
The chemical bases of the various AIDS epidemics: recreational drugs, anti-viral chemotherapy and malnutrition

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In 1981 a new epidemic of about two-dozen heterogeneous diseases began to strike non-randomly growing numbers of male homosexuals and mostly male intravenous drug users in the US and Europe. Assuming immunodeficiency as the common denominator the US Centers for Disease Control (CDC) termed the epidemic, AIDS, for acquired immunodeficiency syndrome. From 1981–1984 leading researchers including those from the CDC proposed that recreational drug use was the cause of AIDS, because of exact correlations and of drug-specific diseases. However, in 1984 US government researchers proposed that a virus, now termed human immunodeficiency virus (HIV), is the cause of the non-random epidemics of the US and Europe but also of a new, sexually random epidemic in Africa. The virus-AIDS hypothesis was instantly accepted, but it is burdened with numerous paradoxes, none of which could be resolved by 2003: Why is there no HIV in most AIDS patients, only antibodies against it? Why would HIV take 10 years from infection to AIDS? Why is AIDS not self-limiting via antiviral immunity? Why is there no vaccine against AIDS? Why is AIDS in the US and Europe not random like other viral epidemics? Why did AIDS not rise and then decline exponentially owing to antiviral immunity like all other viral epidemics? Why is AIDS not contagious? Why would only HIV carriers get AIDS who use either recreational or anti-HIV drugs or are subject to malnutrition? Why is the mortality of HIV-antibody-positives treated with anti-HIV drugs 7–9%, but that of all (mostly untreated) HIV-positives globally is only 1.4%? Here we propose that AIDS is a collection of chemical epidemics, caused by recreational drugs, anti-HIV drugs, and malnutrition. According to this hypothesis AIDS is not contagious, not immunogenic, not treatable by vaccines or antiviral drugs, and HIV is just a passenger virus. The hypothesis explains why AIDS epidemics strike non-randomly if caused by drugs and randomly if caused by malnutrition, why they manifest in drug- and malnutrition-specific diseases, and why they are not self-limiting via anti-viral immunity. The hypothesis predicts AIDS prevention by adequate nutrition and abstaining from drugs, and even cures by treating AIDS diseases with proven medications.

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1. Origins of the AIDS epidemics of the US, Europe and Africa

1.1 AIDS in the US and Europe

In the spring of 1981 the US Centers for Disease Control (CDC), the nation's sentinel of infectious diseases, first

reported a mysterious epidemic of previously known diseases that selectively affected growing numbers of young male homosexuals, intravenous drug users and a few minor risk groups such as hemophiliacs and recipients of blood transfusions (Centers for Disease Control 1981a, b, 1986). The diseases of the new AIDS epidemic included

Keywords. AIDS not contagious; AIDS not immunogenic; drug-free HIV survivors; HIV-free AIDS; mortality on anti-HIV drugs; non-exponential AIDS epidemics; non-random US/European AIDS; pediatric drug-AIDS; random African AIDS; recreational drug-AIDS

Abbreviations used: CDC, US Centers for Disease Control; NIH, US National Institutes of Health; FDA, US Food and Drug Administration; WHO, World Health Organization.

Kaposi's sarcoma, bacterial and fungal (pneumocystis and candida) pneumonia, oral yeast infections, dementia, diarrhea, herpes, tuberculosis, lymphoma, weight loss, toxoplasmosis, chronic fevers, etc. (table 1), (Centers for Disease Control 1986). A similar, non-random epidemic was soon also reported in Europe by the World Health Organization (WHO), (Downs *et al* 1987). The selective distribution of these epidemics in the US and European population immediately suggested risk group- or lifestyle-specific causes.

However, the plethora of AIDS diseases was not, and still is not randomly distributed even among the different risk groups (table 2). For example, Kaposi's sarcoma was exclusively diagnosed in male homosexual risk groups using nitrite inhalants and other psychoactive drugs as aphrodisiacs (Newell *et al* 1984; Haverkos *et al* 1985; Selik *et al* 1987; Duesberg 1988; Haverkos and Dougherty 1988; Beral *et al* 1990). Bacterial pneumonia was primarily diagnosed in children from mothers using psychoactive drugs during pregnancy (Novick and Rubinstein 1987; Duesberg 1988, 1992; Centers for Disease Control and Prevention 1997). Tuberculosis and pneumonia were, and still are more prevalent in intravenous drug users and "crack" (cocaine) smokers than in other risk groups (Lerner 1989; Duesberg 1992; Duesberg and Rasnick 1998). Pneumocystis pneumonia and dementia are common in both of these risk groups (Selik *et al* 1987; Duesberg 1992; Duesberg and Rasnick 1998).

Hemophiliacs and other transfusion recipients from the US and Europe exclusively present with pneumonia and yeast infections (Curran *et al* 1984; Duesberg 1992, 1995c). The non-random distribution of these diseases in different risk groups, then and now, again suggests risk group-specific causes, rather than a common one.

Only 3 months after first detecting the new epidemics of old diseases, the CDC named all of them, AIDS, for Acquired Immune Deficiency Syndrome, assuming that immunodeficiency was their common denominator (Centers for Disease Control 1981b). According to their most recent AIDS definition of 1993, there are now 26 AIDS defining diseases (Centers for Disease Control 1986, 1992). However, about one third of the CDC's collection of AIDS diseases are neither caused by, nor necessarily associated with immunodeficiency (table 1), (Duesberg and Rasnick 1998). Examples are Kaposi's sarcoma, lymphoma, dementia, and weight loss (see table 1 for the share of these among the AIDS diseases of the US in 1997). Although the rest of the CDC's AIDS diseases are indeed microbial diseases, they are typically opportunistic microbial diseases, in which the causative microbe depends on a defective immune system to cause disease. Examples are tuberculosis, yeast infections and pneumocystis pneumonia (Duesberg 1992), (table 1). By contrast, in a normal immune system, opportunistic microbes are harmless passengers – the reason why such diseases are

Table 1. CDC-defined AIDS diseases and "conditions" in the US in 1997, the last year the Centers for Disease Control published the distribution of AIDS diseases.

Disease	AIDS-diagnosis	Percentage of cases*	Case numbers per 60,161
No disease	< 200 T cells and antibody against HIV	61	36,634
Microbial disease	Pneumocystis	38	9,145
	Candidiasis	16	3,846
	Tuberculosis and Mycobacteria	15	3,537
	Cytomegalovirus	7	1,638
	Pneumonia	5	1,347
	Herpes virus	5	1,250
	Cryptococcus	5	1,168
	Toxoplasmosis	4	1,073
Non-microbial disease	Weight loss/wasting	18	4,212
	Kaposi's sarcoma	7	1,500
	Dementia	6	1,409
	Lymphoma/leukemia	4	850
	Cervical cancer	1	144

*According to the CDC, "The sum of percentages is greater than 100 because some patients are reported with more than one illness [disease or condition]. Of persons reported with AIDS-defining opportunistic illnesses, 65% also were reported with severe HIV-related immunosuppression [corresponding almost exactly to the share of the microbial diseases among the total of 23,527 AIDS-defining diseases]. . . . The 36,634 adults/adolescents presented on this table are those persons reported with immunosuppression as their only AIDS-indicator condition [rather than disease]" (Centers for Disease Control and Prevention 1997).

Table 2. Risk group-specific AIDS diseases.

Disease	Risk groups					
	Male homosexual	Intravenous drugs	AZT recipient	US, Europe child	Hemophilia, transfusion	African
Kaposi's sarcoma	++*					
Lymphoma	+		+			
Bacterial pneumonia				++		+
Tuberculosis		++		++		++
Dementia	+	+	+	+		
Weight loss	+	+	+	+		++
Yeast	+	+	+	+	+	+
Pneumocystis pneumonia	++	+	+	+	+	

*+ and ++ represent common and highly representative diseases respectively.

not transmitted to healthy contacts, as for example to the doctors that treat AIDS patients (see below, § 3, table 4).

Since 1981 the AIDS epidemics of the US and Europe have increased steadily for a decade and, after reaching peaks in the early 1990s, they have all decreased to about 1/2 of their peak levels now (figure 1a), (World Health Organization 2001b). By 2001 the US epidemic had generated a total of 816,149 AIDS cases and the European epidemic 251,021 AIDS cases (Centers for Disease Control and Prevention 2001; World Health Organization 2001b). To this day, the AIDS epidemics of the US and Europe have remained highly non-random: 80% of all patients from the US and 80% from Europe are males (World Health Organization 2001a). In the US about 2/3 of all AIDS cases are male homosexuals and about 1/3 are male and female intravenous drug users. In Europe about 1/2 are male homosexuals and about 1/2 are intravenous drug users [note that over 75% of intravenous drug users are males (Duesberg and Rasnick 1998)]. In addition both epidemics include fringe groups of hemophiliacs and other transfusion recipients (1%) and children born to drug-addicted mothers (1%) (World Health Organization 2001a).

1.2 African epidemic

A new AIDS epidemic was also claimed to have emerged in sub-Saharan Africa in 1984 (Bayley 1984; Piot *et al* 1984; Seligmann *et al* 1984; Van de Perre *et al* 1984; Quinn *et al* 1986, 1987). In sharp contrast to its US/European namesakes, the African AIDS epidemic is randomly distributed between the sexes and not restricted to behavioural risk groups (Blattner *et al* 1988; Duesberg 1988; World Health Organization 2001a). Hence sub-Saharan African AIDS is compatible with a random, either microbial or chemical cause.

The African epidemic is also a collection of long-established, indigenous diseases, such as chronic fevers, weight

loss, alias "slim disease", diarrhea and tuberculosis (table 2), (Colebunders *et al* 1987; Konotey-Ahulu 1987a, b, 1989; Pallangyo *et al* 1987; Duesberg 1992). However, the distribution of AIDS-defining diseases in Africa differs strongly from those in the US and Europe (table 2). For example, the predominant and most distinctive AIDS diseases in the US and Europe, *Pneumocystis carinii* pneumonia and Kaposi's sarcoma, are almost never diagnosed in Africa (Goodgame 1990; Abouya *et al* 1992).

According to the WHO the African epidemic has increased from 1984 until the early 1990s, similar to the epidemics of the US and Europe, but has since leveled off to generate about 75,000 cases annually (figure 1c), (World Health Organization 2001b, and back issues). By 2001, Africa had reportedly generated a cumulative total of 1,093,522 cases (World Health Organization 2001b). However, there are three reasons for questioning these numbers:

- (i) During the African AIDS epidemic, the sub-Saharan African population has grown, at an annual rate of about 2.6% per year – from 378 million in 1980 to 652 million in 2000 (US Bureau of the Census International Data Base 2001). Thus Africa had gained since 1980 274 million people, the equivalent of the whole population of the US! Therefore, a possible, above-normal loss of 1 million Africans over a period in which over 200 millions were gained is statistically hard, if not impossible to verify – unless the African AIDS diseases were highly distinctive.
- (ii) However, the African AIDS-defining diseases are clinically indistinguishable from conventional African morbidity and mortality (see above).
- (iii) Further the HIV-based definition of AIDS (see § 3) can not be used in Africa to distinguish AIDS-defining from otherwise indistinguishable diseases, because as of 1985 the WHO decided at a conference in Bangui, Africa, to accept African AIDS diagnoses without HIV-tests (see § 3). This was done because these tests are unaffordable

