987; Winkelstein *et al.*, 1987; Seage *et al.*, 1992). However, if HIV were the cause of AIDS, the donors should have the same AIDS risk as the recipients, because recipients can only be infected by HIV donors. No microbe can survive that is only unidirectionally transmitted. All venereal microbes are therefore bitransitive. Indeed, Haverkos found no differences in sexually transmitted diseases between those practicing receptive and insertive intercourse (Haverkos, 1988b). The probable reason for the higher AIDS risk associated with receptive anal intercourse is that this sexual practice directly correlates with a 2-fold (van Griensven *et al.*, 1987; Seage *et al.*, 1992) to an 8-fold (Moss *et al.*, 1987; Haverkos, 1988b) enhanced use of nitrite inhalants and other aphrodisiac drugs that facilitate anal intercourse (Sections 4.3.2 and 4.6).

(4) A Canadian study reports that every one of 87 HIV-positive male homosexual AIDS patients had used nitrite inhalants. Those who had used over 20 "hits" per month were more likely to have Kaposi's sarcoma and sarcoma plus pneumonia than those who had used less than 20 hits per month. HIV-free controls, described in a previous report of the same cohort (Section 4.5) (Marion *et al.*, 1989), were not mentioned in this study (Archibald *et al.*, 1992). The authors concluded that a "sexually transmitted agent," which is even more difficult to transmit than HIV(!) (Section 3.5.1), would explain the Kaposi's sarcomas among the AIDS patients. The nitrites were proposed to be a cofactor of this cofactor of HIV (Archibald *et al.*, 1992). Thus nitrites were necessary for AIDS in HIV-positives.

To determine whether HIV was indeed necessary for these AIDS cases, the incidence of AIDS-defining diseases in HIV-positive and negative homosexuals who are matched for the duration and extent of drug consumption must be compared. This is what the Canadian team has

recently attempted to do in a study termed “HIV causes AIDS: a controlled study” (Craib *et al.*, 1992). The study asserts to meet the challenge of “Duesberg [who] wrote in 1988 (*Science*, 1988; 242: 997–998) and repeated in public addresses in 1991 that the necessary comparisons in controlled cohorts were not available. . . .”

However the study failed to match the HIV-free control group with the HIV-positives for the extent and duration of drug consumption. It mentions that 49% of the HIV-negatives had used “psychoactive drugs,” but fails to mention the percentage of drug users among the HIV-positives. In their previous study 100% of the HIV-positive AIDS patients had used such drugs (Archibald *et al.*, 1992). In addition the authors failed to recognize that HIV-infection is a marker for the duration of drug consumption. Since an average of 1000 sexual contacts is required for sexual transmission of HIV (Section 3.5.2), HIV is a marker for the dosage of sexual stimulants that is used for 1000 contacts. Thus HIV-positives would have used more sexual stimulants, the equivalent for 1000 contacts, than HIV-negatives. Indeed the authors acknowledge problems with “claims that AIDS is caused by other exposures and not by HIV . . . the problem may be semantics. No one has ever disputed that cofactors play a very important role . . .” (Craib *et al.*, 1992). Moreover the authors failed to mention whether AZT was prescribed to the HIV-positives.

(5) A survey of 99, including 92 “gay or bisexual,” AIDS patients from an “HIV clinic” at St. Mary’s Hospital in London reports that 78% used “poppers” (nitrite inhalants), 78% cannabis, 76% cigarettes, 68% alcohol, and 48% “ecstasy” (amphetamines). In addition, the patients received an average of three unspecified medications, probably including AZT (Valentine *et al.*, 1992). HIV-tests were not reported, but are assumed to be positive because the patients were in an “HIV clinic.” Clearly, the multiplicity of drugs consumed by these patients could be relevant to their pathogenesis.

(6) A European survey of HIV-positive infants with AIDS found that “nearly all children were born to intravenous-drug-abusing mothers” and that AIDS was 9.4 times more likely in children whose mothers had AIDS symptoms before delivery than in those who had no symptoms (Mok *et al.*, 1987). “Children with drug withdrawal symp-

toms” were most likely to develop diseases, those with no withdrawal symptoms but “whose mothers had used recreational drugs in the final 6 months of pregnancy were intermediate on all indices, whereas children of former drug users did not significantly differ from those born to women who had no history of i.v. drug use” (European Collaborative Study, 1991). An American survey reported that 63 of 68 infants “with symptomatic HIV infections” had “at least one parent who had AIDS or was in an AIDS high-risk group” (Belman *et al.*, 1988). Since the risk of infants to develop AIDS increased with maternal drug consumption and increased 10-fold with maternal AIDS symptoms, it would appear that disease or subclinical deficiencies during pregnancy rather than perinatal infection by HIV are responsible for pediatric AIDS.

4.4.2. AIDS from AZT and AZT Plus Confounding Recreational Drug Use

(1) A placebo-controlled study, sponsored by Burroughs Wellcome the manufacturer of AZT, investigated 289 patients with “unexplained” weight loss, fever, oral candidiasis, night sweats, herpes zoster and diarrhea for the licensing of the drug as AIDS therapy in the U.S. (Fischl *et al.*, 1987; Richman *et al.*, 1987). All but 13 of these patients were males. The study was planned for 6 months, but it was interrupted after 4 months, because by then the therapeutic benefits of AZT seemed too obvious to continue the placebo control:

(a) after 4 months on AZT 1 out of 145 in the AZT group but 19 out of 137 in the placebo group had died. Therefore the study claimed that AZT can “decrease mortality”;

(b) T-cell counts first increased from 4–8 weeks and then declined to pretreatment levels within 4 months;

(c) the lymphocyte count decreased over 50% in 34% of the AZT recipients but in only 6% of the control group;

(d) 66 in the AZT group suffered from severe nausea, compared to only 25 in the control group;

(e) muscle atrophy was observed in 11 AZT recipients but in only 3 from the control group. Yet, the primary claim of the study, “decreased mortality” from AZT is not realistic if one considers that 30 out of the 145 in the AZT-group depended on multiple transfusions to survive

anemia, compared to only 5 out of the 137 in the placebo group. Thus the number of subjects in the AZT-group who would have died from severe anemia if untreated was larger, i.e. 30, than the AIDS deaths and anemias of the control group combined, namely 19+ 5. The “decreased mortality”-claim is further compromised by numerous “concomitant medications” other than transfusions for AZT-specific diseases and failure to match the AZT and placebo groups for the cumulative effects of prior and parallel recreational drug use. In addition some of the AZT-specific AIDS diseases observed in the placebo group appear to be due to patient-initiated “drug sharing” between AZT and placebo recipients (Lauritsen, 1990; Duesberg, 1992d; Freestone, 1992) and falsification of the case report forms (Lauritsen, 1992).

Moreover the low mortality of 0.7% (1/145) claimed by the licensing study for the first 4 months on AZT could not be extended in a follow-up study which found the “survival benefits” of AZT rapidly declining after the original 4 month period. By 18 months 32% of the original AZT group had died and 35% of the former control group, which by then had also received AZT for 12 months (Fischl *et al.*, 1989).

Since the original study considered AZT effective in decreasing AIDS mortality, subsequent placebo-controlled studies were deemed unethical. But the low mortality claimed by the licensing study has not been confirmed by later studies, which observed mortalities of 12–72% within 9–18 months (see items (3) to (6) below). In addition, a CDC study has recently reported a mortality of 82% in a cohort of 55 AIDS patients that had been on AZT for up to 4 years (Centers for Disease Control, 1991)—hardly recommending AZT as an AIDS therapy.

The brief transient gains of T-cells observed upon AZT treatment by the licensing study may reflect compensatory hemopoiesis, random killing of pathogenic parasites (Elwell *et al.*, 1987) and the influence of concomitant medication, including multiple transfusions (Richman *et al.*, 1989). Indeed the study concluded, based on the “hematological toxicity” described above, that “... the initial beneficial immunological effects of AZT may not be sustained” (Richman *et al.*, 1987). A French study confirms “... the decrease of cell counts below the initial value after a few months of AZT suggests that this drug might be toxic to cells” (see item (3) below) (Dournon *et al.*, 1988). And a recent Amer-

ican study also confirms "... no effects on rates of progression to lower CD4 + lymphocyte counts in (6 month) transition intervals" (Section 4.3.3) (Graham et al., 1991). Moreover, the manufacturer states, "A modest increase in mean CD4 (T4) counts was seen in the zidovudine group but the significance of this finding is unclear as the CD4 (T4) counts declined again in some patients" (Medical Economics Data, 1992).

(2) In view of the reported success of AZT as AIDS therapy, the drug was also tested for licensing as AIDS prophylaxis by much of the same team, including Fischl, Richman and Volberding, and again with support from the manufacturer Burroughs Wellcome (Volberding *et al.*, 1990). The study treated AIDS-free, HIV-positive 25- to 45-year-old male homosexuals and intravenous drug users with "fewer than 500 T-cells" for one year either with AZT or with a placebo. The expected annual AIDS risk for intravenous drug users and male homosexual risk groups is about 4–6% per year without AZT (Section 3.4.4.4).

The study reports AIDS diseases in: (1) 11 out of 453 on 500 mg AZT per day, (2) 14 out of 457 on 1500 mg AZT per day and (3) 33 out of 428 on a placebo (Volberding et al., 1990). Thus the AZT-groups appeared to do better than expected and the placebo group did as expected. Therefore it was claimed that AZT prevents AIDS.

However, the price for the presumed savings of 22 (33–11) and 19 (33–14) AIDS cases with AZT, compared to the placebo group, was high because 19 AZT-specific cases of potentially fatal anemia, neutropenia and severe nausea appeared in the 500 mg AZT-group, and 72 such cases, including 29 anemias requiring life-saving blood transfusions, appeared in the 1500 mg AZT-group. This indicates cytotoxic effects of AZT on hemopoiesis and on the intestines. Although the AZT-specific diseases were not diagnosed as AIDS, neutropenia generates immunodeficiency (Walton *et al.*, 1986) and thus AIDS. If these AZT-specific cases were included in the calculation of benefits from AZT compared to the placebo group, the 500 mg-group no longer benefited and the 1500 mg-group tripled its disease risk.

The study was further compromised by its failure to match the treatment groups for their cumulative recreational drug use prior to and during the study and for the many compensatory treatments for the

AZT-specific diseases of the subjects analyzed. The fact that 8 cases in the control group but only 3 and 1 in the 500 mg and 1500 mg-AZT groups developed AIDS cancers suggests that the control group could have been exposed to higher recreational drug doses.

Since the licensing study considered AZT effective in preventing AIDS, subsequent controlled trials were deemed unethical. However, several subsequent studies cast further doubt on the claim that AZT is a useful AIDS prophylactic. One study reported that persons with “early” AIDS, i.e. AIDS-free persons at risk for AIDS, died at the same rate of 12–14% as AIDS controls and that 82% developed leukopenia within less than a year (see item 6 below) (Hamilton *et al.*, 1992). Another study described “no effects on rates of progression to lower CD4+ lymphocytes . . .” recorded within 6 month periods in over 1200 AIDS-free men on AZT (Section 4.3.3) (Graham *et al.*, 1991). A third study reported that 26 out of 127 HIV-positive, AIDS-free homosexuals had discontinued an unreported dose of AZT within less than 6 months, most because of severe toxicity (Section 4.3.3) (Holmberg *et al.*, 1992). In view of these and other data, it is surprising that a loss of T-cells was not noted in the licensing study (Kolata, 1987).

