



## The AIDS dilemma: drug diseases blamed on a passenger virus

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### Abstract

Almost two decades of unprecedented efforts in research costing US taxpayers over \$50 billion have failed to defeat Acquired Immune Deficiency Syndrome (AIDS) and have failed to explain the chronology and epidemiology of AIDS in America and Europe. The failure to cure AIDS is so complete that the largest American AIDS foundation is even exploiting it for fundraising: 'Latest AIDS statistics – 0,000,000 cured. Support a cure, support AMFAR.' The scientific basis of all these unsuccessful efforts has been the hypothesis that AIDS is caused by a sexually transmitted virus, termed Human immunodeficiency virus (HIV), and that this viral immunodeficiency manifests in 30 previously known microbial and non-microbial AIDS diseases.

In order to develop a hypothesis that explains AIDS we have considered ten relevant facts that American and European AIDS patients have, and do not have, in common: (1) AIDS is not contagious. For example, not even one health care worker has contracted AIDS from over 800,000 AIDS patients in America and Europe. (2) AIDS is highly non-random with regard to sex (86% male); sexual persuasion (over 60% homosexual); and age (85% are 25–49 years old). (3) From its beginning in 1980, the AIDS epidemic progressed non-exponentially, just like lifestyle diseases. (4) The epidemic is fragmented into distinct subepidemics with exclusive AIDS-defining diseases. For example, only homosexual males have Kaposi's sarcoma. (5) Patients do not have any one of 30 AIDS-defining diseases, nor even immunodeficiency, in common. For example, Kaposi's sarcoma, dementia, and weight loss may occur without immunodeficiency. Thus, there is no AIDS-specific disease. (6) AIDS patients have antibody against HIV in common only by definition—not by natural coincidence. AIDS-defining diseases of HIV-free patients are called by their old names. (7) Recreational drug use is a common denominator for over 95% of all American and European AIDS patients, including male homosexuals. (8) Lifetime prescriptions of inevitably toxic anti-HIV drugs, such as the DNA chain-terminator AZT, are another common denominator of AIDS patients. (9) HIV proves to be an ideal surrogate marker for recreational and anti-HIV drug use. Since the virus is very rare (< 0.3%) in the US/European population and very hard to transmit sexually, only those who inject street drugs or have over 1,000 typically drug-mediated sexual contacts are likely to become positive. (10) The huge AIDS literature cannot offer even one statistically significant group of drug-free AIDS patients from America and Europe.

In view of this, we propose that the long-term consumption of recreational drugs (such as cocaine, heroin, nitrite inhalants, and amphetamines) and prescriptions of DNA chain-terminating and other anti-HIV drugs, cause all AIDS diseases in America and Europe that exceed their long-established, national backgrounds, i.e. >95%. Chemically distinct drugs cause distinct AIDS-defining diseases; for example, nitrite inhalants cause Kaposi's sarcoma, cocaine causes weight loss, and AZT causes immunodeficiency, lymphoma, muscle atrophy, and dementia. The drug hypothesis predicts that AIDS: (1) is non-contagious; (2) is non-random, because 85% of AIDS causing drugs are used by males, particularly sexually active homosexuals between 25 and 49 years of age, and (3) would follow the drug epidemics chronologically. Indeed, AIDS has increased from negligible numbers in the early 1980s to about 80,000 annual cases in the early '90s and has since declined to about 50,000 cases (US figures). In the same period, recreational drug users have increased from negligible numbers to millions by the late 1980s, and have since decreased possibly twofold. However, AIDS has declined less because since 1987 increasing numbers of mostly healthy, HIV-positive people, currently about 200,000, use anti-HIV drugs that cause AIDS and other diseases. At least 64 scientific studies, government legislation, and non-scientific reports document that recreational drugs cause AIDS and other diseases. Likewise, the AIDS literature, the drug manufacturers, and non-scientific reports confirm that anti-HIV drugs cause AIDS and other diseases in humans and animals. In sum, the AIDS dilemma could be solved by banning anti-HIV drugs, and by pointing out that drugs cause AIDS modeled on the successful anti smoking campaign.

*An unflinching determination to take the whole evidence into account is the only method of preservation against the fluctuating extremes of fashionable*

*opinion.* Alfred North Whitehead (1861–1947) (Whitehead, 1967).

## Introduction

In the US, the epidemic of the Acquired Immune Deficiency Syndrome (AIDS) has risen from negligible numbers in the early 1980s to about 80,000 annual cases in the early 1990s and has since declined to about 50,000 cases (Centers for Disease Control and Prevention, 1997). In the same period, the European epidemic has reached about 20,000 annual cases in the '90s, and is now also declining slowly (World Health Organization, 1995b; AIDS-Zentrum im Robert Koch-Institut, 1997). To date, the epidemic has generated 650,000 American and over 150,000 European AIDS patients (Figure 1A, C) (World Health Organization, 1995b; AIDS-Zentrum im Robert Koch-Institut, 1997; Centers for Disease Control and Prevention, 1997; Fiala & Lingsens, 1997).

Since 1984, all efforts in research, treatment and prevention of AIDS are based on the hypothesis that AIDS is an infectious immunodeficiency caused by a sexually transmitted virus, termed human immunodeficiency virus (HIV) or 'AIDS virus' (Altman, 1984). According to the Centers for Disease Control (CDC) in Atlanta, the viral immunodeficiency manifests in 30 previously known microbial and non-microbial diseases (Centers for Disease Control and Prevention, 1992). However, 15 years after the discovery of HIV (Barre-Sinoussi et al., 1983), leading scientists and policy makers cannot demonstrate that their efforts against the virus have saved a single life. Despite a cost of over 50 billion dollars to US taxpayers, there is no vaccine and no effective anti-AIDS drug (Gutknecht, 1995; Duesberg, 1996d). The failure to defeat AIDS is so complete that it is now even exploited by America's largest private AIDS research foundation for fundraising:

'Latest AIDS statistics – 0,000,000 cured. Support a cure, support AMFAR.  
Call 1-800-39AMFAR.'

In reality, even the zero-cure admission is a massive understatement of the non-achievements of the HIV hypothesis. Since HIV has become the official cause of AIDS, hundreds of thousands of HIV antibody-positive Americans and Europeans with and without AIDS (!) have been put on lifetime prescriptions of inevitably toxic DNA chain-terminators and protease inhibitors prescribed as anti-HIV drugs (Duesberg, 1992a; Duesberg, 1996d; Hodgkinson, 1996) (see below). Moreover, in the name of the HIV-AIDS hypothesis, recreational drug use is not only disregarded as a cause of AIDS, it is instead explicitly excused as

a cause of any disease (Kaslow et al., 1989; Ascher et al., 1993; Schechter et al., 1993c), and even promoted with slogans like 'heroin is a blessedly untoxic drug' (Cohen, 1994a). The heroin slogan was published by *Science* in the same year, 1994, in which 3,522 Americans died and 64,013 were hospitalized because of heroin toxicity (see below, Table 4). Proponents of the HIV hypothesis blame the high morbidity and mortality of drug addicts only on HIV.

In contrast to AIDS, all infectious epidemics of the past, such as polio, cholera, tuberculosis, small pox, and syphilis, have long been brought under control, or even eliminated, at a fraction of the cost of AIDS, and with technology that was far less sophisticated than what is available now. As a result of these scientific triumphs of the past, only less than one percent of us now die from infectious diseases (Cairns, 1978). In view of this, the continued failures of the war on AIDS call into question the hypothesis that AIDS is an infectious disease.

In the meantime, the multibillion dollar AIDS research effort also proved to be disappointing. Despite unprecedented efforts by thousands of virologists and hundreds of thousands of medical scientists, there are numerous unanswered questions about the AIDS epidemic in America and Europe:

1. Why would antibodies against HIV (a positive HIV test), which are so effective that leading AIDS researchers cannot detect HIV in most AIDS patients (Gallo, 1991; Weiss, 1991; Cohen, 1993), not protect against AIDS?
2. Why have doctors and nurses never caught AIDS from over 800,000 American and European AIDS cases – particularly in the absence of a HIV vaccine?
3. Why are 9 out of 10 AIDS patients males?
4. Why are about two-thirds male homosexuals?
5. Why are one-third intravenous drug users?
6. Why are most AIDS patients 25-49 years old, and why don't teenagers get AIDS?
7. Why is AIDS new, although HIV is long-established in the US and Europe?
8. Why would a new, sexually transmitted disease not have exploded in the millions of heterosexually active Americans and Europeans – just as syphilis once did in medieval Europe?
9. Why did HIV-positive American hemophiliacs live over twice as long in 1987 as they did in the pre-AIDS era, and why has their mortality increased ten-fold after the introduction of the anti-HIV drug AZT?