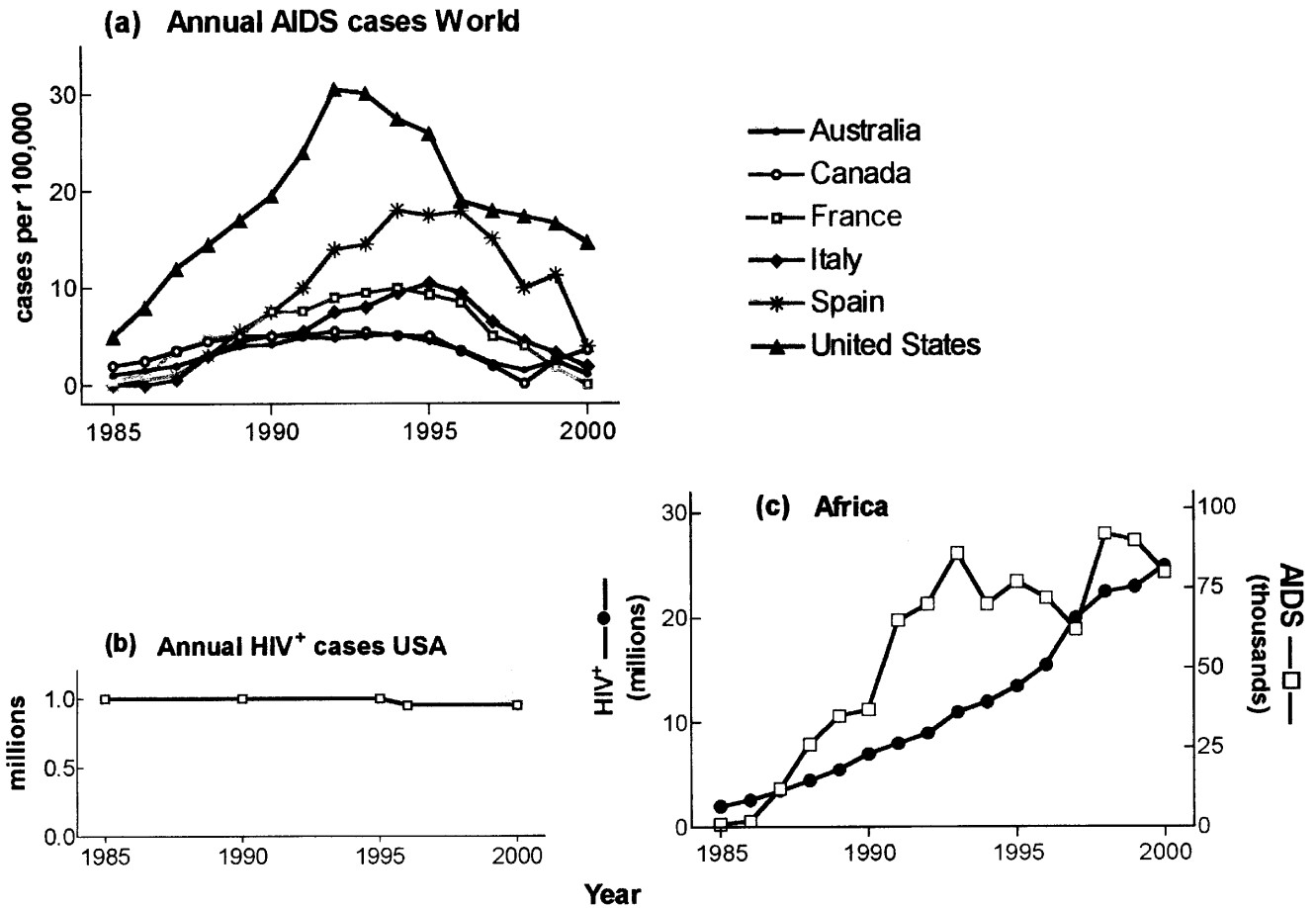


Figure 1. Time courses of (a) the American, European and Australian AIDS epidemics since 1985 according to Centers for Disease Control and Prevention 2001 and redrawn from the World Health Organization (1999), (b) the incidence of HIV antibody carriers in the US since 1985 according to the Centers of Disease Control (CDC), and (c) the incidence of AIDS and HIV antibody carriers in Africa according to the WHO. See text for further explanations and references.

in most African countries (World Health Organization 1986; Fiala 1998; Fiala *et al* 2002). Thus without the CDC's HIV standard (§ 3), the diagnosis of African AIDS is arbitrary.

In view of the many epidemiological and clinical distinctions of African AIDS from its US/European namesakes and the many uncertainties about the diagnosis of African AIDS, both the novelty of African AIDS and its relationship to the US/European AIDS epidemics have been called into question (Hodgkinson 1996; Fiala 1998; Shenton 1998; Gellman 2000; Stewart *et al* 2000; Malan 2001; Fiala *et al* 2002; Gisselquist *et al* 2002; Ross 2003). Indeed, all available data are compatible with an old African epidemic of malnutrition and poverty-associated diseases under a new name (Konotey-Ahulu 1987a, b; Oliver 2000; Stewart *et al* 2000).

In the following we will try to find the most probable causes for the various AIDS epidemics based on epidemiological, clinical, microbial and biochemical evidence.

2. 1981–1984: The “lifestyle”-AIDS hypothesis

Hardly anybody remembers now, that shortly after the origins of the AIDS epidemics in the US and Europe scientists had already discovered that illicit psychoactive and aphrodisiac drugs, consumed at massive doses, were the common denominators and probable causes of the new AIDS patients. Drugs such as cocaine, heroin, nitrite inhalants, amphetamines, steroids and lysergic acid had become widely available and popular in the US and Europe during and after the Vietnam war and the coincident era of “gay liberation” (legal indemnity of homosexuality)

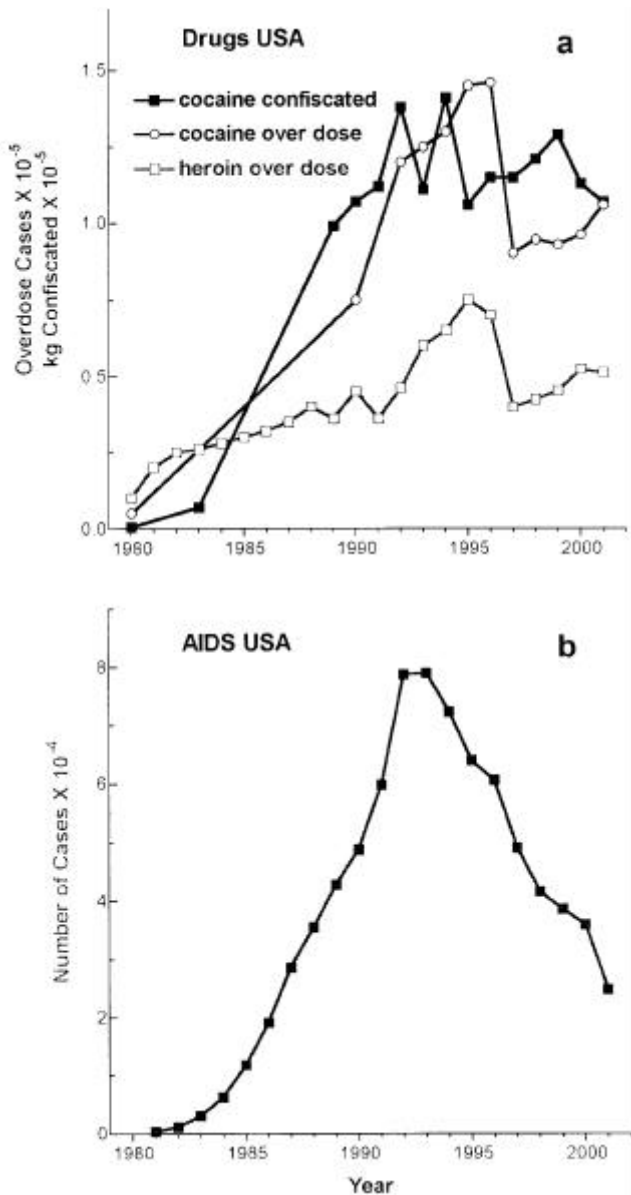


Figure 2. (a) Chronology of the recreational drug epidemic in the US since 1980, based on various indicators. The data for cocaine confiscations are from the US Bureau of Justice Statistics, table 4.36 Federal drug seizures (1988–2001), Federal drugs seizures (1989–2001), (www.albany.edu/sourcebook) and from Duesberg and Rasnick (1998). The data for cocaine and heroin hospital emergencies are from the Substance Abuse and Mental Health Services Administration-Office of Applied Studies (2002), from Emergency Department Trends of the Drug Abuse Warning Network, Final Estimates 1994–2001, Rockville, MD (2002), and from the Drug Abuse Warning Network, Dept. Health Human Services, August, DAWN Series: D-21, DHHS Publication No. (SMA) 02-3635 (<http://www.drugabusestatistics.samhsa.gov>). (b) The chronology of the US AIDS epidemic according to the CDC (Centers for Disease Control 2001).

(Duesberg and Rasnick 1998). The phenomenon was dubbed the “drug explosion” in the US and Europe. Its chronology is documented in figure 2 based on cocaine and heroin hospital-emergencies and confiscations of cocaine. Figure 2 extends drug use statistics described by us earlier until 2001 (Duesberg and Rasnick 1998), and also compares the chronologies of the drug and AIDS epidemics in the US (see also figure 1a and § 4).

The first series of publications linking homosexual AIDS with drugs, particularly aphrodisiac nitrite inhalants (Gottlieb *et al* 1981), was published in the *New England Journal of Medicine* in 1981 together with an editorial by AIDS researcher David Durack suggesting that drugs are the causes of AIDS (Durack 1981). Dozens of further drug-AIDS studies soon followed from all prominent AIDS researchers of the time, including Blattner, Bregman, Curran, Dougherty, Des Jarlais, Drotman, Friedman-Kien, Goedert, Haverkos, Jaffe, Marmor, McManus, Mildvan, Moss, Newell, Oppenheimer, Ortiz, Rivera, and Stoneburner (Goedert *et al* 1982; Marmor *et al* 1982; McManus *et al* 1982; Jaffe *et al* 1983; Mathur-Wagh *et al* 1984; Newell *et al* 1984; Haverkos *et al* 1985; Moss 1987; Haverkos and Dougherty 1988; Stoneburner *et al* 1988; Oppenheimer 1992).

Even the CDC, normally just a survey agency, conducted epidemiological studies of their own, which confirmed that male homosexuals at risk for AIDS and with AIDS were using batteries of recreational and aphrodisiac drugs (table 3), (Jaffe *et al* 1983). Not even one male homosexual at behavioural risk for AIDS or with AIDS was found to be drug-free by the CDC. However, some CDC investigators suggested that nitrites depend on “infectious cofactors” to cause AIDS diseases (Haverkos 1988).

The perfect correlations between recreational drug use and AIDS became the basis for the hypothesis that drugs, or the drug use-“lifestyle” is the cause of AIDS (Shilts

Table 3. CDC 1983*: Drug use by American male homosexuals with AIDS and at risk for AIDS.

Drugs	Percentage users among 50 AIDS cases and 120 at risk for AIDS
Nitrite inhalants	96
Ethylchloride	35–50
Cocaine	50–60
Amphetamines	50–70
Phenylcyclidine	40
LSD	40–60
Metaqualone	40–60
Barbiturates	25
Marijuana	90
Heroin	10
Drug-free	None reported

*(Jaffe *et al* 1983).

1987; Oppenheimer 1992). Moreover, the findings that specific drugs, as for example nitrite inhalants, correlated with specific AIDS diseases, such as immune suppression and Kaposi's sarcoma, directly support the lifestyle hypothesis (Goedert *et al* 1982; Marmor *et al* 1982; Haverkos and Dougherty 1988).

By contrast, the African epidemic had been reduced right from its presumed origin in 1984 to the consequences of malnutrition and lack of drinkable water, alias poverty, consistent with its random distribution in the population (Mims and White 1984; Seligmann *et al* 1984).

In sum all clinical and epidemiological data available on AIDS in 1984 made a coherent case for lifestyle- or chemical AIDS, caused by recreational drugs or malnutrition.

3. 1984: The virus-AIDS hypothesis takes over

By 1983 AIDS had become big enough in the American and European press to pique the interest of the influential infectious disease establishment, particularly the cancer virus hunters. At that time the virus hunters had been engaged for over a decade in president Nixon's War on Cancer with unsuccessful attempts to find a human cancer virus (Duesberg 1996b; Fujimura 1996; de Harven 1999). Now they were looking for new diseases that could be attributed to viruses (Duesberg 1987). Perhaps AIDS could at last yield clinically relevant lymphoma-, Kaposi's sarcoma- or immunodeficiency-viruses (Duesberg 1996b).

Indeed, virus hunters from the CDC were the first to alert the public that AIDS may be "transmissible" (Francis *et al* 1983). A similar alert came from a French virus team, which had discovered a retrovirus in a homosexual man at risk for AIDS, which a year later became the accepted cause of AIDS (Barre-Sinoussi *et al* 1983). News, that the cause of AIDS may be a virus, and thus transmissible to the general population, immediately set off a national panic that opened the doors for new surveillance programs by the CDC and predictably set off a race among virus hunters for the AIDS virus (Shilts 1987).

According to an international press conference called by the US Secretary of Health and Human Services in Washington DC on 23 April 1984, that race was won by government researchers from the NIH who had found in some AIDS patients antibodies against a new retrovirus closely related to a hypothetical human leukemia virus (Altman 1984). The virus was introduced as fortunate fallout of the failed War on Cancer. The next day the new virus was already termed, the "AIDS virus", by the *New York Times* (Altman 1984). Overnight nearly all AIDS researchers dropped the lifestyle-AIDS hypothesis to work on the new "AIDS virus", which was already endorsed by the US government. The CDC's director of the Task

Force on Kaposi's Sarcoma and Opportunistic Infections, James Curran, was the only one who later announced the reason for his conversion to the new "AIDS virus": "That's where the money is" (Shilts 1987).