(3) A French study investigated the effects of AZT on 365 AIDS patients. The patients included 72% male homosexuals and 11% intravenous drug users with a median age of 36 years and with opportunistic infections and Kaposi’s sarcoma. The study, the largest of its kind, observed new AIDS diseases, including leukopenia, in over 40% and death in 20% within 9 months on AZT (Dournon *et al.*, 1988). The AIDS diseases of 30% worsened during AZT treatment. The study reported no therapeutic benefits 6 months after initiating AZT therapy. The authors concluded: “. . . the rationale for adhering to high-dose regimens of AZT, which in many instances leads to toxicity and interruption of treatment, seems questionable.”

(4) A Dutch study treating 91 male AIDS patients, averaging 39 years, after 67 weeks on AZT, observed mortality in 72% and AZT-specific myelotoxicity, requiring on average 5 blood transfusions, in 57%. About 34% of the myelotoxicity manifested in anemia and 20% in leukopenia. The authors concluded that “the majority of patients . . . cannot be maintained on these (AZT) regimens, most commonly due

to the development of hematological toxicity” (van Leeuwen *et al.*, 1990).

(5) An Australian study involving 308 homosexual and bisexual men with Kaposi’s sarcoma, lymphoma and opportunistic infections and a median age of 36 years, reported 30% mortality within 1–1.5 years on AZT. In addition one or more new AIDS diseases, including pneumonia, candidiasis, fever, night sweats and diarrhea were observed in 172 (56%) within one year (Swanson *et al.*, 1990). Moreover, 50% needed at least one blood transfusion and 29% needed multiple blood transfusions to survive AZT treatment. Yet the authors concluded that the “risk:benefit ratio (is) advantageous to AIDS patients” (Swanson *et al.*, 1990).

(6) A comparison of the effects of indefinite AZT treatment on 170 HIV-positive AIDS-free persons with “early” AIDS to 168 with “late” AIDS indicated that the mortality was the same in both groups, i.e. 12–14% per 1–1.5 years (Hamilton *et al.*, 1992). The median age of the AZT recipients was 40 years; 63% were male homosexuals and 25% were intravenous drug users. AZT-specific diseases were observed in most “early AIDS” cases, i.e. leukopenia in 82%, severe leukopenia in 14%, anemia in 20%, severe anemia requiring transfusions in 5%, nausea in 40% and skin rashes in 47%. This indicates directly that AZT is toxic for AIDS-free HIV carriers, and that AZT toxicity is sufficiently dominant over other AIDS causes that it accelerates the progression to death of AIDS-free HIV carriers to the same rate that is observed in late AIDS patients (Duesberg, 1992d). The authors concluded that AZT, contrary to the Wellcome-sponsored study from 1987 conducted for licensing AZT, does not extend life.

(7) The annual lymphoma incidence of AZT-treated AIDS patients, with Kaposi’s sarcoma, pneumonia and wasting disease, was reported to be 9% by the National Cancer Institute and was calculated to be 50% over three years (Pluda *et al.*, 1990). The estimate of the 3-year incidence of lymphoma from this study was recently revised down to 31% (Yarchoan *et al.*, 1991). An independent study observed in a group of 346 AIDS patients in London, most of whom were on AZT, “during the past three years a progressive increase in the number of patients dying from lymphoma, . . .” to a current total of 16% in 1991 (Peters

et al., 1991). And a CDC study reported a 15% lymphoma incidence during 24 months on AZT (Centers for Disease Control, 1991).

The lymphoma incidence of untreated, HIV-positive AIDS risk groups is 0.3% per year, derived from the putative average progression rate of 10 years from HIV to AIDS (Moss *et al.*, 1988; Lemp *et al.*, 1990; Duesberg, 1991a) and the 3% incidence of lymphoma in AIDS patients (Centers for Disease Control, 1992b). Therefore, the annual lymphoma risk of AZT recipients is about 30 times higher than that of untreated HIV-positive counterparts. It appears that the chronic levels of the mutagenic AZT, at 20–60 μM (500–1500 mg/person/day), were responsible for the lymphomas (Section 4.6.2).

An alternative interpretation suggests that AZT had prolonged life sufficiently to allow HIV to induce the lymphomas directly or via immunodeficiency (Pluda *et al.*, 1990; Centers for Disease Control, 1991). However, this interpretation is flawed for several reasons: (1) Cancers, including malignant lymphomas, are not consequences of a defective immune system (Section 3.5.8). (2) There is as yet only a model for how HIV, the presumed killer of T-lymphocytes, could also cause cancer (Section 3.5.14) (Gallo, 1990). (3) AZT-induced lymphomas lack HIV-specific markers (McDunn *et al.*, 1991). (4) Several studies indicate that AZT does not prolong life (see above) (Dournon *et al.*, 1988; van Leeuwen *et al.*, 1990; Hamilton *et al.*, 1992; Kolata, 1992).

(8) Ten out of 11 HIV antibody-positive, AZT-treated AIDS patients recovered cellular immunity after discontinuing AZT in favor of an experimental HIV vaccine (Scolaro *et al.*, 1991). The vaccine consisted of an HIV strain that was presumed to be harmless, because it had been isolated from a healthy carrier who had been infected by the virus for at least 10 years. Since there was no evidence that the hypothetical vaccine strain differed from that by which the patients were already naturally vaccinated, the only relevant difference between the patients before and during the vaccine trial was the termination of their AZT treatment. It follows that AZT treatment is at least a necessary, if not a sufficient, cause of immunodeficiency in HIV-positives.

(9) Four out of 5 AZT-treated AIDS patients recovered from myopathy two weeks after discontinuing AZT; two redeveloped myopathy on

renewed AZT treatment (Till and MacDonnell, 1990), indicating that AZT is at least necessary for myopathy in HIV-positives.

(10) Four patients with pneumonia developed severe pancytopenia and bone marrow aplasia 12 weeks after the initiation of AZT therapy. Three out of 4 recovered within 4–5 weeks after AZT was discontinued (Gill *et al.*, 1987), indicating that AZT is necessary for pancytopenia in HIV-positives.

4.5. Drug Use Sufficient for AIDS Indicator Diseases in the Absence of HIV

Studies demonstrating AIDS-defining diseases in drug users in the absence of HIV are chronologically and geographically censored by the virus-AIDS hypothesis. Before the general acceptance of this hypothesis in the U.S., there were numerous American studies blaming AIDS on recreational drugs, but afterwards there was but one American report describing HIV-free Kaposi's sarcomas in homosexuals who had used such drugs, and only a few American and some European studies describing AIDS-defining diseases in HIV-free intravenous drug users (see below).

If HIV were necessary for AIDS among drug users, only HIV-positive drug users should develop AIDS. However, there is not even one controlled study showing that among matched drug users only HIV-positives get AIDS. On the contrary, such studies all indicate that drugs are sufficient to cause AIDS.

4.5.1. Drugs Used for Sexual Activities Sufficient for AIDS Diseases

(1) The first five AIDS cases, diagnosed in 1981 before HIV was known, were male homosexuals who had all consumed nitrite inhalants and presented with *Pneumocystis* pneumonia and cytomegalovirus infection (Gottlieb *et al.*, 1981).

(2) In 1985 and again in 1988 Haverkos analyzed the AIDS risks of 87 male homosexual AIDS patients with Kaposi's sarcoma (47), Kaposi's sarcoma plus pneumonia (20) and pneumonia only (20) (Haverkos *et al.*, 1985; Haverkos, 1988b). All men had used several sexual stimulants, 98% had used nitrites. Those with Kaposi's sarcomas reported double

the amount of sexual partners and 4.4-times more receptive anal intercourse than those with only pneumonia. The median number of sexual partners in the year prior to the illness was 120 for those with Kaposi's and 22 for those with pneumonia only. The Kaposi's cases reported 6-times more amylnitrite and ethylchloride use, 4-times more barbiturate use, and twice the methaqualone, lysergic acid and cocaine use than those with pneumonia only. Since no statistically significant differences were found for sexually transmitted diseases among the patients, the authors concluded that the drugs had caused Kaposi's sarcoma.

Although the data for Haverkos' analysis had been collected before HIV was declared the cause of AIDS, Haverkos' conclusion is valid. This is because (1) all patients had AIDS but only the heavy drug users had Kaposi's sarcoma in addition to immunodeficiency and because (2) not all can be assumed to be infected by HIV because transmission depends on an average of 1000 contacts (Section 3.5.2). Indeed, HIV was found in only 24% (Deininger *et al.*, 1990), 31% (van Griensven *et al.*, 1990), 43% (Graham *et al.*, 1991; Seage *et al.*, 1992), 48% (Winkelsstein *et al.*, 1987), 49% (Lemp *et al.*, 1990), 56% (Marion *et al.*, 1989) and 67% (Darrow *et al.*, 1987) of cohorts of homosexuals at risk for AIDS in Berlin, Amsterdam, Chicago-Washington DC-Los Angeles-Pittsburgh, Boston, San Francisco and Canada that were similar to those described by Haverkos.

(3) A 4.5 year tracking study of 42 homosexual men with lymphadenopathy but not AIDS reported that 8 had developed AIDS within 2.5 years (Mathur-Wagh *et al.*, 1984) and 12 within 4.5 years of observation (Mathur-Wagh *et al.*, 1985). All of these men had used nitrite inhalants and other recreational drugs including amphetamines and cocaine, but they were not tested for HIV. The authors concluded that "a history of heavy or moderate use of nitrite inhalant before study entry was predictive of ultimate progression to AIDS" (Mathur-Wagh *et al.*, 1984).

(4) Before HIV was known, three controlled studies compared 20 homosexual AIDS patients to 40 AIDS-free controls (Marmor *et al.*, 1982), 50 patients to 120 controls (Jaffe *et al.*, 1983b) and 31 patients to 29 controls (Newell *et al.*, 1985a) to determine AIDS risk factors. Each study reported that multiple "street drugs" were used as sexual stimu-

lants. And each study concluded that the “lifetime use of nitrites” (Jaffe *et al.*, 1983b) were 94% to 100%-consistent risk factors for AIDS (Newell *et al.*, 1985a).

(5) Early CDC data indicate that 86% of male homosexuals with AIDS had used oral drugs at least once a week and 97% occasionally (Centers for Disease Control, 1982; Haverkos, 1988b). The National Institute on Drug Abuse reports correlations from 69% (Lange *et al.*, 1988) to virtually 100% (Haverkos, 1988a; Newell *et al.*, 1988) between nitrite inhalants and other drugs and subsequent Kaposi’s sarcoma and pneumonia.

(6) A 27- to 58-fold higher consumption of nitrites by male homosexuals compared to heterosexuals and lesbians (Lesbian and Gay Substance Abuse Planning Group, 1991 a,b) correlates with a 20-fold higher incidence of Kaposi’s sarcoma (Selik *et al.*, 1987; Beral *et al.*, 1990) and a higher incidence of all other AIDS diseases in male homosexuals compared to most other risk groups (Tables 1 and 2).