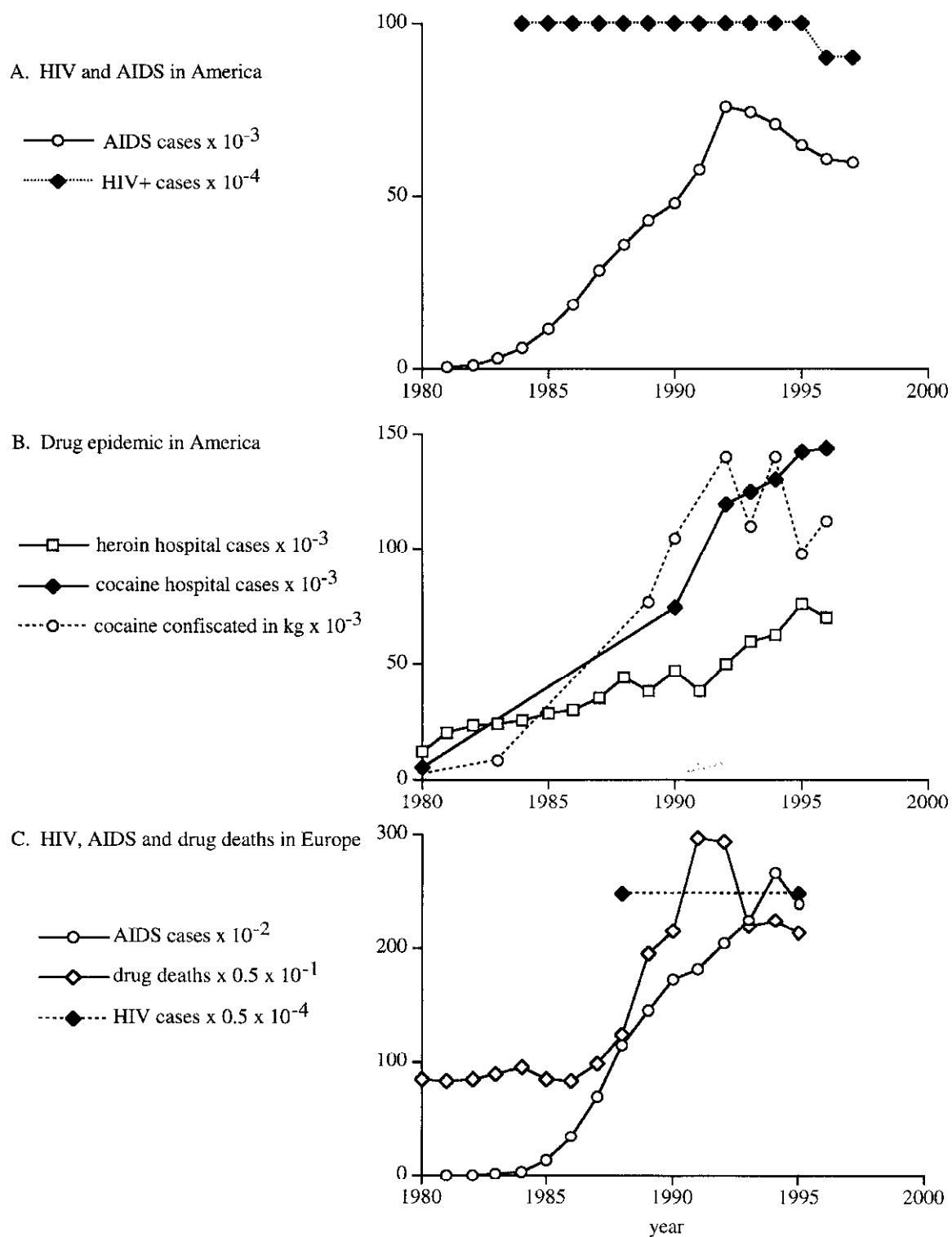


Figure 1.

10. Why does AIDS manifest in totally unrelated diseases, for example, dementia, diarrhea, and Kaposi's sarcoma?
11. Why do only male homosexuals get Kaposi's sarcoma?
12. Why are thousands of AIDS cases HIV-free (Duesberg, 1993e)?

Each of these questions addresses a paradox of the HIV hypothesis. But there are no paradoxes in nature, only flawed hypotheses.

To solve the AIDS dilemma, and to develop a hypothesis that answers all of these questions, we have considered all available facts of the AIDS epidemic in America and Europe, following the classic procedure described by Cairns: '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 the sufferers have in common. This was true for the infectious diseases, the various dietary and vitamin deficiencies, the many kinds of "natural" and industrial poisonings and so on' (Cairns, 1978). The scope of our study is limited to American and European AIDS for reasons that will become apparent in this article. African, Haitian, and Asian AIDS have been covered elsewhere (Duesberg, 1992a; Duesberg, 1996d; Hodgkinson, 1996; Fiala & Lingens, 1997; Fiala, 1998; Shenton, 1998).

### **Common epidemiological properties of AIDS: not contagious and not random – just like lifestyle diseases**

The epidemiological method investigates the distribution and spread of diseases in populations. But its first task is to distinguish between contagious or infectious and non-infectious diseases, because new infectious diseases spread exponentially and thus need to be controlled more urgently than the non-infectious ones.

This critical distinction is made by determining whether a patient had contact with another patient, a process that is called contact tracing. Since human contacts on average are random, the contagious diseases are randomly distributed with regard to sex. Moreover, most infectious diseases are biased in favor of the very young and the very old, because immunity is undeveloped in the young and is failing in the old. Sexually transmitted diseases are biased towards young adults – for example, teenagers and those in their twenties and thirties – because they have more

sexual contacts than older groups (Aral & Holmes, 1991).

By contrast, lifestyle diseases, such as lung cancer from smoking and liver cirrhosis from alcoholism, are *not* randomly distributed with regard to sex but follow the sex distribution of risk groups (Cairns, 1978; Cairns, 1997). Further, the age distribution of lifestyle diseases like lung cancer from smoking and liver cirrhosis from drinking is biased to older age (Cairns, 1978). In the following, we use these epidemiological criteria to determine whether American and European AIDS is infectious or is a lifestyle disease.

### *AIDS not contagious*

The hallmark of all infectious diseases is contagiousness. The professional literature has yet to describe the first doctor who has contracted AIDS (not HIV) from the over 650,000 American and over 150,000 European AIDS patients through the various contacts that occur between doctors and patients (Duesberg, 1996d). Since an estimated one million health care workers are accidentally stuck by needles contaminated by patients per year in the US, and a thousand annually contract hepatitis by this route (Holding & Carlsen, 1998), the absence of AIDS in the American health care workers who have treated 650,000 AIDS patients indicates that AIDS is not contagious.

However, in the non-scientific literature, the CDC claims 25 'possible occupationally acquired AIDS' cases among all the American health care workers who have treated over 650,000 AIDS patients over 17 years (Centers for Disease Control and Prevention, 1997). The CDC did not identify the gender, nor the age, nor the AIDS-defining disease of the 25 health care workers with AIDS. Moreover, the CDC did not provide any evidence that the 25 'possible occupationally acquired AIDS' cases were indeed occupational. This may be difficult to verify since the primary, non-occupational AIDS risk is illicit, recreational drug use, which is not compatible with a medical license. Above all, the CDC did not determine or disclose whether the 25 HIV-positive health care workers had been treated with anti-HIV drugs, which cause immuno-deficiency and other diseases (see below). But even if all of these uncertainties could be resolved, 25 disease cases among 650,000 disease contacts in 17 years are hardly convincing evidence for contagiousness.

Contrary to expectation, female prostitutes also did not pick up AIDS from their clients, unless they were also recreational drug users (Root-Bernstein, 1993; Fi-

ala & Lingens, 1997). Likewise, the wives of several thousand American hemophiliacs with AIDS-defining pneumonias and candidiasis have not contracted AIDS from their husbands (Duesberg, 1995c; Duesberg, 1995d; Duesberg, 1996b; Hodgkinson, 1996).

Not one of tens of thousands of scientists searching for the cause of AIDS has ever contracted AIDS from blood, tissue samples, or patients in the past 17 years (Cohen, 1994a; Duesberg, 1996c; Duesberg, 1996d). The few cases who allegedly contracted AIDS from studying blood samples of AIDS patients appear to be all from the laboratory of the virus researcher Robert Gallo at the National Institutes of Health (NIH) in Bethesda (Cohen, 1994a; O'Brien & Goedert, 1996; O'Brien, 1997). However, until their AIDS diseases and their anti-AIDS treatments are described they cannot even be considered anecdotal cases.