The National Academy, the Institute of Medicine and the CDC quickly united the infectious disease establishment under the leadership of David Baltimore, who had received a Nobel prize for his work on retroviruses, to provide practical recommendations for the intimidated public. Their recommendations were published in two consecutive monographs of *Confronting AIDS* (Institute of Medicine and National Academy of Sciences 1986; Institute of Medicine 1988), and initiated history's biggest and most expensive anti-viral program ever, costing \$ 93.3 billion by 2000 to the US taxpayer alone (Johnson 2000). At the same time an international committee of retrovirologists officially sealed the seemingly tight package of a new "AIDS virus" and the CDC's assumption that immunodeficiency was the common denominator of the 26 AIDS-defining diseases (table 1) by naming it, Human Immunodeficiency Virus (HIV) (Coffin *et al* 1986).

Even before the AIDS virus became the officially accepted cause of AIDS, the CDC had already made antibodies against the virus the only definitive criterion to diagnose any of the heterogeneous diseases as AIDS in 1985 (Centers for Disease Control 1985, 1987, 1992). Their unorthodox decision to use antibodies against the virus (normally functioning as a vaccine), instead of the virus, for the diagnosis of AIDS was based on the flawed analogy with some bacterial pathogens. For example, syphilis bacteria can be pathogenic despite the presence of antibodies, e.g. the Wassermann test for syphilis (Brandt 1988). But viruses are typically unable to enter cells in the presence of anti-viral antibodies – the basis for the effectiveness of Jennerian vaccines. Because of the CDC's decision, AIDS is diagnosed worldwide if antibody against (!) HIV, rather than HIV, is detectable in a patient along with any of the CDC's 26 diseases. Since 1992 even low T-cell counts are diagnosed as a condition, termed "HIV/AIDS", which is treatable with anti-HIV drugs provided it occurs in the presence of antibodies against HIV (Centers for Disease Control 1992), (see table 1, and § 4.2).

3.1 Discrepancies between the predictions of the virus-AIDS hypothesis and the facts

Despite its spectacular birthday the HIV-AIDS hypothesis has remained entirely unproductive to this date: There is as yet no anti-HIV-AIDS vaccine, no effective prevention and not a single AIDS patient has ever been cured – the hallmarks of a flawed hypothesis. Indeed the hypothesis was born with several serious birth defects and has developed further defects since; most of these should

have given pause to HIV-AIDS researchers to rethink and reconsider. However, in the race to claim a share of the new viral cause for AIDS and of virus-based AIDS treatments, “The Trojan horse of emergency” (Szasz 2001) was saddled so quickly that there was little time and no interest to address these defects, not even the most fundamental ones (Weiss and Jaffe 1990; Cohen 1994; O’Brien 1997).

An analysis of the defects of the HIV-AIDS hypothesis based on its failure to predict AIDS facts is shown in table 4. Our analysis is based on the most recent and most authoritative case made for the HIV-AIDS hypothesis since 1984, namely the Durban Declaration that was published in *Nature* in 2000 and has been signed by “over 5,000 people, including Nobel prizewinners” (The Durban Declaration 2000). It can be seen in table 4 that the HIV-hypothesis fails to predict 17 specific facts of AIDS. The most fundamental discrepancy between the HIV-AIDS hypothesis and the facts is the paradox, that a latent, non-cytopathic and immunologically neutralized retrovirus [a virus that is inherently not cytopathic (Duesberg 1987)], that is only present in less than 1 out of 500 susceptible T-cells and rarely expressed in a few of those, would cause a plethora of fatal diseases in sexually active, young men and women. And, that the plethora of the diseases attributed to this virus would not show up for 5–10 years after infection (table 4). As a result of the many discrepancies between the HIV hypothesis and the facts, we conclude that HIV is not sufficient for AIDS, and is most compatible with being a passenger virus.

Surprisingly our conclusion is supported by a survey of AIDS researchers conducted by the *New York Times*, shortly after the publication of the Durban Declaration. At the 20th anniversary of AIDS, on 30 January 2001, the *New York Times* interviewed a dozen leading AIDS researchers for an article that turned into a list of questions, “The AIDS questions that linger” (Altman 2001a), similar to those asked by us in table 4:

“In the 20 years since the first cases of AIDS were detected, scientists say they have learned more about this viral disease than any other, and few have disputed the claim. ... Despite the gains ... experts say reviewing unanswered questions could prove useful as a measure of progress for AIDS and other diseases. Such a list could fill a newspaper, and even then would create debate. (E.g.): How does H.I.V. subvert the immune system? ... Why does AIDS predispose infected persons to certain types of cancer and infections and not others? ... Dr Anthony S Fauci, the director of the National Institute of Allergy and Infectious Diseases, said, ‘It is the rare person who gets up and strips himself of his personal agenda and articulates what we really do not know because by saying that

they would diminish the impact of their own work, which is their agenda’. (Regarding anti-HIV medications:) ... the new drugs do not completely eliminate H.I.V. from the body, so the medicines, which can have dangerous side effects, will have to be taken for a lifetime and perhaps changed to combat resistance. The treatments are now so complicated that it is difficult, expensive and time-consuming to answer basic and practical questions. What combinations of drugs should be started first and when? Why do side effects like unusual accumulations of fat in the abdomen and neck develop? ... Anti-H.I.V. drugs suppress replication of the virus, which should give the functioning parts of the immune system a chance to eliminate remaining virus. That does not happen. ‘So something is bizarre about that, that we don’t understand’, Dr Fauci said. Is a vaccine possible? ... many unanswered questions exist about whether and when one can be developed.”

Thus HIV-AIDS researchers have not solved the discrepancies and paradoxes of the HIV-AIDS hypothesis, but still do not follow the scientific method of searching for alternative explanations (Costello 1995).

Since 19 years of HIV-AIDS research have failed to produce tangible benefits for AIDS patients and risk groups, and since there are no paradoxes in nature only flawed hypotheses, the scientific method calls for an alternative, testable hypothesis. Here we offer one such hypothesis. Our hypothesis extends the early, and now abandoned “lifestyle” hypothesis (§ 2) and subsequent drug-AIDS hypotheses from us and others (Duesberg 1992; Duesberg and Rasnick 1998).

4. Chemical AIDS

“Historically, the first step in determining the cause of any disease has always been to find out if there is anything, apart from the disease itself, that sufferers have in common” (Cairns 1978). However, the traditional search for the cause is only completed, if something that sufferers have in common can also be shown to cause the disease; in other words if Koch’s postulates can be fulfilled (Merriam-Webster 1965). This is true for viruses just as much as for drugs. Following this tradition, we try here to provide proof of principle for our drug and malnutrition hypothesis of AIDS – alias chemical AIDS.

4.1 The chemical-AIDS hypothesis and its predictions

The chemical-AIDS hypothesis proposes that the AIDS epidemics of the US and Europe are caused by recreational drugs, alias lifestyle, and anti-HIV drugs (Duesberg

Table 4. The HIV-AIDS hypothesis*: 17 predictions versus the facts.

No.	Prediction	Fact
1.	Since HIV is “the sole cause of AIDS”, it must be abundant in AIDS patients based on “exactly the same criteria as for other viral diseases.”	But, only antibodies against HIV are found in most patients (1–7)**. Therefore, “HIV infection is identified in blood by detecting antibodies, gene sequences, or viral isolation.” But, HIV can only be “isolated” from rare, latently infected lymphocytes that have been cultured for weeks <i>in vitro</i> – away from the antibodies of the human host (8). Thus HIV behaves like a latent passenger virus.
2.	Since HIV is “the sole cause of AIDS”, there is no AIDS in HIV-free people.	But, the AIDS literature has described at least 4621 HIV-free AIDS cases according to one survey – irrespective of, or in agreement with allowances made by the CDC for HIV-free AIDS cases (55).
3.	The retrovirus HIV causes immunodeficiency by killing T-cells (1–3).	But, retroviruses do not kill cells because they depend on viable cells for the replication of their RNA from viral DNA integrated into cellular DNA (4, 25). Thus, T-cells infected <i>in vitro</i> thrive, and those patented to mass-produce HIV for the detection of HIV antibodies and diagnosis of AIDS are immortal (9–15)!
4.	Following “exactly the same criteria as for other viral diseases”, HIV causes AIDS by killing more T-cells than the body can replace. Thus T-cells or “CD4 lymphocytes . . . become depleted in people with AIDS”.	But, even in patients dying from AIDS less than 1 in 500 of the T-cells “that become depleted” are ever infected by HIV (16–20, 54). This rate of infection is the hallmark of a latent passenger virus (21).
5.	With an RNA of 9 kilobases, just like polio virus, HIV should be able to cause one specific disease, or no disease if it is a passenger (22).	But, HIV is said to be “the sole cause of AIDS”, or of 26 different immunodeficiency and non-immunodeficiency diseases, all of which also occur without HIV (table 2). Thus there is not one HIV-specific disease, which is the definition of a passenger virus!
6.	All viruses are most pathogenic prior to anti-viral immunity. Therefore, preemptive immunization with Jennerian vaccines is used to protect against all viral diseases since 1798.	But, AIDS is observed – by definition – only after anti-HIV immunity is established, a positive HIV/AIDS test (23). Thus HIV cannot cause AIDS by “the same criteria” as conventional viruses.
7.	HIV needs “5–10 years” from establishing antiviral immunity to cause AIDS.	But, HIV replicates in 1 day, generating over 100 new HIVs per cell (24, 25). Accordingly, HIV is immunogenic, i.e. biochemically most active, within weeks after infection (26, 27). Thus, based on conventional criteria “for other viral diseases”, HIV should also cause AIDS within weeks – if it could.
8.	“Most people with HIV infection show signs of AIDS within 5–10 years” – the justification for prophylaxis of AIDS with the DNA chain terminator AZT (§ 4).	But, of “34.3 million . . . with HIV worldwide” only 1.4% [= 471,457 (obtained by subtracting the WHO’s cumulative total of 1999 from that of 2000)] developed AIDS in 2000, and similarly low percentages prevailed in all previous years (28). Likewise, in 1985, only 1.2% of the 1 million US citizens with HIV developed AIDS (29, 30). Since an annual incidence of 1.2–1.4% of all 26 AIDS defining diseases combined is no more than the normal mortality in the US and Europe (life expectancy of 75 years), HIV must be a passenger virus.
9.	A vaccine against HIV should (“is hoped” to) prevent AIDS – the reason why AIDS researchers try to develop an AIDS vaccine since 1984 (31).	But, despite enormous efforts there is no such vaccine to this day (31). Moreover, since AIDS occurs by definition only in the presence of natural antibodies against HIV (§ 3), and since natural antibodies are so effective that no HIV is detectable in AIDS patients (see No. 1), even the hopes for a vaccine are irrational.
10.	HIV, like other viruses, survives by transmission from host to host, which is said to be mediated “through sexual contact”.	But, only 1 in 1000 unprotected sexual contacts transmits HIV (32–34), and only 1 of 275 US citizens is HIV-infected (29, 30), (figure 1b). Therefore, an average un-infected US citizen needs 275,000 random “sexual contacts” to get infected and spread HIV – an unlikely basis for an epidemic!

(Table 4. *Contd.*)

Table 4.

No.	Prediction	Fact
11.	“AIDS spreads by infection” of HIV.	But, contrary to the spread of AIDS, there is no “spread” of HIV in the US. In the US HIV infections have remained constant at 1 million from 1985 (29) until now (30), (see also The Durban Declaration and figure 1b). By contrast, AIDS has increased from 1981 until 1992 and has declined ever since (figure 1a).
12.	Many of the 3 million people who annually receive blood transfusions in the US for life-threatening diseases (51), should have developed AIDS from HIV-infected blood donors prior to the elimination of HIV from the blood supply in 1985.	But there was no increase in AIDS-defining diseases in HIV-positive transfusion recipients in the AIDS era (52), and no AIDS-defining Kaposi’s sarcoma has ever been observed in millions of transfusion recipients (53).
13.	Doctors are at high risk to contract AIDS from patients, HIV researchers from virus preparations, wives of HIV-positive hemophiliacs from husbands, and prostitutes from clients – particularly since there is no HIV vaccine.	But, in the peer-reviewed literature there is not one doctor or nurse who has ever contracted AIDS (not just HIV) from the over 816,000 AIDS patients recorded in the US in 22 years (30). Not one of over ten thousand HIV researchers has contracted AIDS. Wives of hemophiliacs do not get AIDS (35). And there is no AIDS-epidemic in prostitutes (36–38). Thus AIDS is not contagious (39, 40).
14.	Viral AIDS – like all viral/microbial epidemics in the past (41–43) – should spread randomly in a population.	But, in the US and Europe AIDS is restricted since 1981 to two main risk groups, intravenous drug users and male homosexual drug users (§ 1 and 4).
15.	A viral AIDS epidemic should form a classical, bell-shaped chronological curve (41–43), rising exponentially via virus spread and declining exponentially via natural immunity, within months (see figure 3a).	But, AIDS has been increasing slowly since 1981 for 12 years and is now declining since 1993 (figure 1a), just like a lifestyle epidemic, as for example lung cancer from smoking (figure 3b).
16.	AIDS should be a pediatric epidemic now, because HIV is transmitted “from mother to infant” at rates of 25–50% (44–49), and because “34.3 million people worldwide” were already infected in 2000. To reduce the high maternal transmission rate HIV-antibody-positive pregnant mothers are treated with AZT for up to 6 months prior to birth (§ 4).	But, less than 1% of AIDS in the US and Europe is pediatric (30, 50). Thus HIV must be a passenger virus in newborns.
17.	“HIV recognizes no social, political or geographic borders” – just like all other viruses.	But, the presumably HIV-caused AIDS epidemics of Africa and of the US and Europe differ both clinically and epidemiologically (§ 1, table 2). The US/European epidemic is highly nonrandom, 80% male and restricted to abnormal risk groups, whereas the African epidemic is random.