(7) During the last 6–8 years the use of nitrite inhalants among male homosexuals decreased, e.g. from 58% in 1984 to 27% in 1991 in San Francisco (Lesbian and Gay Substance Abuse Planning Group, 1991b). In parallel, the incidence of Kaposi’s sarcoma among American AIDS patients decreased from a high of 50% in 1981 (Haverkos, 1988b), to 37% in 1983 (Jaffe *et al.*, 1983a), to a low of 10% in 1991 (Centers for Disease Control, 1992b). It follows that the incidence of Kaposi’s sarcoma is proportional to the number of nitrite users.

(8) After the discovery of HIV, 5 out of 6 HIV-free male homosexuals from New York with Kaposi’s sarcoma have reported the use of nitrite inhalants (Friedman-Kien *et al.*, 1990). Some of these men had no immunodeficiency. Soon after another six cases of HIV-free Kaposi’s sarcoma were reported in a “high risk population” from New York (Safai *et al.*, 1991). This indicates directly that HIV is not necessary and suggests that drugs are sufficient for AIDS.

(9) A 44-year-old, HIV-free homosexual man from Germany developed Kaposi’s sarcoma and had a T4 to T8-cell ratio of only 1.2. The man “had used nitrite inhalants for about 10 years,” but had no apparent immunodeficiency (Marquart *et al.*, 1991). Likewise, Kaposi’s sarcoma was diagnosed in a 40-year-old, promiscuous HIV-free homosexual

from England who admitted “frequent use of amyl nitrite.” The patient was otherwise symptom-free with a normal T4/T8 cell ratio (Archer *et al.*, 1989). In 1981 an English male homosexual with a “history of amyl-nitrite inhalation,” hepatitis B, gonorrhoea and syphilis was also diagnosed with Kaposi’s sarcoma. In 1984 he was found to be free of HIV, but in 1986 he became antibody-positive (Lowdell and Glaser, 1989).

(10) A prospective study from Canada identified immunodeficiency in 33 out of 166 HIV-free homosexual men (Marion *et al.*, 1989). The study did not mention drug consumption, but a later report on homosexual men with AIDS from the same cohort documented that all had been using either more or less than 20 “hits” of nitrites per month (Section 4.4) (Archibald *et al.*, 1992). Thus nitrites and possibly other drugs were sufficient for immunodeficiency.

Likewise, Lang, *et al.* (1989) described a steady decline of T4-cells in 37 homosexual men in San Francisco from 1200 per μL prior to HIV infection to 600 or less at the time of infection. Although recreational drug use and AZT were not mentioned, other studies of the same cohort of homosexual men from San Francisco described extensive use of recreational drugs (Section 4.3.2) (Darrow *et al.*, 1987; Moss, 1987) and AZT (Lang *et al.*, 1991).

4.5.2. Long-term Intravenous Drug Use Sufficient for AIDS-defining Diseases

(1) Among intravenous drug users in New York representing a “spectrum of HIV-related diseases,” HIV was observed in only 22 out of 50 pneumonia deaths, 7 out of 22 endocarditis deaths, and 11 out of 16 tuberculosis deaths (Stoneburner *et al.*, 1988).

(2) Pneumonia was diagnosed in 6 out of 289 HIV-free and in 14 out of 144 HIV-positive intravenous drug users in New York (Selwyn *et al.*, 1988).

(3) Among 54 prisoners with tuberculosis in New York state, 47 were street-drug users, but only 24 were infected with HIV (Braun *et al.*, 1989).

(4) In a group of 21 long-term heroin addicts, the ratio of helper to suppressor T-cells declined during 13 years from a normal of 2 to less

than 1, which is typical of AIDS (Centers for Disease Control, 1987; Institute of Medicine, 1988), but only 2 of the 21 were infected by HIV (Donahoe *et al.*, 1987).

(5) Thrombocytopenia and immunodeficiency were diagnosed in 15 intravenous drug users on average 10 years after they became addicted, but 2 were not infected with HIV (Savona *et al.*, 1985).

(6) The annual mortality of 108 HIV-free Swedish heroin addicts was similar to that of 39 HIV-positive addicts, i.e. 3–5%, over several years (Annell *et al.*, 1991).

(7) A survey of over a thousand intravenous drug addicts from Germany reported that the percentage of HIV-positives among drug deaths (10%) was exactly the same as that of HIV-positives among living intravenous drug users (Puschel and Mohsenian, 1991). Another study from Berlin also reported that the percentage of HIV-positives among intravenous drug deaths was essentially the same as that among living intravenous drug users, i.e. 20–30% (Bschor *et al.*, 1991). This indicates that drugs are sufficient for and that HIV does not contribute to AIDS-defining diseases and deaths of drug addicts.

(8) In 1989, the annual mortality of 197 HIV-positive, parenteral drug users from Amsterdam with an average age of 29 years was 4% and that of 193 age-matched HIV-negatives was 3% (Mientjes *et al.*, 1992). The annual incidence of pneumonia was 29% in the HIV-positives and 9% in the negatives. Clearly, a 3-fold higher morbidity is intrinsically inconsistent with a near identical mortality. However, the slightly higher mortality of HIV-positives is compatible with the fact that the positives had injected more drugs for a longer time, e.g. 84% of the positives vs 64% of the negatives had injected over the last 5 years, 85% vs 72% over the last 6 months and 59% vs 50% had injected heroin and cocaine.

(9) Lymphocyte reactivity and abundance were depressed by the absolute number of injections of drugs not only in 111 HIV-positive, but also in 210 HIV-free drug users from Holland (Mientjes *et al.*, 1991).

(10) The same lymphadenopathy, weight loss, fever, night sweats, diarrhea and mouth infections were observed in 49 out of 82 HIV-free, and in 89 out of 136 HIV-positive, long-term intravenous drug users in New York (Des Jarlais *et al.*, 1988).

(11) Among intravenous drug users in France, lymphadenopathy was observed in 41 and an over 10% weight loss in 15 out of 69 HIV-positives, and in 12 and 8, respectively, out of 44 HIV-negatives (Espinoza *et al.*, 1987). The French group had used drugs for an average of 5 years, but the HIV-positives had injected drugs about 50% longer than the negatives.

(12) In a group of 510 HIV-positive intravenous drug users in Baltimore, 29% reported one and 19% reported two or more AIDS-defining diseases. In a control group of 160 HIV-negative intravenous drug users matched with the HIV-positives for "current drug use," again 29% reported one and 13% reported two or more AIDS-defining diseases (Munoz *et al.*, 1992).

Nevertheless, the average T-cell count of HIV-negatives was about 2-times higher than that of HIV-positives (Munoz *et al.*, 1992). As in the above French study (Espinoza *et al.*, 1987), this appears to reflect a higher lifetime dose of drugs, because HIV is a marker for the duration and extent of drug consumption (Sections 3.4.3, 4.4 and 5).

(13) Among 97 intravenous drug users in New York with active tuberculosis, 88 were HIV-positive and 9 were HIV-negative; and among 6 "crack" (cocaine) smokers with tuberculosis, 3 were HIV-negative and 3 were positive (Brudney and Dobkin, 1991).

(14) The mental development and psychomotor indices of 8 HIV-infected and 6 uninfected infants were observed from 6–21 months of age. The mothers of each group were HIV-positive and had used intravenous drugs and alcohol during pregnancy (Koch, 1990; Koch *et al.*, 1990; T. Koch, R. Jeremy, E. Lewis, P. Weintrub, C. Rumsey and M. Cowan, unpublished data). The median indices of both groups were significantly below average, e.g. 80/100 mental development and 85/100 psychomotor units. The uninfected infants remained on average about 5/100 units higher. A control group of 5 infants, born to HIV-negative mothers who had also used intravenous drugs and alcohol during pregnancy, also had subnormal indices averaging about 95/100 for both criteria.

The degree of neurological retardation of the infants correlated directly with maternal drug consumption: 80% of the mothers of infected infants were "heavy" and 10% occasional parenteral cocaine users and

33% were “heavy” and 33% occasional alcohol users during pregnancy; 45% of the mothers of uninfected infants were “heavy” and 30% occasional parenteral cocaine users and 35% were “heavy” and 30% occasional alcohol users; and 21% of the HIV-free mothers were “heavy” and 58% occasional parenteral cocaine users and 12% were “heavy” and 44% occasional alcohol users. In addition 66% of the HIV-positive and 63% of the negative mothers reported the use of opiates during pregnancy (T. Koch, R. Jeremy, E. Lewis, P. Weintrub, C. Rumsey and M. Cowan, unpublished data).

(15) The psychomotor indices of infants “exposed to substance abuse in utero” were “significantly” lower than those of controls, “independent of HIV status.” Their mothers were all drug users but differed with regard to drug use during pregnancy. The mean indices of 70 children exposed during pregnancy were 99 and those of 25 controls were 109. Thus maternal drug use during pregnancy impairs children independent of HIV (Aylward *et al.*, 1992).

The same study also reports a “significant difference” based on the HIV status of these children. The mean scores of 12 HIV-positives was 88 and that of 75 negatives was 102. But the study did not break down the scores of the HIV-positive infants based on “exposure to substance abuse in utero.” Indeed, the scores of 4 of the 12 HIV-infected infants were “above average,” i.e. 100–114, and 4 of the 12 mothers did not inject drugs during pregnancy.

(16) Ten HIV-free infants born to intravenous drug-addicted mothers had the following AIDS-defining diseases, “failure to thrive, persistent generalized lymphadenopathy, persistent oral candidiasis, and developmental delay . . .” (Rogers *et al.*, 1989).

(17) One HIV-positive and 18 HIV-free infants born to intravenous drug-addicted mothers had only half as many leukocytes at birth than normal controls. At 12 months after birth, the capacity of their lymphocytes to proliferate was 50–70% lower than that of lymphocytes from normal controls (Culver *et al.*, 1987).

(18) Two studies to test the role of HIV on neurological function confirm the drug-AIDS hypothesis indirectly and directly. The first of these, which excluded users of psychoactive drugs, found that neuropsychometric functions of 50 HIV-negative homosexuals were the

same as those of 33 HIV-positives (Clifford *et al.*, 1990). Another study of intravenous drug users on methadone found that neither the drug-impaired neuropsychological functions of 137 HIV-negatives nor those of 83 HIV-positives were deteriorating over 7.4 months (McKegney *et al.*, 1990). However, the study notes that the functions of HIV-positives were lower than those of HIV-negatives because "a greater number of injections per month, more frequent use of cocaine . . . were strongly associated with HIV seropositivity."

Thus, a critical lifetime dosage of drugs appears necessary in HIV-positives and sufficient in HIV-negatives to induce AIDS-indicator and other diseases.

4.6. Toxic Effects of Drugs Used By AIDS Patients

4.6.1. Toxicity of Recreational Drugs

From as early as 1909 (Achard *et al.*, 1909) evidence has accumulated that long-term consumption of psychoactive drugs leads to immune suppression and clinical abnormalities similar to AIDS, including lymphopenia, lymphadenopathy, fever, weight loss, septicemia, increased susceptibility to infections and profound neurological disorders (Terry and Pellens, 1928; Briggs *et al.*, 1967; Dismukes *et al.*, 1968; Sapira, 1968; Harris and Garret, 1972; Geller and Stimmel, 1973; Brown *et al.*, 1974; Louria, 1974; McDonough *et al.*, 1980; Cox *et al.*, 1983; Kozel and Adams, 1986; Selwyn *et al.*, 1989; Turner *et al.*, 1989; Kreek, 1991; Pillai *et al.*, 1991; Bryant *et al.*, 1992). Since the early 1980s, when T-cell ratios became measureable, low T4 to T8-cell ratios averaging 1 or less were reported in addicts who had injected drugs for an average of 10 years (Layon *et al.*, 1984).