Further, the three million Americans who annually receive blood transfusions for life-threatening diseases (Duesberg, 1992a) should have developed AIDS from blood donors if AIDS were infectious. But there was no increase in AIDS-defining diseases (Table 1) among transfusion recipients in the AIDS era (Ward et al., 1989), and no AIDS-defining Kaposi's sarcoma has ever been observed in millions of transfusion recipients (Haverkos et al., 1994; Duesberg, 1995d; Duesberg, 1996c; Hodgkinson, 1996). Contrary to predictions of transfusable AIDS, the life span of American hemophiliacs has increased more than two fold, from 11 years in the early 1970s to 27 years in 1987 (the year AZT was introduced, see below) because they were prophylactically treated with transfusions of factor VIII (Duesberg, 1995c).

Numerous anecdotal cases of 'discordant couples' confirm that AIDS is not contagious: The tennis star Arthur Ashe lived for 10 years with his wife and had an 8-year-old daughter before he died from AIDS and AZT in 1994, but his family is AIDS-free (Ashe & Rampersad, 1993; Duesberg, 1996d). Also in 1994, the wife of Hollywood actor Paul Glaser died from AIDS and AZT, but after a marriage of 13 years and two children Glaser is healthy to this date (Duesberg, 1996d) (Sherry Thorup, personal communication 1998). Likewise, movie star Rock Hudson died from AIDS wasting and Kaposi's sarcoma in 1985, but Marc Christianson, his last relationship of over two years which began during an era before safe sex, is healthy 13 years later (Hudson & Davidson, 1986; Duesberg, 1996d). Thus, the huge body of literature on AIDS cannot offer convincing evidence that AIDS is

contagious (Duesberg, 1992a; Stewart, 1996). Instead, all published data prove that AIDS is not.

#### *AIDS highly non-random with regard to sex*

Almost 90% of the over 800,000 American and European AIDS patients are men, and only 10% are women (Adams, 1989; Centers for Disease Control and Prevention, 1995; World Health Organization, 1995b; AIDS Zentrum im Robert Koch Institut, 1996; Hodgkinson, 1996; Fiala & Lingens, 1997). In epidemiology, this is as different as day and night from the common 50% distribution between the sexes of all known infectious diseases (Evans, 1982; Evans & Feldman, 1982). Despite widespread heterosexual prostitution, AIDS has remained a male epidemic (Fiala & Lingens, 1997).

#### *AIDS highly non-random with regard to sexual orientation*

Almost two out of three American and a slightly lower percentage of European AIDS patients are male homosexuals (Adams, 1989; Centers for Disease Control and Prevention, 1995; World Health Organization, 1995b; Centers for Disease Control and Prevention, 1996; Hodgkinson, 1996; Fiala & Lingens, 1997). Thus, among men, the AIDS epidemic is non-randomly distributed with regard to sexual orientation.

#### *AIDS non-random with regard to age*

Over 80% of the American and European AIDS patients are 25 to 49 years old – an age group that is traditionally the least likely to develop infectious diseases (Centers for Disease Control and Prevention, 1995; World Health Organization, 1995b). About 5 to 7% are 20 to 24 years of age, and most of the remainder are between 50 and 59 years old. About 1% of the AIDS patients are babies (i.e., 500 to 800 annual cases in America and 500 to 600 in Europe), and virtually none are between 5 and 20 years old (Centers for Disease Control and Prevention, 1995; World Health Organization, 1995b; Centers for Disease Control and Prevention, 1997). Thus, AIDS is highly non-random with regard to age, and its age bracket is not compatible with infectious disease. The absence of AIDS in teenagers directly calls into question the view that AIDS is a venereal disease.

*AIDS progresses non-exponentially – unlike infectious disease, and unlike the HIV epidemic*

In America and Europe, AIDS has progressed slowly and non-exponentially over 10 years and then declined slowly (Figure 1 A and C, see also page 105) (Centers for Disease Control and Prevention, 1996; Centers for Disease Control and Prevention, 1997; Fiala & Lingens, 1997; National Commission on AIDS, July 1991) – just like a lifestyle disease (Cairns, 1978). By contrast, a new viral epidemic, such as, for example, a seasonal flu, spreads exponentially in a susceptible population within weeks or months (Fenner et al., 1974; Evans, 1982; Mims & White, 1984). This characteristic of an infectious epidemic, pathogenic (like flu) or not (like HIV), was first discovered in the early 19th century by the British scientist William Farr, and has since been called Farr's law (Bregman & Langmuir, 1990).

Subsequently, infectious epidemics, particularly viral epidemics, disappear again within weeks or months as a result of anti-viral or microbial immunity and the selection of resistant survivors (Stewart, 1968; Fenner et al., 1974; Mims & White, 1984). This generates the conventional bell curve, a rapid rise of cases within weeks or months, followed by a decline as immunity arises and susceptible hosts die out (Bregman & Langmuir, 1990). But there is no evidence for the emergence of immunity as the AIDS epidemic continues to progress (Figure 1A and C). Thus, according to Farr's law, American and European AIDS is incompatible with infectious disease but consistent with non-contagious lifestyle diseases.

*Subepidemics with distinct diseases*

From the very beginning, AIDS epidemiologists have recorded non-random distributions of AIDS-defining diseases in different AIDS risk groups. For example, Kaposi's sarcoma is almost exclusively found in male homosexuals (Selik et al., 1987; Beral et al., 1990), weight loss and tuberculosis predominate in intravenous drug users, and pneumonia and candidiasis are almost the only two of the 30 AIDS-defining diseases that are diagnosed in hemophiliacs and other transfusion recipients (Duesberg, 1992a; Duesberg, 1996d; Hodgkinson, 1996). Thus, the AIDS epidemic breaks down into subepidemics based on subepidemic-specific diseases. Distinct, subepidemic-specific diseases rule out a common infectious and a common non-infectious cause. They indicate that several independent causes generate the AIDS epidemic.

In sum, AIDS epidemiology is incompatible with infectious disease. In view of this, even the CDC has called into question the original basis for the assumption that AIDS is infectious, which was tracing sexual contacts between patients (Auerbach et al., 1984; Shilts, 1987). The CDC now admits that it 'may be difficult to identify [AIDS from sexual contacts] because most persons with AIDS have had contact with many different people. In particular, drug users and homosexual and bisexual men may have had contact with hundreds of partners that they did not know very well' (Drotman et al., 1995).

Nevertheless, some AIDS-defining diseases, such as tuberculosis, candidiasis, and pneumonia, are in principle infectious, microbial diseases, although immunodeficiency is not (Table 1). Thus, to understand why some people are at risk for these diseases and others are not, one must analyze the conditions under which microbes cause disease. It has long been known that most microbes are only conditionally pathogenic with the primary condition being immunodeficiency (Stewart, 1968; Mills & Masur, 1990; Moberg & Cohn, 1991). For example, although tuberculosis is a contagious disease, it is rarely transmitted to doctors treating tuberculosis patients because doctors are rarely immunodeficient.

It would appear that there must be common, non-infectious causes that render AIDS patients susceptible to both the infectious and non-infectious AIDS diseases. In order to identify these causes, we shall first analyze the diseases AIDS patients have in common and then search for their cause.

**What AIDS patients have in common: neither an AIDS - specific disease, nor any one of 30 previously known AIDS-defining diseases, nor even immunodeficiency, but only antibody against HIV – by definition**

In 1981, the CDC, the US government's institution for the surveillance of infectious diseases, first recorded an increase of 12 previously known diseases above their long established, national backgrounds (Centers for Disease Control, 1981; Selik et al., 1984). Nearly all of the patients were 'promiscuous' male homosexuals and intravenous drug users (Durack, 1981; Centers for Disease Control, 1986). The list of these diseases has since been increased to about 30 (Centers for Disease Control and Prevention, 1992). Since every one of these diseases has been previously known, the AIDS

Table 1. AIDS-defining diseases in the U. S. in 1995<sup>1</sup>

Immuno-deficiencies	(in %)	Non-immuno-deficiencies	(in %)
pneumonia	33	wasting/weight loss	15
candidiasis	14	Kaposi's sarcoma	6
tuberculosis <sup>2</sup>	10	dementia	3
cytomegalovirus	7	lymphoma	2
toxoplasmosis	4		
herpesvirus	4		
diarrhea	2		
total	74		26

<sup>1</sup> (Centers for Disease Control and Prevention, 1995).

<sup>2</sup> includes other mycobacterial infections.

epidemic offers neither a new, nor even a specific, disease of its own.