*All quotes are from The Durban Declaration, the most authoritative edition of the HIV-AIDS hypothesis to date, which was signed “by over 5000 people, including Nobel prizewinners” and published in *Nature* in 2000 (The Durban Declaration 2000). **Numbers in parentheses are for the following references: (1) (Marx 1984); (2) (Gallo *et al* 1984); (3) (Altman 1984); (4) (Duesberg 1987); (5) (Duesberg 1988); (6) (Duesberg 1994); (7) (Duesberg and Bialy 1996); (8) (Levy *et al* 1984); (9) (Hoxie *et al* 1985); (10) (Anand *et al* 1987); (11) (Langhoff *et al* 1989); (12) (Duesberg 1996b); (13) (Weiss 1991); (14) (Cohen 1993); (15) (McCune 2001); (16) (Harper *et al* 1986); (17) (Schnittman *et al* 1989); (18) (Hazenberg *et al* 2000); (19) (Duesberg 1988); (20) (Blattner *et al* 1988); (21) (Enserink 2001); (22) (Fields 2001); (23) (Centers for Disease Control 1992); (24) (Duesberg and Rasnick 1998); (25) (Duesberg 1992); (26) (Clark *et al* 1991); (27) (Daar *et al* 1991); (28) (World Health Organization 2001b); (29) (Curran *et al* 1985); (30) (Centers for Disease Control and Prevention 2001); (31) (Cohen 2003); (32) (Jacquez *et al* 1994); (33) (Padian *et al* 1997); (34) (Gisselquist *et al* 2002); (35) (Duesberg 1995c; Hoots and Canty 1998); (36) (Mims and White 1984); (37) (Rosenberg and Weiner 1988); (38) (Root-Bernstein 1993); (39) (Hearst and Hulley 1988); (40) (Sande 1986); (41) (Bregman and Langmuir 1990); (42) (Anderson 1996); (43) (Fenner *et al* 1974); (44) (Blattner *et al* 1988); (45) (Duesberg 1988); (46) (Blanche *et al* 1989); (47) (Rogers *et al* 1989); (48) (European Collaborative Study 1991); (49) (Connor *et al* 1994); (50) (World Health Organization 2000); (51) (Duesberg 1992); (52) (Ward *et al* 1989); (53) (Haverkos *et al* 1994); (54) (Simmonds *et al* 1990); (55) (Duesberg 1993d).

1992, 1996b; Duesberg and Rasnick 1998), and by other non-contagious risk factors such as immunosuppressive proteins associated with transfusions of blood clotting factors (Duesberg 1995c; Hoots and Canty 1998). According to our hypothesis pediatric AIDS is due to prenatal consumption of recreational and anti-HIV drugs by unborn babies together with their pregnant mothers (Duesberg 1992; Duesberg and Rasnick 1998). The chemical basis of African AIDS is proposed to be malnutrition and lack of drinkable water (Duesberg 1992, 1996b; Duesberg and Rasnick 1998) – exactly as proposed originally by the now leading HIV-AIDS researchers Fauci and Seligmann: “The commonest cause of T-cell immunodeficiency worldwide is protein-calorie malnutrition” (Seligmann *et al* 1984) and others (Mims and White 1984), (see also § 1).

The chemical AIDS hypothesis makes the following testable predictions:

- (i) Patients of the various epidemics have drug use, medications, malnutrition or other chemical pathogens in common.
- (ii) Distinct chemical pathogens cause distinct AIDS-defining diseases. Since chemicals are not self-replicating, like viruses, pathogenicity is dose- and thus also time-dependent (Duesberg and Rasnick 1998). Take for example the average 20 years of smoking to cause cancer (figure 3b), (Cairns 1978).
- (iii) Since there is no immunity against drugs or malnutrition, neither drug-, nor malnutrition-diseases, nor the corresponding epidemics are self-limiting. In contrast to an infectious epidemic, the time curves of chemical epidemics are not bell-shaped (see figure 3).
- (iv) People who are not subject to drugs or malnutrition, or discontinue drug use or malnutrition before irreversible damage has occurred, do not develop AIDS, regardless of antibodies against HIV.

Here, we will focus on new and poorly known evidence confirming each of these predictions, but also make references to prior supportive evidence by others and us.

4.2 Prediction 1: AIDS coincides with recreational and anti-viral drugs in the US and Europe and with malnutrition in Africa

4.2a Recreational drugs: Annually, the CDC and the WHO confirm that about 1/3 of all AIDS patients from the US and about 1/2 of those from Europe are intravenous users of cocaine, heroin, amphetamines and other illicit, psychoactive drugs, since the beginning of the AIDS epidemics (see § 1). Most babies with AIDS in the US and Europe are also born to mothers who have used recreational drugs (and antiviral drugs, see below) during pregnancy according to the CDC, the WHO and independent publications reviewed below and previously (Duesberg 1992; Duesberg and Rasnick 1998). In addition

the CDC and WHO confirm that about 2/3 of the AIDS patients in the US and 1/2 of those in Europe are male homosexuals (§ 1), but, after the lifestyle hypothesis was abandoned in 1984, they did no longer report their drug use.

However, rare independent investigations have confirmed continued use of illicit recreational drugs by male homosexuals ever since the origins of the epidemic (Lauritsen and Wilson 1986; Haverkos and Dougherty 1988; Rappoport 1988; Duesberg 1992; Lauritsen 1994; Duesberg and Rasnick 1998). Since there is no general knowledge about the male-homosexual-AIDS-drug connection now, we have summarized in table 5 rare, post-1984 studies which demonstrate that male homosexuals with AIDS or at risk for AIDS have continued to use nitrite inhalants, amphetamines, cocaine, heroin, steroids, and other recreational drugs to this date, just as originally shown by the proponents of the lifestyle hypothesis including the CDC (see § 2, table 3) (Duesberg and Rasnick 1998). As this article went to press, the *San Francisco Chronicle* published a 3-part front-page story on how

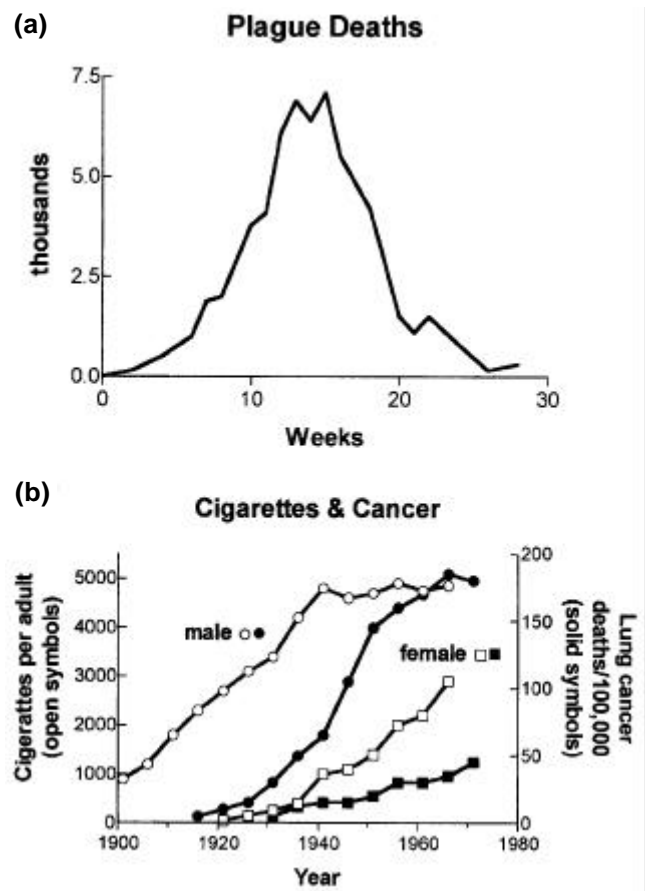


Figure 3. The time course of (a) a classical microbial epidemic, the plague in London of 1665 adapted from Anderson (1996), and (b) of the classical behavioural, or “lifestyle” epidemics of smoking and lung cancer in men and women in England in the 20th century, adapted from Cairns (1997).

“Crystal Meth (amphetamine) fuels HIV”. According to the article “the state’s top AIDS and HIV prevention officials came up with the smoking gun of all statistics: Gay men in California who use speed are twice as likely to be HIV-positive . . .” (Heredia 2003a). But the question whether meth “fuels” AIDS without HIV was not asked, even though the featured case of a gay meth-addict had AIDS-defining dementia and opportunistic infections (Heredia 2003a, b). Further, we confirm and extend, in tables 6 and 7, the correlations between maternal drug use and baby-AIDS documented previously (Novick and Rubinstein 1987; Duesberg 1992; Root-Bernstein 1993; Duesberg and Rasnick 1998; Farber 1998). The continuation of drug use in the HIV era is not surprising in the absence of any advice from the medical establishment, that nitrite inhalants and other drugs may cause AIDS (Lerner 1989).

4.2b Anti-viral DNA chain terminators and protease inhibitors: It is also little known that since 1987 thousands of US citizens and Europeans with AIDS (Kolata 1987), and that since 1990 even larger numbers of healthy HIV antibody-positives are on lifetime prescriptions of inevitably toxic DNA chain-terminators, such as azidothymidine (AZT), and protease inhibitors as anti-HIV drugs (Volberding *et al* 1990). The original doses of these prescriptions were 1.5 g per day of AZT or other DNA chain-terminator for clinically ill patients (Fischl *et al* 1987) and 0.5 g per day for asymptomatic, HIV-positives with low T-cell counts (Volberding *et al* 1990). As of

1996 the DNA chain-terminators were mixed with HIV-protease inhibitors to generate so-called “drug cocktails” (Ho 1995; Stolberg 2001). By 1996 200,000 US citizens (Hall 1996), and by 2001/2002 over 450,000 (France 2001; Altman 2002), were taking prescriptions of such drugs to prevent or cure AIDS (Stolberg 2001). Due to the CDC’s 1993-definition of AIDS, well over half of these 450,000 treated subjects were clinically healthy at the time they started taking the anti-HIV drugs (table 1) and are thus not patients (Centers for Disease Control 1992; Centers for Disease Control and Prevention 1997). The asymptomatic HIV-positives are treated according to the slogan, “Time to hit HIV, early and hard”, that was introduced by the *New England Journal of Medicine* in 1995 (Ho 1995). Thus recreational and anti-HIV drugs are the common denominator of AIDS in the US, and also in Europe (see below).

4.2c African AIDS coincides with malnutrition: The case for malnutrition and lack of drinkable water as the common denominator and probable cause of African AIDS in the HIV-era has been made by scientific (Mims and White 1984; Seligmann *et al* 1984; Konotey-Ahulu 1987a, b, 1989; Fiala 1998; Oliver 2000; Stewart *et al* 2000; Ross 2003) and non-scientific observers (Hodgkinson 1996; Shenton 1998; Malan 2001). The non-scientific observers even include the United Nations (Namango and World Food Program of the United Nations 2001) and president Mbeki of South Africa (Cherry 2000; Gellman 2000).

4.3 Prediction 2: Drugs cause AIDS and other diseases

4.3a Literature confirms that illicit recreational drugs cause AIDS defining and other drug-specific diseases: We have recently summarized the evidence from over 60 publications, beginning in 1909 (Achard *et al* 1909), which prove that regular consumption of illicit recreational drugs causes all AIDS defining and additional drug-specific diseases at time and dose-dependent rates (Duesberg 1996b; Duesberg and Rasnick 1998). At recreational doses, addictions ranging from years to over a decade are typically required to reach pathogenic thresholds. Thus the literature confirms the original “lifestyle”- or drug AIDS hypothesis.

4.3b Epidemiological drug dose-AIDS-response curves: In figure 2 (§ 2) we have already shown that the chronology of the epidemic of illicit drug-use in the US during the 1980s and 1990s closely paralleled the US AIDS epidemics, see also Duesberg and Rasnick (1998). A report from the White House, underwritten by president Clinton, provides additional data: It states in 1996 that the number of regular users of illicit recreational drugs in the US soared from a negligible background in the early 1960s to a high of 25 million, or about 10% of the US population, in the late 1980s (Clinton and The White House 1996; Duesberg and Rasnick 1998). Since its peak

Table 5. Studies describing illicit recreational (IR) and antiviral (AV) drug use by male homosexuals with AIDS and at risk for AIDS in the HIV era, since 1984.