Intravenous drugs can be toxic directly and indirectly. Indirect toxicity can be due to malnutrition, because of the enormous expense of illicit drugs, or to septicemia because most illicit drugs are not sterile (Cox *et al.*, 1983; Stoneburner *et al.*, 1988; Lerner, 1989; Buehler *et al.*, 1990; Pillai *et al.*, 1991; Luca-Moretti, 1992). Typically, intravenous drug users develop pneumonia, tuberculosis, endocarditis and wasting disease (Layon *et al.*, 1984; Stoneburner *et al.*, 1988; Braun *et al.*, 1989; Brudney and Dobkin, 1991). Oral consumption of cocaine and other psychoactive drugs has been reported to cause pneumonitis, bronchi-

tis, edema (Ettinger and Albin, 1989) and tuberculosis (Brudney and Dobkin, 1991). Physiological and neurological deficiencies, including mental retardation, are observed in children born to mothers addicted to cocaine and other narcotic drugs (Fricker and Segal, 1978; Lifschitz *et al.*, 1983; Alroomi *et al.*, 1988; Blanche *et al.*, 1989; Root-Bernstein, 1990a; Toufexis, 1991; Finnegan *et al.*, 1992; Luca-Moretti, 1992). According to the National Institute on Drug Abuse, "Cocaine is currently the drug of greatest national concern, from a public health point of view . . ." (Schuster, 1984).

Because inhalation of alkyl nitrites relaxes smooth muscles, it has been prescribed since 1867 against angina pectoris and heart pain at doses of 0.2 mL (Cox *et al.*, 1983; Newell *et al.*, 1985b; Shorter, 1987; Seage *et al.*, 1992). No AIDS defining diseases have been reported at these doses in patients with those relatively severe, terminal cardiovascular diseases (Cox *et al.*, 1983; Shorter, 1987), possibly because they did not live long enough to develop them. However, immediate and late toxicities have been observed in recreational users who have inhaled millilitres of nitrite inhalants (Newell *et al.*, 1985b; Schwartz, 1988). Alkyl nitrites are directly toxic as they are rapidly hydrolyzed *in vivo* to yield nitrite ions, which react with all biological macromolecules (Osterloh and Olson, 1986; Maikel, 1988). Addicts with 0.5 mM nitrite derivatives and 70% methemoglobin in blood have been recorded (Osterloh and Olson, 1986). Toxicity for the immune system, the central nervous system, the hematologic system and pulmonary organs has been observed after short exposure to nitrites in humans and in animals (Newell *et al.*, 1985b, 1988; Wood, 1988). In 1982, Goedert *et al.* found that the helper to suppressor T-cell ratio was lower in homosexual men who had used volatile nitrite inhalants than among nonusers. Further, alkyl nitrites were shown to be both mutagenic and carcinogenic in animals (Jorgensen and Lawesson, 1982; Hersh *et al.*, 1983; Mirvish *et al.*, 1988; Newell *et al.*, 1985b, 1988).

By comparing the AIDS risk factors of 31 homosexual men with AIDS to 29 without, Newell *et al.* and others determined a direct "dose-response gradient": the higher the nitrite usage the greater the risk for AIDS (Marmor *et al.*, 1982; Newell *et al.*, 1985a; Haverkos and Dougherty, 1988) and deduced a 7–10 year lag time between chronic

consumption and Kaposi's sarcoma (Newell *et al.*, 1985b). Likewise, a French study of homosexual men with and without AIDS who had inhaled nitrites documents that "cases were significantly older (approximately 10 years) than controls" (Section 4.3.2) (Messiah *et al.*, 1988). Also, a German study observed Kaposi's sarcoma in an HIV-free man after he had inhaled nitrites for 10 years (Section 4.5.1) (Marquart *et al.*, 1991). These studies indicate that about 10 years of nitrite inhalation are necessary to convert "controls" to "cases."

In view of this several investigators have proposed that nitrite inhalants cause pulmonary and skin Kaposi's sarcoma and pneumonia by direct toxicity on the skin and oral mucosa (Centers for Disease Control, 1982; Marmor *et al.*, 1982; Haverkos *et al.*, 1985; Mathur-Wagh *et al.*, 1985; Newell *et al.*, 1985a; Lauritsen and Wilson, 1986; Haverkos, 1990). Because of their toxicity a prescription requirement was instated for the sale of nitrite inhalants by the Food and Drug Administration in 1969 (Newell *et al.*, 1985b) and because of an "AIDS link" (Cox, 1986) the sale of nitrites was banned by the U.S. Congress in 1988 (Public Law 100-690) (Haverkos, 1990) and by the "Crime Control Act of 1990" (January 23, 1990).

Although a necessary role of HIV in HIV-positive AIDS patients cannot be excluded, this role would be stoichiometrically insignificant compared to that of the drugs. This is because drug molecules exceed HIV molecules by over 13 orders of magnitude. Given about 10^{10} leukocytes per human, of which at most 1 in 10^4 are actively infected (Section 3.5.1), and that each actively infected cell makes about 100 viral RNAs per day, there are only 10^6 T-cells with 10^2 HIV RNAs in an HIV-positive person. By contrast, 1 mL (or 0.01 mol) of amyl nitrite with a molecular weight of 120 contains 6×10^{21} molecules, or 6×10^7 nitrite molecules, for every one of the 10^{14} cells in the human body. Thus, based on molecular representation, HIV's role in AIDS, if it existed, would have to be catalytic in comparison with that of drugs.

Pillai, Nair and Watson conclude from a recent review on the role of recreational drugs in AIDS: "Circumstantial and direct evidence suggesting a possible role for drug . . . induced immunosuppression appears overwhelming. What is required now is better and more accurate detection of substance abuse, a direct elucidation of the immune and related

mechanisms involved, and appropriate techniques to analyze it" (Pillai *et al.*, 1991).

4.6.2. Toxicity of AZT

Since 1987 AZT has been used as an anti-HIV agent (Section 4.3.3) based on two placebo-controlled studies reporting therapeutic and prophylactic benefits (Section 4.4.2). However, AZT was originally developed in the 1960s for cancer chemotherapy to kill human cells via termination of DNA synthesis (Cohen, 1987; Yarchoan and Broder, 1987a; Yarchoan *et al.*, 1991). The primary AZT metabolites are 3'-termini of DNA which are cell-killing, 3'-amino-dT which is more toxic than AZT, and 5'-O-glucuronide which is excreted (Cretton *et al.*, 1991). As a chain terminator of DNA synthesis, AZT is toxic to all cells engaged in DNA synthesis. AZT toxicity varies a great deal with the subject treated due to differences in its uptake and in its cellular metabolism (Chernov, 1986; Elwell *et al.*, 1987; Yarchoan and Broder, 1987b; Smothers, 1991; Yarchoan *et al.*, 1991).

AZT is prescribed as AIDS prophylaxis or therapy at 500–1500 mg per day, corresponding to a concentration of 20–60 $\mu\text{mol/L}$ in the patient. Prior to the licensing of AZT, Burroughs Wellcome, the manufacturer of the drug, and the NIH have jointly claimed selective inhibition of HIV by AZT *in vitro* because human lymphoblasts and fibroblasts appeared over 1000-fold more resistant to AZT (inhibited only at 1–3 mM) than was replication of HIV (inhibited at 50–500 nM) (Furman *et al.*, 1986). On this basis they calculated an *in vitro* antiviral therapeutic index of 10^4 . This "selective" sensitivity of HIV to AZT was explained in terms of a "selective interaction of AZT with HIV reverse transcriptase" (Furman *et al.*, 1986). Accordingly the manufacturer informs AZT recipients: "The cytotoxicity of zidovudine [AZT] for various cell lines was determined using a cell growth assay . . . ID₅₀ values for several human cell lines showed little growth inhibition by zidovudine except at concentrations $> 50 \mu\text{g/mL}$ ($\geq 200 \mu\text{M}$) or less." (Medical Economics Data, 1992). Further, it informs them that enterobacteria including *E. coli* are inhibited "by low concentrations of zidovudine [AZT]," between 0.02 and 2 μM AZT, just like HIV (Medical Economics Data, 1992).

However, an independent study showed in 1989 that AZT is about

1000-times (!) more toxic for human T-cells in culture, i.e. at about 1 μM than the study conducted by its manufacturer and the NIH (Avramis *et al.*, 1989). Other studies have also found that AZT inhibits T-cells and other hemopoietic cells *in vitro* at 1–8 μM (Balzarini *et al.*, 1989; Mansuri *et al.*, 1990; Hitchcock, 1991). Since normal deoxynucleotide triphosphates are present in the cell at micromolar concentrations, toxicity of AZT should be expected in the micromolar range. Indeed, when AZT is added at a micromolar concentration to the culture medium, it and its phosphorylated derivatives quickly reach an equivalent or higher concentration in the cell, and thus effectively compete with their natural thymidine counterparts (Avramis *et al.*, 1989; Balzarini *et al.*, 1989; Ho and Hitchcock, 1989; Hitchcock, 1991).

Thus the low cellular toxicity reported by the manufacturer and the NIH for human cells appears erroneous—possibly because “the clinical development of AZT was exceedingly rapid; it was approved for clinical use in the U.S. about 2 years after the first *in vitro* observation of its activity against HIV” (Yarchoan *et al.*, 1991). It follows that AZT does not selectively inhibit viral DNA synthesis and is prescribed at concentrations that exceed 20- to 60-fold the lethal dose for human cells in culture.

In view of its inevitable toxicity, the rationale of using AZT as an anti-HIV drug must be reconsidered and its potential antiviral effect must be weighed against its toxicity.

4.6.2.1. *AZT not a rational anti-HIV drug.* A rational antiviral therapy depends on proof that the targeted virus is the cause of the disease to be treated and that toxicity for the virus outweighs that for the host cell. Such proof cannot be supplied for AZT for the following reasons:

- (1) There is no proof that HIV causes AIDS (Section 3.3).
- (2) Even if the hypothesis that HIV causes AIDS by killing T-cells were correct, it would be irrational to kill the same infected cells twice, once presumably with HIV and once more with AZT.
- (3) Since many healthy persons with antibodies against HIV have equal or even higher percentages of infected T-cells than AIDS patients (Section 3.3), there is no reverse transcription of HIV during progres-

sion to AIDS that could be targeted with AZT. Even if some reverse transcription occurred in antibody-positive persons, AZT could not differentially inhibit viral DNA, because HIV DNA comprises only 9 kb but cell DNA comprises 10^6 kb. Thus cell DNA is a 100,000-fold bigger target for AZT than HIV. And even if AZT showed a 100-fold preference for reverse transcriptase of HIV over cellular DNA polymerase, as has been claimed by the study conducted by Burroughs Wellcome and the NIH (Furman *et al.*, 1986), cell DNA would still be a 1000-fold bigger target for AZT than viral DNA. It follows that cell DNA is the only realistic target of AZT in antibody-positive persons.