From the beginning, the AIDS diseases fell into two categories that are totally unrelated to each other: (1) the non-immunodeficiency diseases including dementia, the cancers Kaposi's sarcoma and leukemia, and weight loss, and (2) the immunodeficiency diseases, including the viral and microbial pneumonias, the diarrheas, yeast, and other opportunistic infections (Table 1).

Despite the profound disparity in the AIDS diseases, the CDC proposed in 1982 that immunodeficiency was their common denominator (Centers for Disease Control, 1982). But since immunodeficiency was not considered to be infectious in 1982, AIDS was not classified as an infectious epidemic. Moreover, immunodeficiency, no matter what its cause, is not a known cause of cancer, dementia, and weight loss (Duesberg, 1992a; Duesberg, 1996c). Accordingly, there is no immunodeficiency in many AIDS patients with Kaposi's sarcoma (Afrasiabi et al., 1986; Murray et al., 1988; Spornraft et al., 1988; Archer et al., 1989; Friedman-Kien et al., 1990).

On April 23, 1984, the Secretary of Health & Human Services (HHS), the directors of the CDC and NIH, and the NIH's virus researcher Robert Gallo announced jointly at an international press conference in Washington that a previously unknown virus, since called HIV, was the 'probable cause of AIDS' (Altman, 1984). The only evidence for this claim was the presence of antibody against the 'AIDS virus' in most, but not all, AIDS patients that Gallo and his collaborators had analyzed (Gallo et al., 1984). Even to this date, AIDS researchers cannot find HIV antibody in

all (Duesberg, 1993e), nor infectious HIV in most, AIDS patients (Duesberg, 1992a; Duesberg, 1996d; Duesberg, 1996c).

Despite the instant popularity of an immunodeficiency virus, the hypothesis had four serious birth defects:

1. Oblivious of AIDS epidemiology, Gallo, other virus researchers, and the secretary of HHS tacitly assumed that 'where there is a virus, or even just antibody against one, there must be an infectious disease' [according to the proverbial, 'where there are hoofbeats, there must be horses'] (Blattner et al., 1988; Weiss & Jaffe, 1990; Harden, 1992; O'Brien & Goedert, 1996; O'Brien, 1997). But the epidemiology of AIDS is incompatible with infectious disease.
2. Gallo and other AIDS researchers apparently had overlooked that antibodies are hardly a cause of disease. Instead, antibodies offer the only known protection against viral pathogenesis – the operating principle of vaccination (Blattner et al., 1988; Weiss & Jaffe, 1990; O'Brien & Goedert, 1996; O'Brien, 1997).
3. Gallo and other AIDS researchers apparently also overlooked that their hypothesis failed Koch's first postulate, according to which every case of a disease should contain the suspected causative microbe (Merriam-Webster, 1965; Harden, 1992).
4. They also overlooked that over a third of all American AIDS patients in 1984 (Duesberg, 1991; Duesberg, 1992a), and 26% in 1996, do not even have immunodeficiency diseases (Table 1).

Nevertheless, the 'AIDS virus' was accepted without delay, and without review, even before it was published in a scientific journal (Altman, 1984). Both the threat of a new infectious epidemic and the endorsement by the US government immediately established the virus-AIDS hypothesis as a national article of faith.

In 1985, one year after Gallo's announcement, the CDC officially redefined AIDS as an infectious disease. According to the CDC's 1985 AIDS definition, HIV had become the one and only definitive diagnostic criterion of AIDS (Centers for Disease Control, 1985). Moreover, HIV had become the first virus to cause 30 fatal diseases, but no specific disease of its own – because each of the 30 diseases can result from previously known causes. Thus, as of 1985, the following definition applied:

Any of 30 diseases + HIV = AIDS

Any of the same 30 diseases - HIV = any of the 30

diseases.

This definition has made AIDS an ‘HIV disease’ (Institute of Medicine & National Academy of Sciences, 1986), although there was no proof in 1985 that HIV causes AIDS (Duesberg, 1987; Blattner et al., 1988; Duesberg, 1988). And there has been no proof since (Cohen, 1995; Mullis, 1996; Balter, 1997). On the contrary, according to *Nature Medicine*, two papers in 1998 drove ‘the final nail in the coffin’ of the hypothesis that HIV causes AIDS by killing T-cells (Roederer, 1998).

The CDC’s HIV-based AIDS definition has relegated into a gray zone thousands of HIV-free AIDS cases that had been diagnosed clinically and on the basis that the patient belonged to an ‘AIDS risk group’ (Duesberg, 1993c). While the existence of HIV-free AIDS continued to be acknowledged despite the 1985 AIDS definition (Centers for Disease Control, 1987; Centers for Disease Control and Prevention, 1992), HIV-free cases could now be readily dismissed as an argument against the HIV hypothesis by classifying them as non-AIDS cases under their old names (see 6). By allowing AIDS diagnosis without HIV yet insisting on an HIV-based AIDS definition, the CDC has created many unnecessary problems that have obscured AIDS research to this date (see pages 102, 103) (Duesberg, 1993e; Duesberg, 1996d).

Moreover, from the beginning, the AIDS and HIV epidemics were not at all compatible. Instead they have moved in different directions in the US and Europe since both can be tested simultaneously – despite their linkage by the HIV hypothesis. In the US, the HIV epidemic has been steady at about one million from 1985, with small downward adjustments since 1996 (Figure 1A) (Curran et al., 1985; Duesberg, 1992a; Duesberg, 1996c; Krieger, 1996; World Health Organisation, 1996; Centers for Disease Control and Prevention, 1997). New HIV infections have even declined fivefold from 1985 until 1993, based on over 50 million blood donations collected in that time (Figure 2A) (Centers for Disease Control, 1995). By contrast, during the same time period AIDS has increased from a few dozen to over 50,000 cases annually (Figure 1A).

In Europe, a steady 0.5 million individuals were positive between 1988 and 1995 (Figure 1C) (Mann et al., 1988; Merson, 1993; World Health Organization, 1995a). In Germany, the number of new infections declined about twofold during the same time period in which AIDS rose from none to 2000 cases per year

(Figure 2B) (AIDS-Zentrum im Robert Koch-Institut, 1997; Fiala & Lingens, 1997).

Since the American and European AIDS and HIV epidemics move in totally different directions, the HIV hypothesis cannot explain AIDS. Moreover, the steady state of infection proves that HIV is an old, long-established virus in the US and Europe (see Farr’s law above) (Bregman & Langmuir, 1990; Duesberg, 1992a).

Thus, AIDS patients have neither an HIV- nor an AIDS-specific disease, nor any one of the 30 AIDS-defining diseases, nor even immunodeficiency, in common. However, by definition they all share antibodies against HIV.

### **Drugs – the common denominator of AIDS in America and Europe: (a) Recreational drugs**

Hardly any one now remembers that before the popular virus-AIDS hypothesis many American and some European investigators had postulated that the epidemic was a collection of drug diseases (Durack, 1981; Rappoport, 1988; Adams, 1989; Oppenheimer, 1992; Hodgkinson, 1996).

#### *The lifestyle hypothesis*

In an editorial of the *New England Journal of Medicine* in 1981, long before AIDS was claimed to be infectious, David Durack proposed: ‘Perhaps one or more of these recreational drugs is an immunosuppressive agent. The leading candidates are the nitrites, which are now commonly inhaled to intensify orgasm’ (Durack, 1981). The reason for the early suspicion of drugs was simple. Based on data from the CDC and from independent investigators, nearly all AIDS patients were either male homosexuals who had used recreational drugs as aphrodisiacs and psychoactive stimulants, or were heterosexual intravenous drug users (Durack, 1981; 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; Krieger & Caceres, 1985; Newell et al., 1985a; Newell et al., 1985b; Lauritsen & Wilson, 1986; Haverkos & Dougherty, 1988b; Rappoport, 1988). For example, James Curran, the director of the CDC, stated between 1981 and 1982: ‘At this point our best clue to the cause of the disease was ‘poppers’ [nitrite inhalants]’ (Fettner & Check, 1985). Curran’s clue was gleaned from anecdotal evidence