Drugs	Reference and Year
IR	(Haverkos <i>et al</i> 1985; Newell <i>et al</i> 1985)
IR	(Lauritsen and Wilson 1986)
IR	(Darrow <i>et al</i> 1987)
IR	(Haverkos and Dougherty 1988; Rappoport 1988)
IR	(Adams 1989; Archer <i>et al</i> 1989; Kaslow <i>et al</i> 1989)
IR	(Lifson <i>et al</i> 1990; Ostrow <i>et al</i> 1990)
IR	(Eggers and Weyer 1991)
IR	(Archibald <i>et al</i> 1992)
IR + AV	(Ascher <i>et al</i> 1993b; Ostrow <i>et al</i> 1993; Schechter <i>et al</i> 1993)
IR + AV	(Lauritsen 1994; Sadownick 1994; Veugelers <i>et al</i> 1994)
IR	(Haverkos and Drotman 1995)
IR	(Gibbons 1996; Haverkos 1996; Haverkos and Drotman 1996)
IR	(McNall and Remafedi 1999)
IR	(Craib <i>et al</i> 2000; Dukers <i>et al</i> 2000; Pauk <i>et al</i> 2000)
IR	(Colfax <i>et al</i> 2001; Diamond <i>et al</i> 2001; Mansergh <i>et al</i> 2001; Mattison <i>et al</i> 2001; Woody <i>et al</i> 2001)
IR + AV	(Botnick <i>et al</i> 2002; Bull <i>et al</i> 2002)

IR, Illicit recreational drugs such as nitrite and other inhalants, amphetamines, cocaine, heroin, steroids; AV, antiviral drugs such as DNA chain terminators, protease inhibitors and others.

Table 6. Diseases and mortality of HIV antibody-positive people on anti-HIV drugs that do not occur in, or exceed the rates of untreated controls.

AIDS-defining	Other	References
Immunodeficiency, Leukopenia	Anemia, Neutropenia	(Gill <i>et al</i> 1987; Kolata 1987; Richman <i>et al</i> 1987; Dournon <i>et al</i> 1988; Jacobson <i>et al</i> 1988; Mir and Costello 1988; Alcabes <i>et al</i> 1993; Poznansky <i>et al</i> 1995; Kline <i>et al</i> 1998; Levy 1998; Anonymous 1999; Blanche <i>et al</i> 1999; Mocroft <i>et al</i> 1999)
Fever	Nausea	(Richman <i>et al</i> 1987; Volberding <i>et al</i> 1990; Smothers 1991; Race <i>et al</i> 1998; Fellay <i>et al</i> 2001)
Dementia		(Hitchcock 1991; Smothers 1991; Bacellar <i>et al</i> 1994)
Weight loss		(Poznansky <i>et al</i> 1995; Moye <i>et al</i> 1996; Anonymous 1999)
Lymphoma		(Pluda <i>et al</i> 1990)
Diarrhea	Lipodystrophy, "protease paunch"	(Duesberg and Rasnick 1998; Levy 1998; Brinkman <i>et al</i> 1999; Carr <i>et al</i> 2000; Fellay <i>et al</i> 2001)
	Muscle atrophy	(Richman <i>et al</i> 1987; Dalakas <i>et al</i> 1990; Till and MacDonnell 1990; Hitchcock 1991; Blanche <i>et al</i> 1999)
	Mitochondrial dysfunction	(Dalakas <i>et al</i> 1990; Blanche <i>et al</i> 1999; Carr <i>et al</i> 2001)
	Hepatitis	(Braeu <i>et al</i> 1997; Saves <i>et al</i> 1999; Carr <i>et al</i> 2000, 2001; France 2001)
	Birth defects	(Kumar <i>et al</i> 1994; Newschaffer <i>et al</i> 1999)
	Nephritis	(Fogelman <i>et al</i> 1994)
	Lactic acidosis	(Scalfaro <i>et al</i> 1998)
	Heart infarct	(Levy 1998; Altman 2001a; Carr <i>et al</i> 2001)
Death	Death	(Dournon <i>et al</i> 1988; Goedert <i>et al</i> 1994; Seligmann <i>et al</i> 1994; Veugelers <i>et al</i> 1994; Fischl <i>et al</i> 1989, 1995; Duesberg 1995c, 1996a; Sabin <i>et al</i> 1996; Anonymous 1999; Mocroft <i>et al</i> 1999; de Souza <i>et al</i> 2000; Kuhn <i>et al</i> 2000)

Table 7. Diseases and mortality in HIV-free human babies, and in HIV-free animals treated with anti-HIV drugs before and after birth (BB, AB).

Species	AIDS-defining	Other disease	References
Human Babies (AB)	Fever, Pneumonia	Anemia, Mitochondrial dysfunction	(Blanche <i>et al</i> 1999), (Heresi <i>et al</i> 1997)
Animals (AB): Mice, rats, dogs, monkeys	Lymphopenia, Weight loss, Leukemia, T-cell depletion, Thymic atrophy, Death of 25/30 mice	Anemia, neutropenia, Thrombocytopenia, Bone marrow depletion, Lymphotoxicity, Myelodysplasia, Muscle atrophy, Nephrotoxicity, Hepatotoxicity	(Ayers 1988; Cronkite and Bullis 1990; Thompson <i>et al</i> 1991; McKallip <i>et al</i> 1995; Omar <i>et al</i> 1996; Grossman <i>et al</i> 1997; Inoue <i>et al</i> 1997; Gerschenson <i>et al</i> 2000)
Animals (BB)	Death	Lung, liver, vaginal cancer, Retarded development, Abortion	(Toltzis <i>et al</i> 1993; Olivero <i>et al</i> 1997)

in the late 1980s-early 1990s, the US drug epidemic has declined to an estimated 13 million regular users in 1996 (Los Angeles Times 1998; White House Office of National Drug Control Policy 1998), again roughly paralleling the course of the AIDS epidemic (figure 2).

In addition, a fast-rising epidemic of volatile nitrite inhalants, primarily among male homosexuals, was identified in the US by Newell *et al*, Lauritsen and Wilson,

and the National Institute on Drug Abuse (Lauritsen and Wilson 1986; Haverkos and Dougherty 1988; Newell *et al* 1988). It started in the late 1970s – immediately preceding the male homosexual AIDS sub-epidemics of Kaposi's sarcomas and pneumonias (§ 1). Newell *et al* (1988) documented that recreational nitrite inhalant – alias "popper" – use had increased from negligible numbers in the 1960s to 5 million users of one ounce per week (!)

in 1979 in the US. They even recorded the first nitrite-linked Kaposi-cases, 3 years before the first description of AIDS. The following surveys lend further support to the many synchronies between the drug and AIDS epidemics: Duesberg (1988), Haverkos and Dougherty (1988), Rappoport (1988), Duesberg (1992), Oppenheimer (1992), Lauritsen (1994), Haverkos (1996), and Duesberg and Rasnick (1998).

We think that the chronological overlaps between the epidemics of drug-use and drug-specific AIDS diseases are epidemiological dose-response curves, and thus correlative proof of principle that drugs cause AIDS.

4.3c HIV-AIDS researchers confirm that recreational drugs cause AIDS and other diseases – despite efforts to suppress this information: In their efforts to promote the view that “HIV is the sole cause of AIDS” (The Durban Declaration 2000), the proponents of the HIV hypothesis try to exclude all non-HIV causes, particularly the illicit drugs that are the basis of the competing “lifestyle”-AIDS hypothesis.

For example, the *Lancet* published in 1993 a Canadian epidemiological study, “HIV and the etiology of AIDS”, which found that 88% of AIDS cases in a cohort of male homosexuals at risk for AIDS had used nitrite inhalants and that 75–80% of the same cohort had also used “cocaine, heroin, amphetamines, lysergic acid dimethyl amide, or methylenedioxy amphetamine” (Schechter *et al* 1993). One of the subjects even passed away on an “overdose” of recreational drugs during the study. In addition an undisclosed percentage (but in 1993 certainly a high percentage, see above) was also prescribed the DNA chain-terminator AZT as anti-HIV drug (Duesberg 1993a, c). Thus not a single drug-free AIDS patient was identified. But, the study concluded, “drugs and sexual activity is rejected by these data” as causes of AIDS. Nevertheless, the authors acknowledged that their study “does not rule out a role for cofactors . . .”.

Publishing in a high-profile commentary in *Nature* a Californian HIV-AIDS team also investigated the question, “Does drug use cause AIDS?” (Ascher *et al* 1993a). The authors studied 215 HIV-positive homosexual AIDS patients of which all were either “heavy” or “light” users of nitrites, of which unnamed percentages had also consumed amphetamines, cocaine and marijuana, and of which an unnamed percentage was also prescribed the DNA chain-terminator AZT as anti-HIV drug (Ascher *et al* 1993a; Duesberg 1993a, c, 1995a). Thus again, not a single drug-free patient was identified (Duesberg 1993a, c; Ascher *et al* 1995; Ellison *et al* 1996; Duesberg and Rasnick 1998). But, the authors concluded that, “when controlled for HIV serostatus, there is no overall effect of drug use on AIDS”. Moreover, the authors did not inform the reader that their unpublished databank included 45 patients which had used drugs and had AIDS-defining diseases, but were HIV-free, and thus did not support

their conclusion (Ellison *et al* 1996). But the authors did inform the reader that, “The energies of Duesberg and his followers could better be applied to unraveling the enigmatic mechanism of the HIV pathogenesis of AIDS”. Despite numerous other *ad hominem*s, and the fact that Duesberg was named 13 times, the editor of *Nature* vetoed a response, and even published his veto in an article, “Has Duesberg a right of reply?” (Maddox 1993).

Science also quoted a toxicologist ready to blame all consequences of heroin addiction on HIV, because “heroin is a blessedly un toxic drug” (Cohen 1994).

In an anonymous response to our hypothesis that drugs cause AIDS (Duesberg 1995b; Duesberg and Rasnick 1998), the National Institutes of Health (NIH) acknowledge drug-AIDS correlations on their website, *The evidence that HIV causes AIDS*, but reject causation: “Because many HIV-infected mothers abuse recreational drugs, some (unnamed researchers) have argued that maternal drug use itself causes pediatric AIDS. However, studies (un-referenced) have consistently shown that babies who are not HIV-infected do not develop AIDS (per HIV-AIDS definition), regardless of their mothers’ drug use.” Despite, “similar rates of alcohol, tobacco, cocaine, heroin and methadone use . . .” none of “248 uninfected children” would develop AIDS (National Institute of Allergy and Infectious Diseases and National Institutes of Health 2001). However, the NIH does not mention even one study that has ever described an AIDS-baby born to an HIV-positive, but drug-free mother, to prove their website’s claim that “HIV causes AIDS”. The NIH also does not mention the AIDS-defining and other birth defects of HIV-free “crack” (cocaine) babies born to HIV-free drug-addicted mothers that are described in the literature (Toufexis 1991; Duesberg 1992; Duesberg and Rasnick 1998).

In contrast to the studies selected and promoted by the NIH, the scientific literature has shown that nearly all AIDS-babies from the US and Europe were born to mothers who had used either recreational or anti-HIV drugs or both during pregnancy (Duesberg and Rasnick 1998), (§ 4.2a and 4.3d, e, table 7).

4.3d Anti-HIV drugs cause AIDS defining and other drug-specific diseases – regardless of the presence of antibodies to HIV: The fundamental problem of any chemical anti-virus “therapy” is that the cell carries out all viral biochemical functions. Thus all anti-viral treatments are inevitably anti-cell treatments. In the case of HIV, this problem is compounded by the notorious biochemical inertia of HIV in antibody-positive people, and by the extremely low multiplicity of infection of only 1 in 500 T-cells (table 4). Thus in antibody-positive people there are no HIV functions that could be targeted by DNA chain-terminators. As a result all treatments designed to inhibit nucleic acid and protein synthesis of HIV with

DNA chain-terminators only inhibit cellular nucleic acid and protein synthesis. Since elimination of the few cells that are latently infected is clinically not detectable, “therapeutic” results are typically reported in terms of various lab markers, primarily the “viral load”, rather than in restored health (Ho *et al* 1995; Wei *et al* 1995; Palella *et al* 1998; Hogg *et al* 2001). Even the term “viral load” is deceptive, because it suggests that there is a high virus titer, although infectious virus is typically not detectable. Instead this term describes the amount of viral DNA fragments that can be generated *in vitro* by the polymerase chain reaction from RNA of rare, antibody neutralized virus or of DNA of rare, latently infected cells isolated from the patient (table 4), (Duesberg 1993b; Duesberg and Bialy 1996). Nevertheless, the anti-HIV drugs have the unintended benefits of functioning as true “anti-biotics”, because of their general toxicity to all living things. As such they will also reduce the load of opportunistic microbial diseases that affect most AIDS patients (table 1), (Cohen 1987; Palella *et al* 1998). In the following we briefly review the primary effects of the DNA chain-terminators and protease inhibitors on the biochemistry of the cell.