(4) Since AZT cannot distinguish infected from uninfected leukocytes and on average less than 1 in 1000 is infected (Section 3.3), AZT must kill at least 1000 leukocytes in AIDS patients and in asymptomatic HIV-carriers to kill just 1 infected cell—a very high toxicity index, even if HIV were the cause of AIDS.

It follows that there is no rational basis for AZT therapy or prophylaxis for AIDS (Duesberg, 1992d).

4.6.2.2. Toxicity of AZT in AIDS Patients and AIDS-free Persons. The following AZT-specific diseases have been recorded in AIDS patients, in AIDS-free persons and animals treated with AZT, based on studies listed here (Section 4.4.2) and reviewed elsewhere (Smothers, 1991; Medical Economics Data, 1992):

(1) anemia, neutropenia and leukopenia in 20–80%, with about 30–57% requiring transfusions within several weeks (Gill *et al.*, 1987; Kolata, 1987; Richman *et al.*, 1987; Dournon *et al.*, 1988; Walker *et al.*, 1988; Swanson *et al.*, 1990; van Leeuwen *et al.*, 1990; Smothers, 1991; Hamilton *et al.*, 1992),

(2) severe nausea from intestinal intoxication in up to 45% (Richman *et al.*, 1987; Volberding *et al.*, 1990; Smothers, 1991),

(3) muscle atrophy and polymyositis, due to inhibition of mitochondrial DNA synthesis in 6–8% (Richman *et al.*, 1987; Bessen *et al.*, 1988; Gorard and Guilodd, 1988; Helbert *et al.*, 1988; Dalakas *et al.*, 1990; Till and MacDonnell, 1990; Yarchoan *et al.*, 1991; Hitchcock, 1991),

- (4) lymphomas in about 9% within 1 year on AZT (Section 4.4.2),
- (5) acute (nonviral) hepatitis (Dubin and Braffman, 1989; Smothers, 1991),
- (6) nail dyschromia (Don *et al.*, 1990; Smothers, 1991),
- (7) neurological diseases including insomnia, headaches, dementia, mania, Wernicke's encephalopathy, ataxia and seizures (Smothers, 1991), probably due to inhibition of mitochondrial DNA (Hitchcock, 1991),
- (8) 12 out of 12 men reported impotence after 1 year on AZT (Callen, 1990),
- (9) in addition AZT is carcinogenic in mice, causing vaginal squamous carcinomas (Cohen, 1987; Yarchoan and Broder, 1987a), and it transforms mouse cells *in vitro* as effectively as methylcholanthrene (Chernov, 1986).

Overall AZT is not a rational prophylaxis or a therapy for AIDS and is capable of causing potentially fatal diseases, such as anemia, leukopenia and muscle atrophy. Yet, despite its predictable toxicity, AZT is thought to have serendipitous therapeutic and prophylactic benefits according to those investigators who have studied its effects together with the manufacturer for licensing of the drug (Section 4.4.2) (Fischl *et al.*, 1987; Richman *et al.*, 1987; Volberding *et al.*, 1990). Confronted with the difficulties in rationalizing anti-HIV prophylaxis and therapy with AZT, the Wellcome researcher Freestone cites the Burroughs Wellcome study analyzed above (Section 4.4.2, item 1): "the primary end-point for the study was death (1 in 145 zidovudine recipients, 19 in 137 placebo recipients . . .)—an end-point little subject to observer error or bias" (Freestone, 1992).

The popularity of AZT as an anti-HIV drug can only be explained by the widespread acceptance of the virus-AIDS hypothesis, the failure to consider the enormous difference between the viral and cellular DNA targets and a general disregard for the long-term toxicity of drugs (Section 6). In the words of the retrovirologist Temin "but the drug generally becomes less effective after six months to a year . . ." (Nelson *et al.*, 1991)—a euphemism for its fatal toxicity by that time. This is a probable reason that AZT was licensed without long-term studies in

animals compatible with human applications and that the need for such studies is neither mentioned nor called for in reviews of its toxic effects in humans (Chernov, 1986; Yarchoan and Broder, 1987b; Smothers, 1991; Yarchoan *et al.*, 1991), although AZT must be the most toxic drug ever approved for indefinite therapy in America. Even the manufacturer acknowledges that "... the drug has been studied for limited periods of time and long term safety and efficacy are not known" (Shenton, 1992) and recommends that "patients should be informed... that the long-term effects of zidovudine are unknown at this time" (Medical Economics Data, 1992). And after prescribing it for five years, even AIDS "experts" have recently expressed doubts about the "survival benefit" of AZT (Kolata, 1992).

4.7. Drug-AIDS Hypothesis Correctly Predicts the Epidemiology and Heterogeneous Pathology of AIDS

(1) The long-term consumption of drugs, but not the hosting of a latent virus, predicts drug-specific pathogenicity after "long latent periods." These long latent periods of HIV are in reality the lag periods that recreational drugs (Schuster, 1984; Newell *et al.*, 1985b) and frequent transfusions of foreign proteins take to cause AIDS-defining diseases (Section 3.4.4.5). Drugs are molecularly abundant (Section 4.6.1) and biochemically active as long as they are administered and thus cumulatively toxic over time. It is for this reason that it typically takes 5–10 years for recreational drugs, and months for AZT, to cause AIDS-defining and other diseases (Sections 3.1 and 5). But HIV, after a brief period of immunogenicity (Clark *et al.*, 1991; Daar *et al.*, 1991), is chronically dormant and thus molecularly and biochemically irrelevant for the rest of the host's life.

(2) Drugs and other noninfectious agents also exactly predict the epidemiology of AIDS. About 32% of American AIDS patients are confirmed intravenous drug users, 60% appear to use recreational drugs orally, and an unknown but large percentage of people in both behavioral and clinical AIDS risk groups use AZT. Moreover, the consumption of recreational drugs by AIDS patients is probably under-reported because the drugs are illicit, and because medical scientists and support for research are currently heavily biased in favor of viral-AIDS

(Section 6) (Ettinger and Albin, 1989; Lerner, 1989; Duesberg, 1991b). In sum, more than 90% of American AIDS is correlated with drugs. The remainder would reflect the natural background of AIDS-defining diseases in the U.S. (Duesberg, 1992). Indeed, only drug users do not benefit from the ever improving health parameters and increasing life spans of the Western World (Hoffman, 1992; The Software Toolworks World Atlas™, 1992). The widespread use of AZT in hemophiliacs (Section 4.3.3) unfortunately predicts a new increase in their mortality.

The dramatic increase in America in the consumption of all sorts of recreational drugs since the Vietnam War also explains the simultaneous increase of AIDS in intravenous drug users and male homosexuals (Centers for Disease Control, 1992b). AIDS of both risk groups followed closely the above listed drug-use statistics during the last 15 years, with increases in 1987 that corresponded to the expanded AIDS definition (Centers for Disease Control, 1987) and the introduction of AZT treatment. By contrast a sexually transmitted AIDS would have spread much faster among homosexuals than among intravenous drug users (Weyer and Eggers, 1990; Eggers and Weyer, 1991). The apparent exponential spread of AIDS during the period from 1984 to 1987 (Heyward and Curran, 1988; Mann *et al.*, 1988; Weyer and Eggers, 1990) probably reflected an exponential spread of “AIDS testing,” which resulted in an exponential spread of AIDS diagnoses for drug diseases (Section 4.2). AIDS testing had increased from 0 in 1984 to 20 million tests per year in 1986 in the U.S. alone (Section 3.6).

(3) The drug hypothesis further predicts that the 50–70% of American and 50–80% of European intravenous drug users who are HIV-free (Stoneburner *et al.*, 1988; Turner *et al.*, 1989; Brenner *et al.*, 1990; U.S. Department of Health and Human Services, 1990; National Commission on AIDS, 1991), and the HIV-free male homosexuals who use sexual stimulants will develop the same diseases as their HIV-positive counterparts—except that their diseases will be diagnosed by their old names. This has been amply confirmed for intravenous drug users (Section 4.5). But since AIDS research became dominated by the virus-hypothesis, only a few studies have published HIV-free homosexual immunodeficiencies and “AIDS cases” (Section 4.5, Note added in proof). Yet more such cases must exist because the CDC allows “pre-

sumptive diagnosis” of HIV disease and only about 50% of all American AIDS cases are confirmed positives (Sections 2.2 and 3.4.1) and because only about 50% of homosexuals from many different cohorts at risk for AIDS are confirmed HIV-positive (Section 4.5.1).

(4) The drug hypothesis also correctly predicts drug-specific AIDS diseases in distinct risk groups due to distinct drugs (Sections 2.1.3, 3.4.5 and 5, Table 2).

4.8. Consequences of the Drug-AIDS Hypothesis: Risk-Specific Preventions and Therapies, but Resentment by the Virus-AIDS Establishment

The drug-AIDS hypothesis predicts that the AIDS diseases of the behavioral AIDS risk groups in the U.S. and Europe can be prevented by stopping the consumption of recreational and anti-HIV drugs, but not by “safe sex” (Institute of Medicine, 1988; Weiss and Jaffe, 1990; Maddox, 1991b) and “clean needles,” i.e. sterile injection equipment (National Commission on AIDS, 1991) for toxic and unsterile street drugs. Indeed AIDS has continued to increase in all countries that have promoted safe sex to prevent AIDS for over 5 years now (Centers for Disease Control, 1992b; World Health Organization, 1992a; Anderson and May, 1992). Further, the hypothesis raises the hopes for risk-specific therapies.

According to the drug-AIDS hypothesis, AZT is AIDS by prescription. Screening of blood for antibodies to HIV is superfluous, if not harmful, in view of the anxiety that a positive test generates among the many believers in the virus-AIDS hypothesis (Grimshaw, 1987) and the toxic AZT prophylaxis prescribed to many who test “positive.” Eliminating the test would also reduce the cost of the approximately 12 million annual blood donations in the U.S. (Williams *et al.*, 1990) and of examining annually 200,000 recruits and 2 million servicemen for the U.S. Army (Burke *et al.*, 1990) by \$12 to \$70 each (Irwin Memorial Blood Bank, San Francisco, personal communication).

Further, it would lift travel restrictions for antibody-positives to many countries including the U.S. and China, and would lift quarantine for HIV-positive Cubans, and would acquit all those antibody-positive Americans who are currently imprisoned for having had sex with antibody-negatives, and would grant to HIV “antibody-positives” the same

chances to be admitted to a health insurance program as to those who have only antibodies to other viruses.

Despite its many potential blessings, the drug hypothesis is currently highly unpopular—not because it would be difficult to verify, but because of its consequences for the virus-AIDS establishment (Section 6). The drug hypothesis is very testable epidemiologically and experimentally by studying the effects of the drugs consumed by AIDS patients in animals. Indeed most tests have already been done (Section 4). To disprove this hypothesis it would be necessary to document that an infectious agent exists which—in the absence of AZT (!) causes AIDS diseases above their normal background in the nondrug using population. The medical, ethical and legal consequences of the drug-AIDS hypothesis, should it prevail, have recently been summarized under the title “Duesberg: An enemy of the people?” (Ratner, 1992). Ratner points out that, “The loss of confidence of Americans in their scientists and perhaps, by extension, their physicians, could rival their current disillusionment with politicians” and wonders, “What would happen to the reservoir of good will painstakingly built up for the victims of AIDS?”