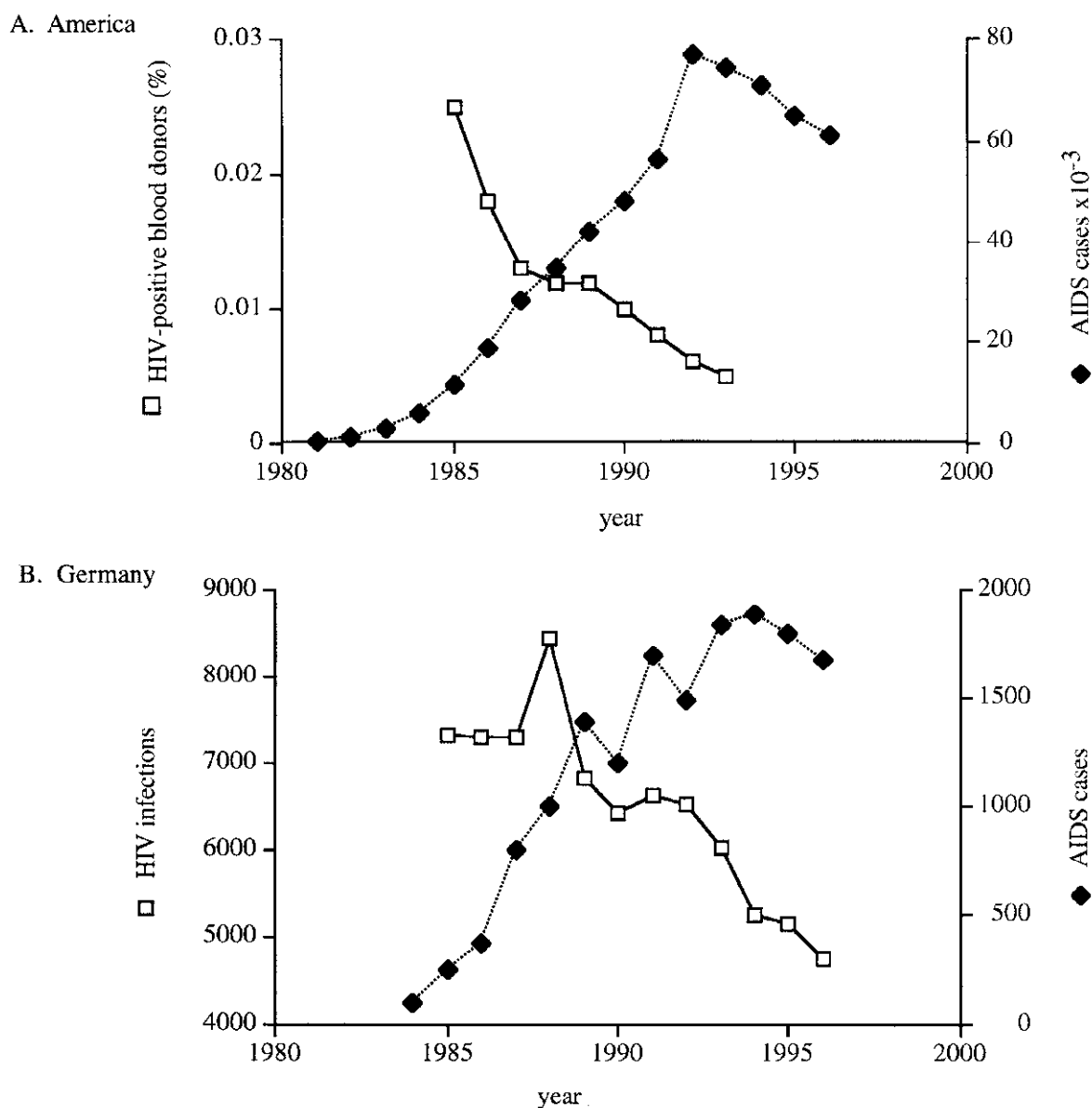


Figure 2. (A) New HIV infections in percent of over 50 million blood donors, and AIDS cases per year in America (Centers for Disease Control, 1995). (B) New HIV infections and AIDS cases per year in Germany (AIDS-Zentrum im Robert Koch-Institut, 1997).

including the first two Kaposi's sarcoma patients seen by Alvin Friedman-Kien, professor of dermatology at New York University. Both of these patients were male homosexuals who 'had a multiplicity of sexual partners over an extended period of time as well as using a variety of recreational drugs – cocaine, marijuana, LSD, THC, MDA, and amyl nitrite.' Friedman-Kien regularly called CDC officials to report his experience with AIDS: '...as patients started coming in, it turned

out that all of them, 100 percent, had been using amyl nitrite' (Fettner & Check, 1985).

Evidence continued to mount strongly supporting a connection between nitrite use, other recreational drugs, and AIDS. This included articles by James Goedert and William Blattner from the NIH, the CDC's sister institution (Goedert et al., 1982), by Harry Haverkos with the CDC's Kaposi's Sarcoma Opportunistic Infection (KSOI) task force, and an abundance of other studies on the immunotoxic and

Table 2. Drug use by homosexuals with AIDS and at risk for AIDS

DRUGS	Atlanta <sup>1</sup> 1983: 50 AIDS, 120 risk	San Francisco <sup>2</sup> 1987: 492 risk	San Francisco <sup>3</sup> 1990: 182 AIDS	Chicago <sup>4</sup> 1993: 5000 AIDS + risk	San Francisco <sup>5</sup> 1993: 215 AIDS, 230 risk*	Vancouver <sup>6</sup> 1993: 136 AIDS, 229 risk	USA, Europe, Australia <sup>7</sup> 1994: 114 AIDS, 289 risk	London, Manchester <sup>8</sup> 1996: 685 risk
nitrite inhalants	96%	82%	79%	71–100%	100%	98%	50%	80%
ethylchloride	35–50			18				
cocaine	50–60	84	69	40–66	yes	yes	12–34	40
amphetamines	50–70	64	55	26–46	yes	yes	6–27	48 (ecstasy)/ 57 (speed)
phenylcyclidine	40	22	23	yes				
LSD	40–60		49	yes				48
metaqualone	40–60	51	44					
barbiturates	25	41	30	yes				
marijuana	90		85	88			41–68	76
heroin	10	20	3	yes				25
alcohol			46	95			90	95
cigarettes			33	yes			50	58
AZT					most	most	15–64	
drug-free	0	0	0	0	0	0	0	0

<sup>1</sup> (Jaffe et al., 1983); <sup>2</sup> (Darrow et al., 1987); <sup>3</sup> (Lifson et al., 1990); <sup>4</sup> (Kaslow et al., 1989), (Ostrow et al., 1990), (Ostrow et al., 1993); <sup>5</sup> (Ascher et al., 1993), (Ellison et al., 1996); <sup>6</sup> (Schechter et al., 1993b), (Craddock, 1996); <sup>7</sup> (Veugelers et al., 1994); <sup>8</sup> (Gibbons, 1996). \* See text.

Note: Not one of these studies reported a drug-free man with AIDS or at risk for AIDS.

carcinogenic effects of nitrite inhalants (Newell et al., 1984; Haverkos & Dougherty, 1988a; Haverkos & Dougherty, 1988b). An English team reported in 1984 that 86% of male homosexual AIDS patients from St Mary's Hospital in London had inhaled nitrites compared to 86.4% from clinics in New York, San Francisco, and Atlanta (McManus et al., 1982). In 1983, two dozen of America's leading AIDS investigators including Friedman-Kien, Curran, and CDC worker Harold Jaffe, later director of the CDC's HIV/AIDS Division, had conducted extensive epidemiological studies which revealed overwhelming drug use, including nitrite inhalants, cocaine, and amphetamines by all homosexual AIDS patients studied (Table 2) (Jaffe et al., 1983).

Drugs seemed to be the most plausible explanation for the restriction of AIDS to risk groups, because drug consumption was the only health risk male homosexuals and intravenous drug users had in common (Krieger & Caceres, 1985). This original drug-AIDS hypothesis was euphemistically called the 'lifestyle hypothesis' (Oppenheimer, 1992). Indeed, massive supplies of illicit recreational drugs such as nitrite (poppers) and ethylchloride inhalants, cocaine, heroin, amphetamines, phenylcyclidine, and LSD had reached

America and Europe since the Vietnam War and were the only statistically significant new health risks that had affected these countries since World War II (see page 103).

Suddenly, after the announcement of the discovery of the 'AIDS virus' by Gallo at the international press conference in Washington on April 23, 1984 (see above), the lifestyle hypothesis was dropped without notice – as if it never existed – in favor of the virus-AIDS hypothesis. Since then, any revisionism was immediately regarded as obsolete, or in the words of David Baltimore even as a 'pernicious and irresponsible' obstacle (Booth, 1988) in the war against the AIDS virus (Weiss & Jaffe, 1990; Cohen, 1994a; Duesberg, 1996d; O'Brien & Goedert, 1996; O'Brien, 1997). Henceforth, recreational drugs were only studied, if at all, as risk factors of HIV infection or as obstacles in anti-HIV medications.