(i) *DNA chain-terminators*: Currently nearly all anti-HIV prescriptions include DNA chain-terminators, that were originally developed 40 years ago, long before AIDS, to kill growing human cells as cancer therapy by terminating DNA synthesis (Horwitz *et al* 1964). Considering their mechanism of action, the DNA chain-terminators are inevitably cytotoxic, and thus immunotoxic like most other chemotherapies (Stedman’s Medical Dictionary 1982; Oliver 2000). DNA chain-terminators were first licensed as anti-HIV drugs in 1987, although their immunotoxicity or bone “marrow suppression” was immediately recognized (Kolata 1987; Richman *et al* 1987), see also (Nussbaum 1990; Duesberg 1996b). The inevitable immunotoxicity and lethality of AZT was confirmed in AIDS patients within less than a year after its licensing as anti-HIV-AIDS drugs (Kolata 1987; Richman *et al* 1987; Dournon *et al* 1988; Mir and Costello 1988). The label of a 100 mg-sample from the Sigma Chemical Co, a non-medical supplier, even advertises the inevitable toxicity of the DNA chain-terminator AZT with a skull and cross bones (figure 4a).

Yet, the DNA chain-terminators are currently prescribed at doses of about 500 mg per day (§ 4.2, and below). For example, a typical prescription flask with 100 capsules of 100 mg *Retrovir* (AZT) from the medical supplier Burroughs Wellcome instructs its late user (see below), “Take 1 capsule 5 times daily”, but does not mention cellular or human toxicity (see figure 4b). Even HIV-positive pregnant mothers are prescribed 500 mg of AZT per day during the second and third trimester of preg-

nancy to reduce the probability of transmission of HIV to their babies by 17% (from 25% to 8%) – at the cost of having to treat 100% of the pregnant mothers and babies with AZT (Connor *et al* 1994), (see table 6 and particularly table 7 below for consequences).

(ii) *HIV protease inhibitors*: The HIV protease inhibitors were designed to inhibit specifically auto-proteolytic processing of HIV proteins, which is necessary for HIV assembly (Fields 2001). But since no therapeutic effects were observed at the low doses at which these inhibitors “block HIV replication in the test tube” (The Durban Declaration 2000), the “anti-viral” doses were increased 4–5 orders of magnitude above what is needed to render HIV noninfectious *in vitro*, or to 1 to 2 g of inhibitor per day (Rasnick 1997). The high doses of protease inhibitors currently administered to patients are at minimum 50 times that needed to completely inhibit the cellular, intestinal aspartyl protease cathepsin D (calculation based on the Roche inhibitor Saquinavir; the Abbott inhibitor Ritonavir is 1000 times more potent against cathepsin D than Saquinavir), (Deeks *et al* 1997).

Mice in which cathepsin D is deleted develop anorexia, their “Thymus and spleen undergo massive destruction with fulminant loss of T and B cells”, and die about 26 days after birth (Saftig *et al* 1995). Thus protease inhibitors can cause at least three AIDS defining diseases, anorexia (weight loss), T-cell deficiency and death (see § 4e). In addition, diarrhea – which is also an AIDS defining disease (Centers for Disease Control 1985, 1986, 1987) – is a common problem with all the protease inhibitors (table 6).

(iii) *Drug cocktails*: AZT and other DNA chain-terminators are now typically supplemented by inhibitors of proteases to form drug “cocktails” (Ho 1995; Palella *et al* 1998; Day 2000; Stolberg 2001). A daily dose of these includes about 1 g of one or more DNA chain-terminators per clinically ill person and 0.5 g per asymptomatic HIV-positive per day (Stolberg 2001) (see also § 4.2b), which is the equivalent of $1.5\text{--}3 \times 10^6$ molecules of DNA chain-terminators per body cell!

Here, we present evidence that anti-HIV drugs cause AIDS defining diseases, other diseases and death, both (i) in the presence and (ii) in the absence of HIV.

(i) *Diseases and death in HIV-positives treated with anti-HIV drugs*: A sudden 10-fold increase in the mortality of HIV-positive British hemophiliacs, right after the introduction of AZT in 1987, made scientific headlines in 1995, because the increased mortality was attributed to HIV by the authors of the study, i.e. Darby *et al* (1995), as well as by the editor of *Nature*, “More conviction on HIV and AIDS” (Maddox 1995). Even the editor of the *Lancet* wrote an editorial asking, “Will Duesberg now concede defeat” (Horton 1995)? Darby *et al* based their conclusion on the sudden 10-fold increase of the hemo-

philiacs' mortality in 1987, shown in figure 5, on the facts that the increased mortality was restricted to HIV-positive hemophiliacs and that the increase was independent of the degree of hemophilia (which is inversely proportional to the life expectancy of the patient).

But, by 1987 transfusions of blood and factor VIII had already infected most hemophiliacs for a long time. Most of them were already infected before 1984 (about 75% in the US), because all blood supplies with HIV antibodies were banned after the introduction of the HIV-antibody



Figure 4. (a) The label on a bottle containing 100 mg of the DNA chain-terminator AZT from the Sigma Chemical Co, USA. The advisory on the label reads: "TOXIC. Toxic by inhalation in contact with skin and if swallowed. Target organ(s): Blood bone marrow. If you feel unwell, seek medical advice (show the label where possible). Wear suitable protective clothing". The amount of AZT in the bottle is one fifth of the daily dose recommended for asymptomatic HIV-positives (§ 4.2b), and of the daily dose prescribed to pregnant, HIV-positive mothers (§ 4.3d). (b) The label on a prescription flask containing 100 capsules of 100 mg AZT, termed Retrovir, by the medical manufacturer, Burroughs-Wellcome. The prescription of five daily doses of 100 mg AZT was written in 1992 for the HIV-positive but then AIDS-free Cesar Schmitz (§ 4.3d). In contrast to the biochemical manufacturer, the medical manufacturer does not warn about the toxic effects of AZT.

test in 1984 (Duesberg 1995c, 1996a). Moreover, the mortality of hemophiliacs was steadily decreasing since the 1970s until 1987 – despite the presence of HIV (Duesberg 1995c)! Thus the only new risk of mortality, in and after 1987, was not HIV, but AZT. Darby *et al* even acknowledged “treatment, by prophylaxis against *P. carinii* pneumonia or with zidovudine (AZT), has been widespread for HIV-infected haemophiliacs since about 1989 (more accurately since 1987)”. The editor of *Nature* also pointed out that, “Darby *et al* failed to provide full details of the drug regimen followed”

(Maddox 1995). The AZT-mortality hypothesis would of course also explain why the new hemophilia mortality was independent of the severity of the hemophilia, as Darby *et al* observed. Nevertheless *Nature*, did not accept an alternative interpretation, specifically not from “Those who have made the running in the long controversy over HIV in AIDS, Dr Peter Duesberg of Berkeley, California, in particular . . .” (Maddox 1995). But, the *Lancet* accepted a response, which proposed that AZT treatments were the probable cause of the sudden increase in mortality of hemophiliacs (Duesberg 1995d).

According to researchers from the NIH, AZT also increased the mortality of US hemophiliacs 2-7 times and their AIDS risk 4-5 times compared to untreated controls (Goedert *et al* 1994; Duesberg 1995c). The medical literature describes many more examples of AIDS defining, other diseases and deaths that developed in HIV-positive asymptomatic people or in AIDS patients treated with anti-HIV drugs, which were not observed in untreated controls; some of these are summarized in table 6.

The case of Cesar Schmitz, married to an HIV-free wife and father of an HIV-free healthy child in Miami, FL, is an example of AZT-mediated mortality that did not appear in the medical literature (Duesberg 1996b). But his wife Teresa has recorded his case in sufficient detail for inclusion in this article. In March 1992, an asymptomatic Schmitz was found to be HIV-positive at a medical check-up and pressured by his doctor to start AIDS prophylaxis by AZT (figure 4b). Immediately after initiation of AZT treatment, Schmitz developed “nausea, diarrhea and weight loss”. In 1994 he decided, “against his doctors will,” to discontinue AZT medication, and “All of a sudden, like magic, no more symptoms” (Duesberg 1996b). But, in 1998 Schmitz developed lymphoma, which is a typical, late “side effect” that appears in 46% of patients 36 months after initiation of AZT therapy (Pluda *et al* 1990). In view of this and pressure from his doctors Schmitz started AZT therapy again. Within months he was “paralyzed”, suffered from “unbearable cramps” and became incontinent (probably from mitochondrial dysfunction, see table 6), which his doctor explained as “side effects of one of the drugs he was taking”. And in October 1998 Schmitz passed away (T Schmitz, personal communication).

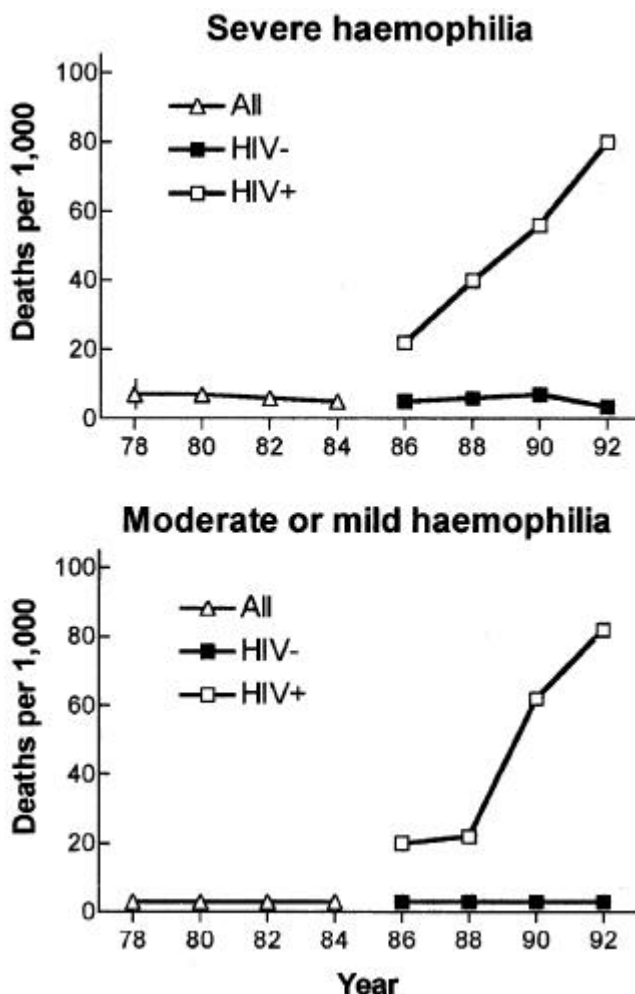


Figure 5. Mortality of HIV antibody-positive (+) and negative (-) British hemophiliacs after the introduction of the DNA chain-terminator AZT, alias zidovudine, in 1987 as anti-HIV therapy. The data are from a study by Darby *et al* (1995) that considered HIV to be the cause of the sudden increase of hemophilia mortality after 1987. Instead of HIV, which has been diagnosed in hemophiliacs since 1984, the introduction of AZT as anti-HIV drug explains the sudden rise in mortality in 1987, and also the increase in mortality irrespective of the degree of hemophilia (see § 4.3d).

(ii) *Diseases and death in HIV-free humans and animals treated with anti-HIV drugs:* Table 7 lists rare studies reporting AIDS-defining and other diseases in HIV-free humans and animals treated with anti-HIV drugs. Since all HIV-positive, pregnant mothers are now treated with AZT during the last 6 months of their pregnancy to reduce the natural transmission of HIV to 25 to 50% of their babies, there are now over 50% HIV-free babies born to these mothers who have all been treated with AZT (Connor

et al 1994; The Durban Declaration 2000). Table 7 lists two rare publications that describe the diseases of these HIV-free, AZT-treated babies, such as fevers, pneumonia, anemia, and mitochondrial dysfunction. In addition the table lists studies, which have observed numerous diseases and deaths in HIV-free animals treated with anti-HIV drugs. All of these animal studies were published after the drugs had been licensed for humans (perhaps because licenses once issued are almost impossible to withdraw) and only in specialty journals. Therefore, these results are not known and not discussed in the popular and medical AIDS literature.

The anti-HIV treatment-specific diseases and death summarized in tables 6 and 7 directly support the hypothesis that anti-HIV drugs are at least necessary in the presence of HIV, and are sufficient in its absence to cause most AIDS defining-diseases, other drug-specific diseases, and death. Since about 450,000 US citizens are currently on DNA chain-terminators and protease inhibitors as prophylaxis against, or therapy of AIDS (see above), these drugs alone could have been sufficient to generate all of the 43,158 new AIDS patients reported in the US in 2001 (Centers for Disease Control and Prevention 2001).