5. Drugs and Other Noncontagious Risk Factors Resolve All Paradoxes of the Virus-AIDS Hypothesis

A direct application of the hypothesis that drugs and other noncontagious risk factors cause AIDS proves that it can resolve all paradoxes of the virus-AIDS hypothesis:

(1) It is paradoxical to assume that AIDS is new because HIV is new. HIV is a long-established, perinatally transmitted retrovirus. It just appears new because, being a chronically latent virus, it only became detectable with recently developed technology (Section 3.5.1). Instead drugs are the only new health risks in this era of ever improving health parameters. Thus AIDS is new because the drug epidemic is new.

(2) According to the virus-AIDS hypothesis it is paradoxical that AIDS did not “explode” into the general population as predicted (Institute of Medicine, 1986; Shorter, 1987; Fineberg, 1988; Heyward and Curran, 1988; Blattner, 1991; Mann and the Global AIDS Policy Coali-

tion, 1992). AIDS has remained restricted for over 10 years to only 15,000 annual cases (0.015%) of the over 100 million sexually active heterosexual Americans, and to only 25,000 (0.3%) of the 8 million homosexuals (Centers for Disease Control, 1992b), although venereal diseases (Aral and Holmes, 1991), unwanted pregnancies and births (Hoffman, 1992; The Software Toolworks World Atlas™, 1992) are on the increase in America. (The homosexuals represent about 10% of the adult male population (Turner *et al.*, 1989; Lesbian and Gay Substance Abuse Planning Group, 1991a).) This is because psychoactive drugs and AZT, not HIV, are the causes of AIDS.

(3) The paradox of a virus causing risk group-specific and country-specific AIDS diseases is resolved by distinct nonviral AIDS causes including drugs and other noncontagious pathogens like long-term transfusions and malnutrition (Sections 2.1.3 and 3.4.5, Tables 1 and 2).

(4) The paradox of a male-specific AIDS virus (i.e. 90% of all American and 86% of all European AIDS cases are males), although no AIDS disease is male-specific, is resolved by male-specific behavior and by male genetic disorders. In America and Europe males consume over 75% of all “hard” injected psychoactive drugs (Section 4.3.1), homosexual males are almost exclusive users of oral aphrodisiacs like nitrites (Section 4.3.2) and nearly all hemophiliacs are males.

(5) The paradox of a 10-year-slow AIDS virus, i.e. AIDS occurs only after “latent (!) periods” of HIV that average 10 years in adults and 2 years in babies (Section 2.2), is resolved by the cumulative toxicity of long-term drug use. According to the CDC the “lifetime use” of drugs determines the AIDS risk (Jaffe *et al.*, 1983b). On average 5–10 years elapse in adult drug addicts between the first use of drugs and “acquiring” drug-induced AIDS diseases (Layon *et al.*, 1984; Schuster, 1984; Savona *et al.*, 1985; Donahoe *et al.*, 1987; Espinoza *et al.*, 1987; Weber *et al.*, 1990). The time lag from a nitrite habit to Kaposi’s sarcoma has been determined to be 7–10 years (Newell *et al.*, 1985b). Severe T-cell depletion and immunodeficiency is also “acquired” by hemophiliacs on average only after 14–15 years of treatment with blood concentrates (Section 3.4.4.5).

In babies of drug-addicted mothers AIDS appears much sooner than

in adults because of a much lower threshold of the fetus for drug-pathogenicity. This also resolves the secondary paradox of a discrepancy of 8 years between the “latent periods” of HIV in babies and in adults.

(6) It is paradoxical that American teenagers do not get AIDS, although over 70% are sexually active and about 50% are promiscuous (Turner *et al.*, 1989; Burke *et al.*, 1990; Congressional Panel, 1992) and 0.03% to 0.3% carry HIV (Section 3.5.2). The paradox that a sexually transmitted “AIDS virus” would spare American and European teenagers is resolved by the fact that only years of drug consumption, and years of transfusions for hemophilia (Section 3.4.4.5) will cause AIDS—by which time these teenagers are in their twenties.

(7) The apparent paradox that the same virus would at the same time cause two entirely different AIDS epidemics, one in Africa and the other in America and Europe is an artifact of the AIDS definition. Because of the HIV-based AIDS definition, a new drug epidemic in America and Europe and an epidemic of old Africa-specific diseases like fever, diarrhea and tuberculosis (Section 3.4.4.4) were both called AIDS when HIV became detectable. Since HIV is endemic in over 10% of Central Africans, over 10% of their AIDS-defining diseases are now called AIDS (Section 2.2).

6. Why Did AIDS Science Go Wrong?

6.1. The Legacy of the Successful Germ Theory: A Bias Against Noninfectious Pathogens

Unlike any other scientific hypothesis, the virus-AIDS hypothesis became national American dogma before it could be reviewed by the scientific community. It had been announced by the Secretary of Health and Human Services in 1984 before it had been published in the scientific literature. Unlike any other medical hypothesis it captured the world without ever bearing any fruits in terms of public health benefits. From the beginning the hypothesis has absorbed the critical potential of its many followers with the question, whether Montagnier from France or Gallo from the U.S. had won the race in isolating the “AIDS virus” and who owned the lucrative patent rights for the “AIDS test.” This

question was so consuming that the presidents of the two countries were called to sign a settlement, and a revisionist paper was published by the opponents describing their fierce controversy as an entente cordiale against the real enemy, the “deadly” AIDS virus (Gallo and Montagnier, 1987). During the 1980s press accounts consistently called HIV “the deadly virus” (Duesberg, 1989c).

Clearly, the enthusiastic acceptance of the virus-AIDS hypothesis was not based on its scientific rigor or its fruits. It was instead grounded on the universal admiration and respect for the germ theory. The germ theory of the late 19th century ended the era of infectious diseases, which now account for less than 1% of all mortality in the Western World (Cairns, 1978). It celebrated its last triumph in the 1950s with the elimination of the polio epidemic by antiviral vaccines.

But the germ theory continues to inspire both scientists and the public to believe that a “good” body can be protected against “evil” microbes. Accordingly, even the greatly feared and highly stigmatizing “AIDS test” for a presumably new, sexually transmitted “AIDS virus” was readily sold to all governments, medical associations and even to the AIDS risk-groups (Section 6.2), despite the absence of convincing evidence for transmissibility. In the words of one observer, “The rationale for such programs is often the historical precedent of syphilis screening,” which “never proved to be effective” and led to “toxic treatments with arsenical drugs, assuming the tests were correct . . .” and “deep stigma and disrupted relationships . . .” “Patients required a painful regimen of injections, sometimes for as long as two years” (Brandt, 1988). Even epidemiologists failed to recognize that AIDS and HIV were only spreading in newly-established behavioral and clinical risk groups and that HIV was a long-established virus in the general populations of many countries (Section 3.5.1). Instead of considering noninfectious causes, they simulated “coagents” (Eggers and Weyer, 1991) and “assortative scenarios” (Anderson and May, 1992) to hide the growing discrepancies between HIV and AIDS and intimidated skeptics with apocalyptic predictions of AIDS pandemics in the general populations of many countries that have raised fears and funds to unprecedented levels (Section 1) (Heyward and Curran, 1988; Mann *et al.*, 1988; Mann and the Global AIDS Policy Coalition, 1992; Anderson and May, 1992).

Even now, in an era free of infectious diseases but full of man-made chemicals, scientists and the public share an unthinking preference for infectious over noninfectious pathogens. Both groups share an obsolete microbophobia but tolerate the use or even indulge in the consumption of numerous recreational and medical drugs. Moreover, progressive scientists and policy makers are not interested in recreational and medical drugs and man-made environmental toxins as causes of diseases, because the mechanisms of pathogenesis are predictable. Further, prevention of drug diseases is scientifically trivial and commercially unattractive.

By contrast, microbial and particularly viral pathogens are scientifically and commercially attractive to scientists. Beginning with Peyton Rous, at least 10 Nobel prizes have been given to virologists in the last 25 years. And many virologists have become successful biotechnologists. For example, a blood test for a virus is good business if the test becomes mandatory for the 12 million annual blood donations in the U.S., e.g. the "AIDS test." The same is true for a vaccine or an antiviral drug that is approved by the Food and Drug Administration.

Thousands of lives have been sacrificed to this bias for infectious theories of disease, even before AIDS appeared. For example, the U.S. Public Health Service insisted for over 10 years in the 1920s that pellagra was infectious, rather than a vitamin B deficiency as had been proposed by Joseph Goldberger (Bailey, 1968). Tertiary syphilis is commonly blamed on treponemes, but is probably due to a combination of treponemes and long-term mercury and arsenic treatments used prior to penicillin, or merely to these treatments alone (Brandt, 1988; Fry, 1989). "Unconventional" viruses were blamed for neurological diseases like Kreutzfeld-Jacob's disease, Alzheimer's disease and kuru (Gajdusek, 1977). The now extinct kuru was probably a genetic disorder that affected just one tribe of natives from New Guinea (Duesberg and Schwartz, 1992). Although a Nobel Prize was given for this theory, the viruses never materialized and an unconventional protein, termed "prion," is now blamed for some of these diseases (Evans, 1989c; Duesberg and Schwartz, 1992). Shortly after this incident, a virus was also blamed for a fatal epidemic of neuropathy, including blinding, that started in the 1960s in Japan, but it turned out later to be caused by the

prescription drug clioquinol (Enterovioform, Ciba-Geigy) (Kono, 1975; Shigematsu *et al.*, 1975). In 1976 the CDC blamed an outbreak of pneumonia at a convention of Legionnaires on a “new” microbe, without giving consideration to toxins. Since the “Legionnaire’s disease” did not spread after the convention and the “Legionnaires bacillus” proved to be ubiquitous, it was later concluded that “CDC epidemiologists must in the future take toxins into account from the start” (Culliton, 1976). The Legionnaire’s disease fiasco is in fact the probable reason that the CDC initially took toxins into account as the cause of AIDS (Oppenheimer, 1992).

The pursuit of harmless viruses as causes of human cancer, supported since 1971 by the Virus-Cancer Program of the National Cancer Institute’s War On Cancer, was also inspired by discouraging faith in the germ theory (Greenberg, 1986; Duesberg, 1987; Shorter, 1987; Anderson, 1991; Editorial, 1991; Duesberg and Schwartz, 1992). For example, it was claimed in the 1960s that the rare Burkitt’s lymphoma was caused by the ubiquitous Epstein-Barr virus, 15 years after infection (Evans, 1989c). But the lymphoma is now accepted to be non-viral and attributed to a chromosome rearrangement (Duesberg and Schwartz, 1992). Further, it was claimed that noncontagious cervical cancer is caused by the widespread herpes virus in the 1970s, and by the widespread papilloma virus in the 1980s—but in each case cancer would occur only 30 to 40 years after infection (Evans, 1989c). Noninfectious causes like chromosome abnormalities, possibly induced by smoking, have since been considered or reconsidered (Duesberg and Schwartz, 1992). Further, ubiquitous hepatitis virus was proposed in the 1960s to cause regional adult hepatomas 50 years (!) after infection (Evans, 1989c). In the 1980s the rare, but widely distributed, human retrovirus HTLV-I was claimed to cause regional adult T-cell leukemias (Blattner, 1990). Yet the leukemias would only appear at advanced age, after “latent periods” of up to 55 years, the age when these “adult” leukemias appear spontaneously (Evans, 1989c; Blattner, 1990; Duesberg and Schwartz, 1992). Although the Virus-Cancer program has generated such academic triumphs as retroviral oncogenes (Duesberg and Vogt, 1970) and reverse transcriptase (Temin and Mizutani, 1970), it has been a total failure in terms of clinical relevance. Indeed, the pride

of retrovirologists in retrovirus-specific reverse transcription is the probable reason that inhibition of DNA synthesis with AZT is perceived, even now, as a “specific” antiretroviral therapy (Section 4.3.3).