An article in the gay interest magazine *Genre* captures the 'fashionable opinion' (A. N. Whitehead) of the HIV era: '...the real danger of the drug lies in its crystalline high that engenders dangerous behavior. One doctor recounts the story of a young patient of his who 'bumped' (took crystal [amphetamine]) for four days at Rehoboth Beach – four days of parties and

sex and more parties and more sex. When he came down at last, he felt drained and depressed. But that was nothing compared to how he felt when he discovered he had contracted HIV... The same doc also talks about an HIV patient who got 'methed up' (with methamphetamine) and went off his medication... The doc was angry, but he's wise to the ways of addicts' (Bergling, 1997).

In a rare response to the situation, William Lerner, from the Division of General and Preventive Medicine of the University of Alabama at Birmingham, writes in the *Journal of American Medicine*: 'With few exceptions, the treatment of drug problems in the United States occurs both figuratively and literally, a long distance from the major medical centers. ... the efforts expended stand in stark contrast to those rallied in the investigation of AIDS.... A recent survey of medical centers from the United States revealed fewer than 30 current fellowship programs in alcoholism and drug abuse in the entire country, with approximately 60 enrolled' (Lerner, 1989).

#### *Intravenous drug use is reported for a third of all AIDS patients in the HIV era*

Although the lifestyle hypothesis has been discredited by proponents of the virus-AIDS hypothesis, government statistics and independent publications continue to confirm to this date that about one third of the American, and over one third of the European AIDS patients, are intravenous users of cocaine, heroin, and other drugs (Duesberg, 1992a; World Health Organization, 1995b; Centers for Disease Control and Prevention, 1996; Duesberg, 1996c; Duesberg, 1996d; Hodgkinson, 1996; Duesberg & Rasnick, 1997; Fiala & Lingens, 1997). Indeed, virtually all of the female and heterosexual male AIDS patients are intravenous drug users.

#### *Drug use by male homosexual AIDS patients in the HIV era*

Since HIV had become the consensus cause of AIDS, several large studies of male homosexual volunteers have been set up simultaneously in San Francisco, Chicago, Baltimore, Los Angeles, and Pittsburgh in the US, and in Amsterdam, London, Sidney, and Vancouver abroad, to study risk factors of HIV infection. Some of these studies have reported drug-taking as a risk factor of HIV infection, because psychoactive drugs jeopardize safe sex and increase the number of contacts (Fineberg, 1988; Bergling, 1997; Signorile,

1997b). Inadvertantly, some of these studies also offer evidence for the lifetime dosage of drugs, because lifestyles, including drug use, are often recorded over periods of 10 years or more, a period which is euphemistically called the 'latent period' of HIV by the proponents of the virus-AIDS hypothesis (Duesberg, 1992a; Duesberg, 1994; Duesberg, 1996d). According to David Ostrow, leading epidemiologist of the largest lifestyle study of American homosexual men, the Multi Center AIDS Cohort or MACStudy from Chicago, Baltimore, Los Angeles, and Pittsburgh: 'From the earliest case control studies conducted by the Centers for Disease Control's Task Force on Kaposi's Sarcoma and Opportunistic Infections (Jaffe et al., 1983) to recent studies of predictors of human immunodeficiency virus-type 1 (HIV) infection, recreational psychoactive drug use has been associated with HIV-related illness or infection among homosexual men' (Ostrow et al., 1993).

In the following, we investigate which 'recreational, psychoactive drugs' have been, and continue to be, associated with 'HIV-related illness' of homosexual men.

#### *Nitrite inhalants – the 'gay drug'*

Numerous articles published in the HIV era have documented the continued popularity of nitrite inhalants, the 'gay drug' (Mann, 1995; Mirken, 1995; St. Angelo, 1996; Duesberg & Rasnick, 1997; Lauritsen & Young, 1997), among male homosexuals with AIDS and at risk for AIDS (Shilts, 1987; Lauritsen, 1993; Mann, 1995; Mirken, 1995; Young, 1995; Bethell, 1996; Hodgkinson, 1996; Lauritsen, 1996; St. Angelo, 1996), (see also Table 2). The National Institute on Drug Abuse (NIDA) first drew attention to the nitrite epidemic among male homosexuals in 1988 (Haverkos & Dougherty, 1988b) and called a nitrite-AIDS conference in 1994 to point out that 'nitrite use by gay men in Chicago and San Francisco' has increased in the 1990s after a decline in the late 1980s (Lauritsen, 1994; Haverkos & Drotman, 1995). The San Francisco Department of Health reported in 1991 that 98% of nitrite inhalant users are homosexuals (San Francisco Department of Public Health & Lesbian & Gay Substance Abuse Planning Group, 1991; Ascher et al., 1993). Based on such information, Ostrow et al. concluded that nitrite inhalants show a 'consistent and strong cross-sectional association with ... anal sex' (Ostrow, 1994), and are used because of 'their ability to briefly relax the smooth muscles of the anal sphinc-

ter and thereby facilitate penetration' (Ostrow et al., 1993).

In 1996, a rare survey about the use of nitrite inhalants in continental Europe appeared in the Swiss gay interest journal *aK*, '... seit Jahren von vielen Leuten – vor allem Schwulen – beim Sex zwecks Verstärkung der Lust verwendet wird' [used as a gay drug for years] (Ineichen, 1996). Bottles containing poppers, which cost less than 1 Sfr to produce, sell for up to 58 Sfr in Zurich, Lucerne, Bern, and Basel. According to the journal, sales have been banned in some Swiss states, because amyl nitrites, but not other nitrites, are listed as poisons by the federal public health office, BGA.

#### *Multi drug use*

Just as was the case before the HIV era, American and European male homosexuals at risk for, and with, AIDS combine various recreational drugs (Table 2) (Jaffe et al., 1983; Darrow et al., 1987; Lifson et al., 1990; Duesberg, 1992a; Ascher et al., 1993; Duesberg, 1993b; Schechter et al., 1993a; Schechter et al., 1993c; Buchbinder et al., 1994; Mann, 1995; Mirken, 1995; Young, 1995; Ellison et al., 1996; Lauritsen & Young, 1997). For example, the over 5000 homosexual men of the MACStudy report combinations of 11 recreational drugs in 1993 (Table 2) (Kaslow et al., 1989; Ostrow et al., 1993). Among them, 83% had used one drug, and 60% had used two or more drugs during sex in the previous six months (Ostrow et al., 1990). In the summer of 1996, the magazine *Gay Times* conducted 'the biggest ever survey of gay men's drug use' in England. According to the 685 respondents, 80% used poppers (nitrite inhalants); 48%, ecstasy (amphetamines); 57%, speed (amphetamines); 40%, coke (cocaine); 48%, acid (LSD); 25%, heroin; 76%, cannabis; 58%, cigarettes; 95%, alcohol (Table 2) (Gibbons, 1996). A 'tricontinental seroconverter study' confirms and extends the pattern of multi drug use by male homosexuals with AIDS or at risk for AIDS to America, Europe, and Australia in 1994 (Table 2) (Veugelers et al., 1994).

In an interview with the gay magazine *The Advocate* about a 'Morning party' to benefit the Gay Men's Health Crisis (GMHC) on Fire Island in New York in August 1992, Larry Kramer, founder of GMHC and of the gay AIDS activist organization Act Up, commented:

I loathed the Morning Party. The Morning Party sent me into a depression I cannot begin to de-

scribe. After twelve years of the plague, I should come back and see the organization that was started in my living room having a party like that! ... There were 4,000 or 5,000 gorgeous young kids on the beach who were drugged out of their minds at high noon, rushing in and out of the Portosans [portable toilets] to fuck, all in the name of GMHC (Zonona, 1992).

In his novel *Faggots*, Kramer had offered an earlier client's eye-view of homosexual life-style, including a long list of the 56 most popular recreational drugs as sexual and mental stimulants (Adams, 1989; Duesberg, 1996d).

Among these drugs, amphetamines have recently gained popularity over nitrites as sexual stimulants (Sadownick, 1994; Bergling, 1997; Signorile, 1997b). Says the director of an outpatient treatment center in Los Angeles, 'Look at the demographics. It's such a nasty drug, the way it destroys the body and the mind. Crystal (amphetamine) is a gay person's drug and a gay community problem.' (Sadownick, 1994). According to another observer of gay male culture in America who conducted hundreds of interviews, 'almost 50 percent of the gay men in the ghetto are [now also] using steroids ... cosmetically' (Signorile, 1997a).

In July 1997, *The Advocate* published again two articles confirming and extending the massive drug use by male homosexuals in the 'party circuit'. One was entitled, 'Men behaving badly; the recklessness of the 1970s and early '80s has reappeared on the party circuit, where gay men are indulging in illicit drugs and wild sex with increasing abandon' (Heitz, 1997). The other title was 'Slipping up; unsafe sex is on the rise—and the new AIDS drugs are only one of the culprits' (Gallagher, 1997).