4.3e *The AIDS treatment dilemma: Do anti-HIV drugs, that cause AIDS defining and other diseases, delay progression to AIDS and reduce mortality?:* Despite the inevitable toxicity of anti-HIV drugs, the over 5000 signatories of the Durban Declaration assert that, “drugs that block HIV replication in the test tube also” (i) “delay progression to AIDS”, and (ii) “have reduced AIDS mortality by more than 80%”. However, the authors of the Declaration have not provided a reference for controlled studies in support of their assertion. But, they do acknowledge “That it is crucial to develop new antiviral drugs that ... have fewer side effects” (The Durban Declaration 2000). Since many doctors share the views of the Declaration, we investigate here the evidence for these claims.

(i) *Controlled studies investigating the ability of anti-HIV drugs to “reduce mortality” and “delay progression to AIDS”:* The licensing study of AZT, performed in 1987 by the NIH in collaboration with the drug’s manufacturer Burroughs Wellcome in the US, is the primary placebo-controlled study set-up to test the ability of AZT to reduce the mortality of AIDS (Fischl *et al* 1987; Richman *et al* 1987). The study showed that, after 4 months on AZT, 1 out of 145 AIDS patients died, whereas 19 out of 139 died in the placebo group. The study interpreted this result as evidence for reduced mortality by AZT. However, this interpretation failed to consider that among the 4-month-survivors of AZT, 30 could only be kept alive with multiple blood transfusions because their red cells had been depleted by AZT below survivable levels

(Fischl *et al* 1987; Duesberg 1992). Thus, without life-saving transfusions 30 more AZT-recipients would have died from anemia. In addition many AZT recipients had developed life-threatening bone marrow suppression, neutropenia, macrocytosis, headaches, insomnia and myalgia, that augured poorly for their future survival (Richman *et al* 1987). Indeed, the low mortality of 1/145 reported for the first 4 months on AZT, could not be maintained in a follow-up study, which found the “survival benefits” of AZT rapidly declining after the original 4 months period. By 21 months, 42% of the original AZT group had died and 35% of the control group, which by then had also received AZT for 12 months on a “compassionate” basis (Fischl *et al* 1989). Thus the placebo-controlled, licensing study did not prove that AZT “reduces AIDS mortality by more than 80%” compared to the untreated control.

The ability of AZT to “delay the progression to AIDS” was investigated in 1994 by the largest, placebo-controlled study of its kind, the British-French Concorde study (Seligmann *et al* 1994). This study investigated 1749 HIV-positive, mostly male homosexual subjects divided into untreated and AZT-treated subgroups for the onset of AIDS and death. The Concorde study found in 1994 that AZT is unable to prevent AIDS and increases the mortality of recipients by 25%. In view of this it concluded, “The results of Concorde do not encourage the early use of zidovudine (AZT) in symptom-free HIV-infected adults.” (Seligmann *et al* 1994). Thus there is no controlled evidence that anti-HIV drugs “reduce the mortality of” or “delay progression to AIDS”.

(ii) *Uncontrolled studies investigating the mortality of HIV-positives on HIV drugs:* Despite the discouraging results of these controlled studies, AIDS researchers now credit the more recently developed anti-HIV drug cocktails for a “declining morbidity and AIDS (Palella *et al.* 1998). However, the evidence for “declining morbidity and mortality” is only based on uncontrolled survey studies that investigated how long HIV-positive, clinically healthy subjects, but mostly from AIDS risk groups, survived on various anti-HIV drugs. The largest and most influential of these surveys was conducted by Palella *et al* (1998) who investigated in 1998 1255 anti-HIV drug-treated “patients, each of which had at least one CD4+ count below 100” from nine clinics in the US. However, all of these “patients” were “nonhospitalized”, AIDS-free subjects. “Patients with a diagnosis of cytomegalovirus retinitis or *M. avium* complex disease before study entry or during the first 30 days of follow-up and patients with active *P. carinii* pneumonia at the beginning of follow-up were excluded.” A similar survey investigated in 2001 1219 anti-HIV drug-treated Canadian HIV-positives with less than 200 CD4+ cells, of which 87% were AIDS-free (Hogg *et al* 2001). Neither of these studies mentions drug-free controls. On

this basis the Palella-study found that the mortality of initially asymptomatic, HIV-positive people, which are treated with new anti-HIV drug cocktails, is 8.8% (“8.8 per 100 person-years”) and the Hogg-study found it is 6.7%.

But, in the absence of untreated control groups, the effects of the new anti-HIV drugs on the morbidity and mortality of HIV-positive recipients can not be determined scientifically from the results of these surveys. However, the average annual AIDS mortality of all HIV-positives on this planet [including the minority that is on anti-HIV drugs (The Durban Declaration 2000)] can be estimated for 2000, the year that falls in between the two surveys, based on data provided by the WHO and the Durban Declaration: The WHO and the Declaration report in 2000 34.3 million “living with HIV”, and the WHO reports 471,451 AIDS cases for 2000 (World Health Organization 2001b) (obtained by subtracting the WHO’s cumulative total of 1999 from that of 2000, see also table 4). Thus, even if we assume that all AIDS cases were fatal in 2000, the resulting global mortality rate of HIV-positives would only be 1.4% – and thus 4 to 6 times lower than the 6.7–8.8% mortality rate of HIV-positives treated with anti-HIV drugs in the US and Canada. Therefore, the claims that anti-HIV drugs reduce the mortality of, and delay progression to AIDS are at odds with the AIDS facts reported by the Durban Declaration and the WHO. Contrary to these claims, the controlled trials and uncontrolled surveys listed above prove that anti-HIV drugs (possibly in conjunction with recreational drugs) increase the mortality of HIV positives 4- to 6-fold. It would appear that anti-HIV drugs are prescriptions for, rather than treatments of AIDS.

(iii) *Skepticism about anti-HIV drugs in the medical establishment*: Even in the absence of scientifically controlled studies proving the toxicity of the new anti-HIV drugs, many AIDS doctors and researchers have warned of the numerous toxic effects of these drugs – even the Durban Declaration calls for drugs which “have fewer side effects”. For example, HIV co-discoverer Jay Levy wrote in the *Lancet*, “Caution: should we be treating HIV infection early? . . . No cancer patient takes three or four chemotherapeutic drugs for a lifetime. What is overlooked . . . is that these drugs can be toxic and can be directly detrimental to a natural immune response to HIV.” (Levy 1998). And retrovirus researcher Etienne De Harven describes the treatment of AIDS with DNA chain-terminators as a “so-called therapy worse than the disease itself!” (de Harven 1999).

Because of such concerns about the toxicity of anti-HIV drugs AIDS doctors have recently introduced “structured treatment interruption” (Lori *et al* 2000) or “drug holidays” (Christensen 2000), to allow the patients to recover from the toxic effects of the DNA chain-terminators, such as AZT, ddI, and d4T, and of the protease

inhibitors prescribed to kill HIV. In the words of Kendall Smith from the New York Hospital-Cornell Medical Center, “Right now, the disease is life-threatening (he did not say HIV), on one hand, and the drugs that we have so far have life-threatening toxicities, on the other hand. It puts us between a rock and a hard place.” (Christensen 2000).

In view of this the US government has appointed a panel of AIDS scientists to review the toxic effects of antiviral medications and issued recommendations to restrict prescriptions of anti-HIV drugs that were published by the *New York Times* (Altman 2001b):

“Altering a long-held policy, federal health officials are now recommending that treatment for the AIDS virus be delayed as long as possible for people without symptoms because of increased concerns over toxic effects of the therapies. . . . More recently, concern has grown over nerve damage, weakened bones, unusual accumulations of fat in the neck and abdomen, diabetes and a number of other serious side effects of therapy. Many people have developed dangerously high levels of cholesterol and other lipids in the blood, raising concern that H.I.V.-infected people might face another epidemic—of heart disease. . . . Dr Fauci, who is co-chairman of the panel, said in an interview, ‘We are adopting a significantly more conservative recommendation profile’”. (According to the panel), “Much remains to be learned about how best to treat H.I.V.-infected individuals”.

However, it is hard to understand, why it should have taken AIDS researchers 14 years since the introduction of DNA chain-terminators as anti-HIV drugs (Kolata 1987) to make these observations and issue warnings about the “side effects” of these drugs.

In April 2001, the FDA followed up on these concerns by “ordering drug makers to tone down their upbeat ads for AIDS medications, calling them ‘misleading’ . . . because they imply greater efficacy than demonstrated by substantial evidence, or minimize the risks associated with HIV drugs” (Russell 2001) – again 14 years after approving these drugs for currently 450,000 American recipients.

Many other independent observers have since commented on the “U-turn” of AIDS researchers (Day 2000) from “Hit HIV early and hard” in 1995 (Ho 1995) to reducing, skipping and delaying treatments, and even recalling some anti-HIV drugs (Altman 2001c; Associated Press 2001). Even conservative, nonscientific media such as *Mothering* magazine now warn expecting mothers not to use anti-viral drugs during pregnancy with heart-breaking accounts of the clinical consequences for the babies, and of the bewildering pressures by the medical and even legal authorities on mothers to enforce compliance with prescriptions of DNA chain-terminators for their babies (Farber 1998; Gerhard 2001; Hodgkinson 2001).

But despite a preponderance of evidence against anti-HIV drugs, these drugs have not been restricted or banned by any country except South Africa (Cherry 2000).

4.4 Prediction 3: AIDS diseases and epidemics are not self-limiting via immunity

The drug hypothesis predicts that AIDS is not self-limiting via immunity. Indeed twenty years into the AIDS epidemics, there is no evidence of individual immunity against AIDS, nor is there any evidence that any of the AIDS epidemics is self-limiting (World Health Organization 1999; The Durban Declaration 2000; Centers for Disease Control and Prevention 2001), (see figure 1a, c). According to the Durban Declaration, “there is no end in sight”. Indeed the chronologies of the current AIDS epidemics conform exactly to the time courses of epidemics of chemical diseases that are not self-limiting, such as the American drug epidemic shown in figure 2, and the epidemics of smoking and of subsequent lung cancers in England, shown in figure 3b.

4.5 Prediction 4: No AIDS in the absence of anti-viral and recreational drugs, despite HIV

To test this prediction, HIV antibody-positive people, who are not using drugs, must be identified who survive the average hypothetical latent period from HIV to AIDS of 5–10 years (§ 3, table 4). The following examples meet this prediction.

In 2002 the *San Francisco Chronicle* described a small group of drug-free and AIDS-free long-term survivors of HIV. Among them is a healthy artist who is HIV-positive for 23 years (based on frozen blood samples) and was “chastised by his doctors when he refused to start taking medication” (Hendrix 2002). Further, a 1-year old HIV-positive, AZT-treated baby girl with severe muscle pain, insomnia, nausea and failure to grow was taken off AZT treatments in 1992 based on our hypothesis; as a result the baby immediately recovered (Duesberg 1996b). Now, at the age of 11, she is a completely normal, healthy kid, and a leading player in her school’s soccer team (Sheryl and Steve Nagel, personal communication). In addition, *People* magazine just described a healthy woman who is HIV-positive for an estimated 15 years, and “needs no medication”. The woman has since founded a support group, termed Center for Positive Connections, for HIV-positive heterosexuals in Miami (Chekalos and Rosza 2002). In Los Angeles, Christine Maggiore is HIV-positive since 1992, has given birth to two very healthy children, ages 1 and 5, and has never taken anti-HIV drugs. Maggiore, a former HIV-AIDS counselor, has since also founded a support group, Alive & Well, and has written a

book, *What if everything you thought you knew about AIDS was wrong?*, to instruct HIV-positives not to use anti-HIV drugs (Maggiore 2000). An appendix of the book features letters from 34 Maggiore-graduates, all living over 10 years with HIV but without anti-HIV drugs, or after having discontinued such drugs.