The wishful thinking that viruses cause “slow” diseases and cancers faces four common problems: (1) the diseases or tumors occur on average only decades after infection; (2) the viruses are all inactive, if not defective, during fatal disease or cancer; (3) the “viral” tumors are all clonal, derived from a single cell (with a tumor-specific chromosome abnormality) that had emerged out of billions of identically infected cells of a given carrier; and (4) above all, no human cancers and none of the “slow viral diseases” are contagious (Rowe, 1973; Duesberg and Schwartz, 1992).

Therefore these viruses all fail Koch’s postulates, the acid test of the germ theory. And therefore these viruses are all assumed to be very “slow,” causing diseases only after long “latent periods” that exceed by decades the short periods of days or weeks that these viruses need to replicate and to become immunogenic. Because of their consistent scarcity, defectiveness and even complete absence from some tumors and slow diseases (Duesberg and Schwartz, 1992), the search for the presumably pathogenic latent viruses has been directed either at anti-viral antibodies, i.e. “seroepidemiological evidence” (Blattner *et al.*, 1988), or at artificially amplified viral DNA and RNA (Section 3.3) or at the “activation” of latent viruses, euphemistically called “virus isolation” (Section 2.2).

Accordingly cancer-, AIDS- and other slow-virologists try to discredit Koch’s postulates in favor of “modern concepts of causation.” For example, Evans states that, “. . . Koch’s postulates, great as they were for years, should be replaced with criteria reflecting modern concepts of causation, epidemiology, and pathogenesis and technical advances” (Evans, 1992). And Blattner, Gallo and Temin point out that Koch’s postulates are just a “useful historical reference point” (Blattner *et al.*, 1988), and Weiss and Jaffe find it “bizarre that anyone should demand strict adherence to these unreconstructed postulates 100 years after their proposition” (Weiss and Jaffe, 1990)—but they all fail to identify a statute of limitation for adherence to the virus-AIDS hypothesis. In addition, “cofactors” are assumed (a) to make up for the typi-

cal inertia of the viral pathogens or carcinogens, (b) to account for the clonality of the cancers via a clonal cellular cofactor, and (c) to help to close the enormous gaps between the very common infections and the very rare incidences of “slow” disease or cancer, that even the long “latent periods” could not close (Duesberg and Schwartz, 1992). The tumor virologist Rowe “recognized that the latent period may cover much of the life span of the animal and that the virus did not act alone but that the tumor response might require . . . treatment with a chemical carcinogen” (Rowe, 1973).

Despite the total lack of public health benefits and even negative consequences of these theories, such as the psychologically toxic prognoses that antibodies against HTLV-I or against papilloma virus signal future cancers (Duesberg and Schwartz, 1992), or that antibodies against HIV signal future AIDS and the need for AZT prophylaxis, the public and the majority of scientists have held on to them much longer than was justified in terms of scientific evidence. The irresistible appeal of the germ theory was the basis for each of these unproductive theories of the past, as it is the basis now for the universal and enthusiastic approval of the virus-AIDS hypothesis.

But unlike the mistaken germ theories of the past, the virus-AIDS hypothesis was a windfall not only for (1) the virologists and epidemiologists, but also for (2) the biotechnology companies who could develop virus-tests and antiviral drugs, (3) the AIDS patients who were relieved that a God-given, egalitarian virus rather than behavioral factors were to blame for their diseases, and (4) the politicians who had to confront the public and the gay (homosexual) lobby requesting action against AIDS. Indeed, a thoroughly intimidated public was happy, once more, to be offered protection by its scientists against another “deadly” virus, albeit for the highest price-tag ever.

6.2. Big Funding and Limited Expertise Paralyze AIDS Research

Ironically, AIDS research suffers not only from being tied to an unproductive hypothesis, it also suffers from the staggering funds it receives from governments (Section 1) and from conceptually matched private sources. Intended to buy a fast solution for AIDS, these funds have instead paralyzed AIDS research by creating an instant orthodoxy of retroviro-

ogists that fiercely protects its narrowly focused scientific expertise and global commercial interests (Booth, 1988; Rappoport, 1988; Nussbaum, 1990; Duesberg, 1991b, 1992b; Savitz, 1991; Connor, 1991, 1992).

The leaders of the AIDS orthodoxy are all veterans from the wars on “slow” and cancer viruses. Naturally they were highly qualified to fill the growing gaps in the virus-AIDS hypothesis with their “modern concepts of causation” (Evans, 1992), including long “latent periods,” “cofactors” and “seroepidemiological” arguments of causation (Sections 3.3, 3.4 and 3.5). When it became apparent that the first order mechanism of viral pathogenesis, postulating direct killing of T-cells, failed to explain immunodeficiency, the bewildering diversity of AIDS diseases, the many asymptomatic HIV infections, and HIV-free AIDS cases, the scientific method would have called for a new hypothesis. Instead the virus hunters have shifted the virus-AIDS hypothesis from a failed first order mechanism to a multiplicity of hypothetical second order mechanisms, including cofactors and latent periods, to fill the ever growing discrepancies between HIV and AIDS. By conjugating these second order mechanisms with a multiplicity of unrelated diseases, the virus-AIDS hypothesis has become by far the most mercurial hypothesis in biology. It predicts either diarrhea or dementia or Kaposi’s sarcoma or no disease, 1, 5, 10 or 20 years after 1 or 2000 sexual contacts with an antibody-HIV-positive person with or without an AIDS disease.

But the coup to rename dozens of unrelated diseases with the common name AIDS, proved to be the most effective weapon of the AIDS establishment in winning unsuspecting followers from all constituencies. By making AIDS a synonym for Kaposi’s sarcoma and candidiasis and dementia and diarrhea and lymphoma and lymphadenopathy, the road was paved for a common cause. Who would have accepted, prior to AIDS, that a dental patient caught candidiasis from her doctor’s Kaposi’s sarcoma? Or which scientist would accept it even now knowing the original data rather than just the corresponding press release? According to the sociologist David Phillips “researchers use newspapers as a ‘filter’ to help them decide which scientific article is worth reading” (Briefings, 1991) or more often which article is worth knowing about.

The control of AIDS research by the nationally and internationally funded AIDS orthodoxy via the popular and scientific press is almost total. It instructs science writers that faithfully report every “break-through” in HIV research and every “explosion” of the epidemic. It feeds scientific journals with over 10,000 HIV-AIDS papers annually and with advertisements for HIV tests and antiviral drugs (Schwitzer, 1992). The AIDS doctors are controlled by the companies created, consulted or owned by the AIDS establishment (Barinaga, 1992; Schwitzer, 1992). For example, the Physician’s Desk Reference 1992 instructs AIDS doctors about AZT with an exact copy of Burroughs Wellcome’s instructions. *Science* writers are warned against reporting minority views. For example, Fauci states: “Journalists who make too many mistakes, or who are sloppy, are going to find that their access to scientists may diminish” (Fauci, 1989). And Ludlam points out, “Whilst I support, and encourage the reporting of, minority views . . . If the belief that AIDS is not due to HIV becomes prevalent . . . (it) could lead directly to the deaths of countless misinformed individuals” (Ludlam, 1992). Any challengers are automatically outnumbered and readily marginalized by the sheer volume of the AIDS establishment. For example, the 12,000 scientists attending the annual international AIDS conference held in San Francisco in 1990 were only a fraction of the many who study the information encoded in the 9000 nucleotides of HIV. Says the HIV virologist Gallo when asked about a dissenter: “Why does the Institute of Medicine, WHO, CDC, National Academy of Sciences, NIH, Pasteur Institute and the whole body of world science 100 percent agree that HIV is the cause of AIDS?” (Liversidge, 1989).

Consequently there is no “peer-reviewed” funding for researchers who challenge the virus-AIDS hypothesis (Duesberg, 1991b; Maddox, 1991a; Bethell, 1992; Farber, 1992; Hodgkinson, 1992). Since HIV became the dominant focus of the billion-dollar AIDS-research (Coffin *et al.*, 1986; Institute of Medicine, 1988), there has not been even one follow-up of the many previous studies blaming sexual stimulants and psychoactive drugs for homosexual AIDS (Sections 4.4 and 4.5). None of the former “lifestyle” advocates (Section 2.2) have investigated whether drugs might cause AIDS without HIV. Instead drugs, if mentioned at all, were since described as risk factors for infection by HIV (Darrow

et al., 1987; Moss *et al.*, 1987; van Griensven *et al.*, 1987; Chaisson *et al.*, 1989; Weiss, S.H., 1989; Goudsmit, 1992; Seage *et al.*, 1992)—as if HIV could discriminate between hosts on the basis of their drug habits (Duesberg, 1992a). For example, Friedman-Kien concluded in 1982 and 1983 with Marmor *et al.* (1982) and Jaffe *et al.* (1983b) that the “lifetime exposure to nitrites . . .” was responsible for AIDS (Section 4.3.2). In 1990 he and his collaborators just mentioned nitrite use in HIV-free Kaposi’s sarcoma cases (Friedman-Kien *et al.*, 1990) and in 1992 they blamed viruses other than HIV for HIV-free AIDS cases, and drug use was no longer mentioned (Huang *et al.*, 1992).

Likewise all studies investigating transfusion-mediated immunodeficiency in hemophiliacs were frozen around 1987 (Table 3), once the virus-AIDS hypothesis had monopolized AIDS research. The question whether immunodeficient (!) HIV-free hemophiliacs would ever develop AIDS defining diseases was left unanswered and even became unaskable.

Fascinated by the past triumphs of the germ theory, the public, science journalists and even scientists from other fields never question the authority of their medical experts, even if they fail to produce useful results (Adams, 1989; Schwitzer, 1992). Medical scientists are typically credited for the virtual elimination of infectious diseases with vaccines and antibiotics, although most of the credit for eliminating infectious diseases is actually owed to vastly improved nutrition and sanitation (Stewart, 1968; McKeown, 1979; Moberg and Cohn, 1991; Oppenheimer, 1992). Indeed, the belief in the infallibility of modern science is the only ideology that unifies the 20th century. For example, in the name of the virus-AIDS hypothesis of the American Government and the American researcher Gallo, antibody-positive Americans have been convicted for “assault with a deadly weapon” because they had sex with antibody-negatives, Central Africa dedicates its limited resources to “AIDS testing,” the former U.S.S.R. conducted 20.2 million AIDS tests in 1990 and 29.4 million in 1991 to identify a total of 178 antibody-positive Soviets and communist Cuba even quarantines its own citizens if they are antibody-positive (Section 3.6).