Insiders have described the reasons for drug use by homosexual men. An addiction counselor at the Pride Institute at Solutions in Washington, DC told the gay interest magazine *Genre* 'that crystal [amphetamine] targets the same area of the brain where sexual desires are born and satisfied, hence its popularity at sex parties and sex clubs. That connection is why so many recovering addicts fall off the wagon; they simply cannot escape that intimate association.' (Bergling, 1997). *The Signorile Report on Gay Men* records in 1997 in graphic detail why 'a whole set of drugs is required' for a typical 'hard core gay circuit party' in one of the appropriate Hollywood hotels:

Drugs to space you out. Drugs to keep you up. Drugs to put you to sleep. Drugs to get you up again. Drugs to get you through the day. Drug dealers abound, at least one to a floor, it seems. They are called in their rooms at all hours of the day and night. Ecstasy is a staple, as is cocaine, and special K-ketamine, a horse tranquilizer, which is snorted. But it wouldn't be California and a weekend-long circuit event if there wasn't crystal meth, a stimulant that keeps people up for hours on end, sometimes for 48 hour stints, dancing, posing, and having sex. Some people snort their crystal in powder form, but more and more men are injecting intravenously after liquefying it. ... a taxi driver tells me ... 'It was great to work to, I mean I could work for two days straight through. And of course for sex.'

... outside the ballroom on the grounds of the pool, several men vomit on the lawn, others pass out; at some point paramedics arrive and in the blur from the lights and the drugs, I see someone taken out on a stretcher. He overdosed on something. 'Another circuit casualty,' someone says to me with a smile. Indeed another man tells me that there's an OD [overdose] at just every big party. 'That's how you know it's a good party,' he jokes, 'if you can attract an ambulance or two.'

Later, however, when the partying momentarily stops and I go back to my room, I'm unable to sleep ... I'll need more drugs. ... The opposite choice, staying sober (not being 'medicated' as one of my party chumps puts it)... seems not to offer the authentic circuit experience, and the possibility of finding someone else in the same state is rather nil. Staying sober seems a dismal prospect, as if someone has told a funny joke and you are the only one not 'getting it.'

Burnt out and sometimes hooked on coke (cocaine) and crystal (amphetamines) too, some of these men see their careers and their relationships – not to mention their health, particularly if they are HIV-infected – completely unravel (Signorile, 1997b).

The German gay interest magazine *First* confirms that 'Ecstasy [amphetamines], LSD, and other amphetamines have become indispensable for 'House- and Technoclubs.' The use of 'Partyrogen' and their dangers are described by diverse informational

brochures'. ... Some take so much speed that they can't sleep for 3 days. 'E' (ecstasy) makes contact happy and chummy, LSD develops secret desires, and cocaine makes you just horny. But who enjoys pleasant chemical feelings ... does not care about damned condoms' (Holtermann, 1997). Surprisingly the article recommends free 'drug checking' by the German government-supported AIDS Hilfe, to avoid the 'dangers of drug taking.'

Based on hundreds of interviews, the American gay interest magazine *Out* also analyzed why male homosexuals take drugs: 'Once in the gay world, we often became obsessed with never being excluded again by the popular crowd. For many young men today that obsession leads them to ingest or inject themselves with powerful and dangerous steroids, a trend that appears to have begun slowly in the 1980s and taken off in the past few years' (Signorile, 1997a).

On June 21, 1998, the San Francisco radio station KGO AM warned about combining nitrite inhalants with Pfizer's new erection enhancing drug Viagra (both drugs are vasodilators). Several deaths had occurred among male homosexuals in Southern California who had used these drugs in combination. The warning followed one issued by *The Lancet* on combining orthodox nitrite prescriptions with Viagra: '...Pfizer and the US FDA confirmed that [by May] six deaths had occurred among the million or so men who have used Viagra since its approval on March 27 [1998]' (Bradbury, 1998).

Thus, male homosexuals with AIDS and at risk for AIDS have continued to use drugs in the HIV era to this date, although drug use is no longer investigated as a possible cause of AIDS. Remarkably, not one of the many, NIH-sponsored single- or multi-center cohort studies recording drug use by homosexual men with AIDS or at risk for AIDS has ever identified, or tried to identify, even one AIDS patient who was drug-free (Lauritsen, 1994; Lauritsen & Young, 1997) (see page 101 and Table 2)!

#### *1% of AIDS patients are babies who shared drugs with their mothers before birth*

In the US, between 500 and 800 babies are listed annually as pediatric AIDS cases by the CDC (Centers for Disease Control and Prevention, 1995; Centers for Disease Control and Prevention, 1997). In Europe also, about 500 to 600 babies are annually recorded as AIDS patients (World Health Organization, 1995b). Numerous reports confirm that the mothers of about

80% of these babies were intravenous drug users during pregnancy (Mok et al., 1987; Blanche et al., 1989; European Collaborative Study, 1991; Duesberg, 1992a; Centers for Disease Control and Prevention, 1995; Drug Strategies, 1995; World Health Organization, 1995b; Moye et al., 1996; Rodriguez et al., 1996). The remaining 20% of AIDS babies from non-drug using mothers probably represent the long-established background of infant morbidity and mortality due to congenital diseases, malnutrition and poverty.

A survey by the NIDA conducted in 1994 extends and confirms this scenario: 'More than 5 percent (221,000) of the 4 million women who give birth each year use illicit drugs during their pregnancy' (Drug Strategies, 1995). And the Office of the National Drug Control Policy estimates that '3.2% of the pregnant women (approximately 80,000)' are drug users (White House Office of National Drug Control Policy, 1998). Thus, many more infants have shared drugs with their mothers as embryos than are showing up as AIDS babies.

### **Drugs – the common denominator of AIDS in America and Europe: (b) the anti-HIV drugs and the 'polypharmacy' of anti-AIDS drugs**

In the name of the hypothesis that HIV causes AIDS, several DNA chain-terminators and other chemicals have been licensed since 1987 to cure and prevent AIDS by inhibiting HIV replication. The first of these was the DNA chain-terminator AZT (Kolata, 1987; Nussbaum, 1990; Duesberg, 1996d).

#### *DNA chain-terminators and protease inhibitors as anti-HIV drugs*

Between 1987 and 1996, annually about 200,000 American and a smaller number of European HIV-positives with and without AIDS were put on lifelong prescriptions of cytotoxic DNA chain-terminators such as AZT, as anti-HIV drugs (Duesberg, 1992a; Duesberg, 1996d; Duesberg, 1996c; Hall, 1996; Hodgkinson, 1996). As of 1996, about 150,000 HIV-positive Americans take protease inhibitors that are prescribed as 'cocktails' in conjunction with two DNA chain-terminators, instead of DNA chain-terminators only (Stolberg, 1997).

Because annually at least 200,000 HIV-positive Americans and Europeans with and without AIDS

are on anti-HIV drugs, but currently no more than about 70,000 HIV-positive Americans and Europeans develop AIDS per year (see Introduction), it follows that most American and European AIDS patients are on these anti-HIV drugs. Indeed, in 1998 a national survey has confirmed directly that all American AIDS patients are on anti-HIV drugs (Perlman, 1998).

As of 1993, even HIV-free babies participate in the AZT epidemic, because HIV-positive pregnant women are treated for the last two trimesters with AZT in the hope of preventing HIV transmission (Connor et al., 1994). Although the risk to such children of picking up HIV from their mothers is only 25%, and is only reduced by AZT from 25% to 8%, all HIV-positive pregnant women are injected with AZT during the second and third trimesters, and their babies are injected for six weeks after birth to prevent transmission of HIV, the hypothetical cause of AIDS (Connor et al., 1994; Cotton, 1994; The Lancet, 1994).