Even HIV-AIDS researchers have inadvertently confirmed our prediction of no AIDS in drug-free HIV-positives. For example, David Ho, signatory of the Durban Declaration, points out that in a group of “long-term survivors” of HIV studied in his lab, “none had received antiretroviral therapy” (Cao *et al* 1995). In a parallel publication, Pantaleo *et al* studying a group of long-term “non-progressors” of HIV have made the same observation (Pantaleo *et al* 1995). Ho *et al* recently attributed long-term survival to some special human proteins, termed “defensins” (Zhang *et al* 2002), but acknowledged personally that all long-term survivors had again abstained from anti-HIV therapies (David Ho, personal communication). One wonders why any humans would ever get sick from HIV, if the human genome encodes HIV defensins! Munoz reported that none of the long-term survivors of the largest, federally funded study of AIDS risk factors of homosexual men, the MACS study, had used AZT (Munoz 1995). Fahey *et al* observed that among HIV-positive male homosexuals with less than 200 T-cells per μl , “45% of the group who were AIDS-free $> \text{or} = 3$ years after CD4^+ cells fell below $200 \times 10^6/\text{l}$ had not used these (anti-HIV) treatments.” (Hoover *et al* 1995). According to a university magazine, AIDS researchers Abrams and Levy from the University of California at San Francisco have lectured in 1998 on drug-free long-term survivors of HIV to their medical students (Tanaka 1996; Duesberg and Rasnick 1998). Levy also published in 1998 in the *Lancet*, that “effective antiviral immune response is characteristic of long-term survivors who have been infected for over 20 years, have no symptoms, and have not been on any therapy” (Levy 1998). In 1999, Pitcher *et al* also described a group of 9 “long-term non-progressors (with) untreated HIV-1 infection for 7–15 years”, compared to controls with a “decline of (T cells) with antiretroviral therapy” (Pitcher *et al* 1999). An Australian research team described a group of untreated HIV-positives who were infected by blood transfusions but did not develop AIDS 10 years later (Learmont *et al* 1992). Further, Migueles *et al* (2000) reported that none of 13 long-term survivors had received “antiretroviral therapy”. Carr *et al* (2001) observed even recovery from fatal hypertension, liver failure and mitochondrial dysfunction after discontinuation of antiviral drugs that had been prescribed to a previously healthy HIV-positive man. Thus HIV-AIDS researchers confirm our prediction that HIV-positives, who do not use drugs, do not develop AIDS or may even recover from it.

In an effort to obtain independent proof that abstaining from anti-HIV drugs and recreational drugs is sufficient to survive HIV-infection or even to recover from AIDS, one of us, CK, in 1985 initiated a study of AIDS patients from Kiel, Germany, who have volunteered to abstain from anti-HIV treatments. Remarkably, only 8% (3 of 36) of the patients not treated with anti-HIV drugs have died since their HIV antibodies were first detected, two of them 16 years and one 10 years after their first diagnosis of antibodies against HIV (table 8). Most have recovered from their initial AIDS-indicator symptoms. By contrast, 63% of all German AIDS patients (11,700

out of 18,700) of which most were treated since 1987 with anti-HIV drugs have died (Robert Koch Institut 2000). Thus our relatively small sample supports the hypothesis that without anti-HIV drugs and/or recreational drugs HIV fails to cause AIDS. Indeed without drugs AIDS patients recover, despite the presence of HIV.

4.6 *In sum, the chemical AIDS-hypothesis explains the AIDS facts, and resolves all paradoxes of the HIV-AIDS hypothesis*

Our review shows that the chemical-AIDS hypothesis explains all AIDS facts: the non-random distribution of

Table 8. Long-term HIV survivors not treated with antiviral drugs and abstaining from illicit, psychoactive drugs from the Kiel-Koehnlein study begun in 1985.

Case	Date HIV+	Age	Sex	Clinic	CD4	Illicit drugs	Treatment	Death
1	1985	52	m-homo	Herpes zoster	256	?	None	
2	1985	45	f	Asymptomatic			None	
3	1985–2001	43	m	PCP, TB	4	iv	Antibiotics	Heart failure
4	1985–2001	42	m-homo	Kaposi, PCP		Nitrites	None	Kaposi
5	1985	35	m	Psoriasis			None	
6	1985	38	f	<i>Salmonella sepsis</i>	28		Antibiotics	
7	1985	31	m	Hemophilia, lymphadenopathy	low			
8	1985	30	m	Hemophilia	325		None	
9	1986	17	f (black)	Asymptomatic	450		None	
10	1986	31	m	Asymptomatic				
11	1988	32	f	Asymptomatic			None	
12	1989	49	f	Candida, TB	28	iv	Antibiotics	
13	1990	34	m-homo	Coli-Meningitis, Hydrocephalus	85	Nitrites	Shunt	
14	1991	6	m	Pneumonia			Antibiotics	
15	1991	31	f	Asymptomatic			None	
16	1991–2001	36	f	Toxoplasmosis			1993–94 AZT, antibiotics	Toxoplasmosis
17	1992	33	f	Asymptomatic	460		None	
18	1993	7	f	Asymptomatic			None	
19	1996	52	m-homo	Facial paresis	540	Nitrites	None	
20	1996	39	m-homo	Asymptomatic	485	Nitrites	None	
21	1996	38	m	Pneumonia, Sinusitis	53		Antibiotics	
22	1997	44	m-homo	Colitis		Nitrites	None	
23	1997	37	m-homo	Asymptomatic	223	Nitrites	None	
24	1997	37	m-homo	Thrombocytopenia	700	Nitrites	Cortisone	
25	1997	34	m-homo	Asymptomatic	220	Nitrites	None	
26	1997	33	m-homo	Lymphadenopathy			None	
27	1997	31	m-homo	Thrombocytopenia	357	Nitrites	Cortisone	
28	1997	3	m	Pneumonia			Antibiotics	
29	1998	31	m-homo	Diarrhea		Nitrites	None	
30	1998	40	m-homo	Condyloma, Candida	107	Nitrites	None	
31	1998	39	m-homo	Diarrhea	187	Nitrites	None	
32	1998	27	m-homo	TB of the bowel	18		Antibiotics	
33	2000	35	m-homo	Asymptomatic			None	
34	2000	31	m-homo	Lymphadenopathy, EBV			None	
35	2000	30	m-homo	Asymptomatic	428		None	
36	2000	27	f	Lymphadenopathy, EBV	107		None	

m, Male; f, female; PCP, *Pneumocystis carinii* pneumonia; EBV, Epstein-Barr virus; AZT, azidothymidine; TB, tuberculosis. Evidence for illicit drug use is self reported; iv, intravenous drug use.

drug-AIDS in the US and Europe, the risk-group-specific AIDS diseases in the US and Europe as consequences of risk-group-specific drugs, the random distribution of malnutrition-AIDS in Africa, the non-contagiousness of chemical AIDS, the absence of natural immunity against chemical AIDS, the lifestyle-dependent onset of AIDS diseases – unrelated to, but typically long after infection by HIV, and the time courses of the AIDS epidemics of the US and Europe as consequences of the drug epidemics.

In addition chemical AIDS proves that HIV is not necessary for even one AIDS-defining disease, because (i) drugs and malnutrition cause drug- and malnutrition-specific AIDS diseases regardless of the presence of HIV, because (ii) in HIV antibody-positives and negatives the risk of developing AIDS is proportional to the degree or lifetime dosage of drug use, and (iii) because all AIDS diseases have been diagnosed in HIV-free AIDS risk groups by AIDS researchers (Duesberg 1993d) and also long before the AIDS era (Stedman's Medical Dictionary 1982). Thus HIV meets all criteria of a harmless passenger virus, laid out in table 4 and described previously (Duesberg 1994; Duesberg and Rasnick 1998). In this way our proposal resolves the fundamental paradox of the HIV-AIDS hypothesis: the paradox that a latent, non-cytopathic and immunologically neutralized retrovirus, that is only present in less than 1 out of 500 susceptible T-cells and rarely expressed in a few of those, would cause a plethora of fatal diseases in sexually active, young men and women. And, that the plethora of diseases attributed to this virus would not show up for 5–10 years after infection.

The chemical AIDS hypothesis could be readily refuted by any of the following experiments:

- (i) Demonstrate that in two matched groups, differing only with regard to HIV infection, HIV-positives develop AIDS but HIV-negatives do not (above the low, long-established risk of AIDS defining diseases in the general population). HIV antibody-positive and negative recruits from the US Army, which tests routinely for HIV, would be ideal for this experiment since their health, lifestyles and age are closely matched.
- (ii) Demonstrate that in two matched groups of intravenous drug users, differing only in the presence of HIV, only the HIV-positives develop AIDS diseases.
- (iii) Demonstrate that in two matched groups of HIV-positive humans, differing only in the addiction to recreational drugs, both groups have the same incidence of AIDS-defining diseases.
- (iv) Demonstrate that in two matched groups of HIV-free humans or animals, differing only with regard to the addiction to or treatment with recreational drugs, neither group would develop AIDS defining diseases over time.
- (v) Demonstrate that in two matched groups of HIV-

positives, differing only in the treatment with anti-HIV drugs, the untreated group develops AIDS long before the treated group.

(vi) Demonstrate that in two matched groups of pregnant, HIV-positive mothers, differing only in the now standard treatment with AZT during the last two trimesters, those treated with AZT are free of abortions and deliver healthy babies, but those who are not treated either abort spontaneously or deliver babies with AIDS.

(vii) Demonstrate that in two groups of HIV-positive hemophiliacs matched for age and lifetime dosage of factor VIII, differing only in anti-HIV treatments, those who are untreated have a higher mortality and a higher AIDS risk than treated controls.

Although the controlled studies proposed here follow classical, scientific standards, they are not available in the huge AIDS literature. This is surprising in view of the many AIDS advocacy groups or "activists" reviewing AIDS research for flaws and for new clues. The lack of adequately controlled studies of the long-term effects of recreational drugs and anti-HIV drugs in animals is particularly surprising, because all of these drugs and research funds for AIDS are abundant. Yet despite the scientific intolerance of current AIDS science for alternative hypotheses (Weiss and Jaffe 1990; Cohen 1994; O'Brien and Goedert 1996), the pathogenicity of most of the chemicals proposed here to cause AIDS – illicit drugs, antiviral drugs, and malnutrition – has *de facto* already been proved – even by HIV-AIDS researchers, despite their efforts to the contrary [see above, tables 6 and 7 and Duesberg and Rasnick (1998)].

Suppose the chemical-AIDS hypothesis were confirmed and accepted: AIDS would be entirely preventable by banning anti-HIV drugs, by publicizing that recreational drugs cause AIDS and by adequate nutrition. Moreover, many AIDS patients could still be saved from fatal damage by drug intoxication, if their AIDS-defining diseases were treated with time-proven, disease-specific medications. Such testable predictions are the hallmarks of a good hypothesis.

So, why do current AIDS researchers not investigate and not even consider the role of chemicals in AIDS or study other non-HIV-AIDS theories to solve the AIDS dilemma? The following is an attempt to answer this question.

5. Epilogue

5.1 Why is AIDS research not free to investigate non-HIV hypotheses?

The probable answer to the question, why HIV-AIDS researchers do not study or fund non-HIV-AIDS theories, lays in the structure of the large, government-sponsored

research programs that dominate academic research since World War II (Duesberg 1996b). Such programs favour individual investigators who contribute to the establishment a maximum of data and a minimum of controversy. However, if individual researchers move into new directions, that threaten the scientific and commercial investments of the establishment, the establishment can impose various sanctions via the “peer review system”. The most powerful of these are denial of funding and of publication.

The peer review system derives its power from the little known practice of governments to deputize their authority to distribute funds for research to committees of “experts”. These experts are academic researchers distinguished by outstanding contributions to the current establishment. They alone review the merits of research applications from their peers, and they have the right to elect each other to review committees. Outwardly, this “peer review system” appears to the unsuspecting government and taxpayer as the equivalent of a jury system – free of all conflicts of interest. But, in view of the many professional and commercial investments in and benefits from their expertise, and even of the rewards from their universities and institutions for the corresponding overheads and partnerships – all legal in the US since president Reagan – “peer reviewers” do not fund applications that challenge their own interests (Duesberg 1996b; Lang 1998; Zuger 2001). Since “peer review” is protected by anonymity, does not allow the applicant personal representation or an independent representative, nor a say or even a veto in the selection of the “jury”, and does not allow an appeal, its powers to defend the orthodoxy are unlimited. The corporate equivalent of academia’s “peer review system” would be to give General Motors and Ford the authority to review and veto all innovations by less established carmakers competing for the consumer.

Even the professional journals and the science writers of the public media comply with the interests of government-funded majorities because they depend on their monthly “scientific breakthroughs”, the lucrative advertisements from their companies, and the opinion of their subscribers. For example, an early precursor of this article was written in response to an open invitation from a pharmacology-journal over 3 years ago. But, after considerable pressure on the journal from anonymous “AIDS experts”, the editor requested a reduced article, which was neither accepted nor rejected. Instead, the editor simply dropped all further correspondence. Subsequently, the editor of a prestigious German-based science journal invited another precursor of this article 2 years ago, which received two favourable reviews in short order. But before the manuscript could be revised, the editor informed us that the publisher was concerned about losing subscribers if our paper were published and ceased all

further correspondence. It is this passive resistance that can grind down even the most determined truth seeker.

However, the mere potential to resolve the agony of AIDS by alternative hypotheses, such as ours, should be sufficient reason to replace the medieval “peer review system” by a modern jury system without conflicts of interest and with rights for representation and appeals of the applicant. If the current, unproductive AIDS establishment objects, because AIDS-science is too complex to be understood by non-HIV-AIDS scientists, funding should be withheld until the AIDS establishment finds ways to explain the complexity and merits of its expertise to other scientists.

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