Predictably the AIDS virus hunters, on their last crusade for the germ theory, have no regard for the current drug-use epidemic and its

many overlaps with American and European AIDS. Even direct evidence for the role of drugs in AIDS is fiercely rejected by the virus-AIDS orthodoxy (Booth, 1988; Moss *et al.*, 1988; Kaslow *et al.*, 1989; Baltimore and Feinberg, 1990; Ostrow *et al.*, 1990). Merely questioning the therapeutic or prophylactic benefits of AZT is protested by the AIDS establishment (Baltimore and Feinberg, 1990; Weiss and Jaffe, 1990; Anonymous, 1992; Freestone, 1992; Tedder *et al.*, 1992). The prejudice against noninfectious pathogens is so popular, that the virus-AIDS establishment uses it regularly to intimidate those who propose noninfectious alternatives, to censor their papers (Duesberg, 1992e) and even to question their integrity.

For example, an editorial in *Science* called me a “rebel without a cause for AIDS,” because denying HIV was to deny a cause altogether. The editorial quoted Baltimore as saying I was “irresponsible and pernicious” (Booth, 1988). An article in *Nature* called my drug hypothesis a “perilous message” that would “belittle ‘safe sex,’ would have us abandon AIDS screening . . . and curtail research into anti-HIV drugs.” “Arguments that AIDS (is) the result of evil vapors (poppers (!)), mal’aria . . . (are from) the last century.” “We . . . regard the critics as ‘flat-earthers’ bogged down in molecular minutiae and miasmatic theories of disease, while HIV continues to spread” (Weiss and Jaffe, 1990). This is said even though the article agrees that, “Duesberg is right to draw attention to our ignorance of how HIV causes disease . . .” (Weiss and Jaffe, 1990). Others declare “All attempts by epidemiologists to link AIDS to the use of amyl nitrite or other drugs as a direct cause of disease have failed . . . Duesberg’s continued attempts to persuade the public to doubt the role of HIV in AIDS are not based on facts” (Baltimore and Feinberg, 1990). Gallo called the author of the article, “Experts mount startling challenge to AIDS orthodoxy” in *The Sunday Times* (London) (Hodgkinson, 1992), “irresponsible both to myself (Gallo) and to HIV as the cause of AIDS” (Gallo, 1992). Further, Vandenbroucke and Pardoel argue, “If one is allowed to compare the evolution of scientific theories with the evolution of biologic nature in general, the poppers (nitrite inhalants) episode is the Neanderthal of modern epidemiology” (Vandenbroucke and Pardoel, 1989).

As a consequence there are no studies that investigate the long-term

effects of psychoactive drugs (Lerner, 1989; Pillai *et al.*, 1991; Bryant *et al.*, 1992). The toxicologist Lerner points out that “fewer than 60 are currently enrolled in fellowship programs on alcoholism and drug abuse in the entire country” (Lerner, 1989), although about 8 million Americans alone are estimated to use cocaine (Weiss, S.H., 1989; Finnegan *et al.*, 1992) and many more use other psychoactive drugs regularly (Section 4). This stands in contrast to the 40,000 annual AIDS cases that are studied by at least 40,000 AIDS researchers of which just 12,000 attended the annual International AIDS Conference in San Francisco in 1990.

Instead of warning against drugs, the AIDS establishment “educates” the public with its “clean needle” campaigns that drugs (albeit illegal) are safe, but bugs are not. For example, AIDS researcher Moss, citing Napoleon’s line “On s’engage et puis on voit,” recommends “clean needles” for “harm reduction” (Moss, 1987). Mindful of its educators, the public is unaware and even disinformed about the health risks of recreational drugs. A popular joke in point is the response of two “junkies” (drug addicts) sharing a syringe filled with an intravenous drug to a concerned colleague: “We are safe, because we use a clean needle and condoms.” The long “latent periods” between the gratification from recreational drugs, such as tobacco, alcohol, cocaine and nitrite inhalants, to their irreversible health effects unfortunately give credence to the “perilous message” that drugs are safe but bugs are not.

Particularly the victims of drug consumption prefer egalitarian infectious causes over noninfectious behavioral ones that imply personal responsibility (Shilts, 1985; Lauritsen and Wilson, 1986; Rappoport, 1988; Callen, 1990). For example, the executive director of the San Francisco based national “Project Inform,” an organization operated mainly for and by male homosexuals, Martin Delaney, informs its clients about a study documenting a “level of sexual contact and drug use which was shocking to the general public” as follows: “It (the study) might just as well have noted that most wore Levi’s (jeans) for all this told us about the cause of AIDS” (Project Inform, 1992). The organization collaborates with the NIH and is supported by grants from pharmaceutical companies including Burroughs Wellcome, the manufacturer of AZT (Project Inform, 1992).

In 1987, before AZT, Delaney advised gay men in his book *Strate-*

gies for Survival: A Gay Men's Health Manual for the Age of AIDS about the health effects of nitrite inhalants: "Possible heart damage; fibrillation (compulsive, erratic heart rhythms); possible stroke and resulting brain damage. Conducive to high-risk sexual behavior; distortion of judgment and senses. Statistical link to Kaposi's sarcoma (KS, an AIDS-related cancer); suspected immuno-suppression" (Delaney and Goldblum, 1987). Delaney's advice about amphetamines reads as follows: "Liver and heart damage; neuropathy (nerve damage); possible brain damage; weight loss; nutritional and vitamin depletion; adrenal depletion (uses up the body's energy reserves). Distorted judgment, values, senses, delusions of strength, anxiety, paranoia, rebound depression, financial strain, powerful addiction, conducive to high-risk sexual activity. Likely immunosuppression (not currently measured), potential for unknown and risky drug interactions, complication in treatment of brain disorders." Delaney also warns about the effects of cocaine: "Heart and lung damage, stroke, cardiovascular irregularities, possible physical addiction. Distortion of judgment, values, and senses, dangerous delusions of grandeur and strength, intense anxiety, paranoia, financial strain, leads to poor judgment about high-risk sexual activity. Likely immunosuppression (not currently measured); increased stress, if smoked, complicates treatment of pneumonia." The book also gives the basis for Delaney's intimate knowledge of drug toxicity: "He . . . has done work for the National Institute on Drug Abuse" (Delaney and Goldblum, 1987).

Clearly big science is not always good science, particularly if it is conceptually paralyzed by an unproductive hypothesis. I hope that the scientific evidence collected for this article will focus attention on the noninfectious causes of AIDS and prove that it is not "too late to correct" (Red Queen) the spell of the virus-AIDS hypothesis by the scientific method. Considering noninfectious causes may prove to be as beneficial to the challenge of AIDS as it was, for example, to the challenge of pellagra. Indeed, a few investigators have recently smuggled recreational drugs as "cofactors" of HIV (Haverkos and Dougherty, 1988; Haverkos, 1990) or even more cautiously as cofactors of cofactors of HIV (Archibald *et al.*, 1992) into the highly fundable virus-AIDS hypothesis. One investigator even dared to document that drugs are

sufficient for pediatric AIDS, if only in preliminary reports (Koch, 1990; Koch *et al.*, 1990). A complete report of the data (Section 4.5) was not published for political reasons (Thomas Koch, personal communication). And the “100 percent” consensus on HIV claimed by Gallo in 1989 (Liversidge, 1989) is eroding just a bit in the face of a growing group of dissenters, some of which united in the “Group for the Scientific Reappraisal of the HIV/AIDS Hypothesis” (DeLoughry, 1991; Bethell, 1992; Bialy and Farber, 1992; Farber, 1992; Hodgkinson, 1992; Project Inform, 1992; Nicholson, 1992; Ratner, 1992; Schoch, 1992).

Note Added in Proof

Sparked by an article in *Newsweek* (Cowley, 1992), numerous HIV-free AIDS cases were unexpectedly reported by many independent (!) investigators at the VIII International Conference on AIDS/III STD World Congress in Amsterdam in July 1992 (now a joint meeting with sexually transmitted diseases, STDs). Surprisingly, some of the recently announced HIV-free AIDS cases had been studied for years (Altman, 1992a; Cohen, 1992a,b; Laurence *et al.*, 1992), even by the CDC (Spira and Jones, 1992). As a result the CDC had to alter its long-held position that HIV causes all AIDS to “HIV causes the vast majority of AIDS cases . . .” (Nicholson, 1992). In its monthly *HIV/AIDS Surveillance Reports* the CDC still states that “AIDS is a specific group of diseases which are indicative of severe immunosuppression related to infection with the human immunodeficiency virus (HIV)” (Centers for Disease Control, 1992b). The AIDS risk factors of most of these HIV-free “AIDS patients” were reported to be “intravenous drugs, unprotected sex and transfusions” and the corresponding diseases were Kaposi’s sarcoma and pneumonia (Cowley, 1992).

AIDS-virus matchmakers soon reached the consensus that an as yet undiscovered “new AIDS virus,” that “doesn’t appear any more contagious than HIV” (Cowley, 1992), was to be blamed for HIV-negative AIDS (Bowden *et al.*, 1991; Castro *et al.*, 1992; Huang *et al.*, 1992; Altman, 1992a,b; Cohen, 1992a,b; Laurence *et al.*, 1992). And the director for AIDS research at the NIH reassured the public, “If there is something, scientists will find it” (News Report, 1992). States *The New York Times*, “Arguably, the greatest thrills for a scientist are in discov-

ering a new microbe, a new disease, cure and prevention . . . Many . . . know how quickly the exhilaration that comes from believing they are on the verge of making such a discovery vanishes when the initial findings cannot be confirmed” (Altman, 1992b).

However, the new HIV-free AIDS cases are entirely consistent with those listed above that were caused by drug consumption and other noncontagious risk factors (Section 4.5). Although public recognition of HIV-free AIDS cases is new, the new cases just complement the over 1200 cases of “acquired” immunodeficiency and AIDS-defining diseases described above including 334 hemophiliacs (Section 3.4.4.5, Table 3), 265 male homosexuals (Sections 3.4.4.3 and 4.5), 444 intravenous drug users (Section 4.5) and 183 mostly male tuberculosis patients from Florida (Pitchenik *et al.*, 1987, 1990). If the 2466 HIV-free AIDS cases from Africa were included (Section 3.4.4.8), the number of documented HIV-free AIDS cases would exceed 3000!

Moreover healthy HIV carriers who have been infected for over 10 years and have transmitted their HIV to at least 5 healthy persons via blood transfusions over 7–10 years ago have now received public recognition (Altman, 1992c; Learmont *et al.*, 1992). These cases supplement the 1 million Americans, 0.5 million Europeans, 0.3 million Thais and 6 million Africans who are healthy, although most had been infected by 1985 (Section 3.5.1).

Thus both predictions of the hypothesis that AIDS is noncontagious are now generally accepted: (1) HIV-free AIDS and (2) AIDS-free transmission of HIV. Asks John Maddox, editor of *Nature*, “Does that mean Duesberg has been right all along, and that HIV plays no part in the causation of AIDS?” (Maddox, 1992b). Indeed, it would be an evolutionary miracle if the last decade had generated three different AIDS viruses, HIV-1, HIV-2 and the “new AIDS virus,” when no such virus has ever emerged before in the history of medicine.

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Note added in proof:

Ten years after designating AIDS infectious based on clusters of cases, the CDC published in 1995: “Such clusters may be difficult to identify because most persons with AIDS have had contact with many different people. In particular, drug users and homosexual and bisexual men may have had contact with hundreds of partners that they did not know very well.” (Drotman, Peterman and Friedman-Kien, 1995).

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