#### *'Polypharmacy' of anti-HIV/AIDS drugs*

The consumption of AZT and other DNA chain-terminators by healthy HIV-positives at risk for AIDS and by AIDS patients is typically supplemented by a bewildering list of further prescription and over-the-counter drugs. A list of 23 anti-HIV/AIDS drugs taken by 2801 American HIV-positives, including 524 AIDS patients, is recorded in Table 3 (Hayes et al., 1994). Nearly all of these HIV-positives were male homosexuals (83%) or intravenous drug users (12%) who took those drugs because they wanted to prevent or cure AIDS. Another study entitled 'Polypharmacy among patients attending an AIDS clinic: Utilization of prescribed, unorthodox, and investigational treatments' describes even higher drug use by 189 HIV-positives from San Francisco, of which 94% were male homosexuals and 2% were intravenous drug users (Greenblatt et al., 1991). In telephone interviews, 96% of these people reported prescription drugs; 67%, over-the-counter drugs; 31%, investigational drugs; 29%, recreational drugs; and 29%, 'alternative' drugs. An average of 2.3 medications were taken simultaneously by healthy HIV-positives and 5.6 medications were taken simultaneously by those with AIDS symptoms. The authors of the study 'conclude that use of polypharmacy among some AIDS clinic patients is common, could create an increased risk for adverse drug reactions, and may affect clinical drug trials.' A recent study describing hepatitis as a side effect of protease inhibitors also acknowledges simultaneous pre-

Table 3. Anti-HIV/AIDS drugs taken by HIV-positives<sup>1</sup>

Drug	HIV-positives (n=2,801)
Anti-infectives (see below)	1,584 (57%)
Analgesics/antipyretics	1,539 (55%)
Vitamins	1,307 (47%)
Antihistamines	810 (29%)
Antacids/antidiarrhetics/laxatives	571 (20%)
Anxiolytics/sedatives	517 (18%)
Corticosteroids (topical/systemic)	423 (15%)
Sympathomimetics (adrenergics)	381 (14%)
Antidepressants/tranquilizers	323 (12%)
Antitussives/expectorants	316 (11%)
Electrolytic/caloric diuretics	280 (10%)
Cardiovascular	195 (7%)
Vaccines	133 (5%)
None of the above (confirmed)	0 (0%)
<hr/>	
Anti-Infectives by name	
Penicillins	550 (20%)
Acyclovir	476 (17%)
Topical antifungals	442 (16%)
Erythromycin	376 (13%)
Aerosolized pentamidine	260 (9%)
Cephalosporins	254 (9%)
Co-trimoxazole	246 (9%)
Systemic antifungals	244 (9%)
Tetracyclines	210 (7%)
Miscellaneous $\beta$ -lactam	83 (3%)
Dapsone	84 (3%)

<sup>1</sup> (Fogelman et al., 1994)

scription of 6 to 12 additional drugs, including DNA chain-terminators, to each of three patients (Braeu et al., 1997).

According to an AIDS conference in Chicago, hydroxyurea, another inhibitor of DNA synthesis long used for chemotherapy of leukemia, is now used as 'the first inexpensive drug ... for the 50 percent of HIV-positive Americans not receiving treatment because of its cost' (Maugh II, 1998).

Some insiders have described how the medical establishment urges HIV-positives to take countless anti-HIV drugs and how these drugs affect their lives. In 'Checking in, my chart' the HIV-positive playwright Larry Kramer describes his own polypharmacy of 19 drugs composed by several AIDS luminaries such as Anthony Fauci, David Ho, Joseph Sonnabend, Alvin Friedman-Kien, and others:

AZT, acyclovir [for genital herpes], Zantac, colchicine [mitosis blocker], propranolol, spironolactone, myphyston [for liver cirrhosis and hepatitis], Eucerin, Moisturel, Retin-A, mycolog, flucanone, sulfacet-r, Nizoral [fungal dermatitis], Hisminal and Humbid [bronchitis], and Shaklee vitamins, zinc, NAC and a 'turquoise stone which a fortune teller, many years ago, advised' (Kramer, 1994) – for an annual price tag of \$19,000 (Kramer, 1996).

The polypharmacy of the AIDS patient Peter Di Giulio from San Francisco, who suffers from weight loss, chronic diarrhea, skin ailments, and neuropathy, even exceeds that of Kramer. At an annual cost of just over \$41,000, 'Di Giulio has no choice but to organize his life around his medications': the DNA chain terminators d4T, 3TC; Cytovene (for cytomegalovirus) and Zovirax (for herpes); the protease inhibitor Crixivan; the antifungals Diflucan and Septra (for PCP); anti-mycobacterials Biaxin and Myambutol; the anti-diarrheal Lomotil; Valium for anxiety; and an assortment of ten vitamins and supplements (Tuller, 1996).

The polypharmacy against AIDS also extends to HIV-positive children. The treatments prescribed to an American group of 20 boys and 22 girls with an average age of 11 years serve as an example. These children were originally diagnosed as HIV-positive only at 7 years of age or later, but were HIV-positive from birth due to perinatally acquired HIV (Grubman et al., 1995). At the time of HIV diagnosis, 5 of 42 (12%) had some AIDS-defining disease. Yet all but two of the children were put on anti-HIV/AIDS drugs:

Most of the children are receiving multiple chronic medications, with 90.5% (38 of 42) receiving antiretroviral therapy; 78.6% (33 of 42) receiving PCP prophylaxis; 33.3% (14 of 42) receiving fungal prophylaxis; and 23.8% (10 of 42) receiving herpesvirus prophylaxis. Among the children receiving antiretroviral therapy, 78.9% (30 of 38) are receiving zidovudine [AZT]. Other medications frequently prescribed include meter dose inhalers for reactive airway disease in 33.3% (14 of 42) of patients and nutritional supplements for failure to thrive and wasting syndrome in 52.4% (22 of 42) of patients. Only 2 of the 42 children in the cohort are not receiving any medications, with 4 receiving one medication; 14 receiving two; 10 receiving between 3 and 5; and 12 receiving between 6 and

12 different medications daily. Sixty-two percent (26 of 42) of the children receive monthly intravenous infusions of immunoglobulin (Grubman et al., 1995).

A similar study 'evaluated the safety and efficacy of a three drug regimen in a small group of maternally infected infants' – who had no disease (Luzuriaga et al., 1997). The 'asymptomatic patients' were eight babies, 2 to 8 months old, who were experimentally 'treated' for six months with two DNA chain terminators, AZT and didanosine, and a protease inhibitor 'without clinically important adverse effects.'

### **HIV – a surrogate marker for recreational drug use**

Although standard chemical drug testing of AIDS patients is never reported, and even inquiries about drug use have all but ended since HIV has become the consensus cause of AIDS, unreported drug use can still be estimated with surrogate markers. An ideal marker to study drug use should be one that (1) does not cause a disease by itself, and (2) is rare in non-drug users, but common in drug users. It may come as a surprise that HIV meets these conditions exactly.

#### *HIV a harmless passenger virus*

Whereas the staggering AIDS literature has failed to prove that HIV causes AIDS, it has proved that HIV is a passenger virus (Duesberg, 1994; Duesberg, 1996c; Duesberg & Bialy, 1996). Since a passenger virus is not the cause of a disease, it can be defined as follows:

1. The time of infection relative to the onset of any disease is irrelevant, and unpredictable.
2. The virus can be either active or inactive or latent, i.e. neutralized by antibody, during disease.
3. The virus can be totally absent from the disease.

Indeed, HIV meets each of these criteria:

1. HIV typically infects decades before AIDS occurs (the so-called latent period of HIV), if AIDS occurs at all (Duesberg, 1992a; Duesberg, 1996c). Unless a person is also a drug user, his or her AIDS risk cannot be predicted from HIV.
2. HIV is typically latent during AIDS because it is neutralized by antibodies (Duesberg, 1993c; Duesberg & Bialy, 1996).
3. Thousands of HIV-free AIDS cases have been reported (Duesberg, 1993e). Thus, HIV is a prototypical passenger virus.

The AIDS literature has further shown that HIV is naturally transmitted perinatally (Duesberg, 1992a; Connor et al., 1994; Duesberg, 1994; Duesberg, 1996c). Indeed, perinatal transmission of HIV is 25 to 50% efficient (Duesberg, 1988; Duesberg, 1992a; Connor et al., 1994; Hallauer & Kupsch, 1997), but sexual transmission is less than 0.1% efficient (Peterman et al., 1988; Jacquez et al., 1994; Padian et al., 1997). Therefore, HIV depends on perinatal transmission for survival – just like all other retroviruses (Duesberg, 1987; Duesberg, 1992a).

Because pathogenicity during perinatal transmission would be incompatible with the survival of the host, all perinatally transmitted viruses or microbes must be harmless (Duesberg, 1992a; Duesberg, 1996d). It is for this reason that antibody against HIV is found in at least 17 million healthy humans, including 1 million healthy Americans and 0.5 million healthy Europeans (Figure 1) (Merson, 1993; World Health Organization, 1995a; Centers for Disease Control and Prevention, 1997). This is also why HIV was only discovered recently after technology had been developed to detect latent viruses (Duesberg, 1987; Duesberg, 1992a; Duesberg, 1996d; Duesberg & Bialy, 1996). By contrast, all pathogenic viruses were discovered long ago by the diseases that they cause. Thus, HIV is a harmless passenger virus.

#### *HIV is rare in the US and Europe, and extremely hard to transmit horizontally*

HIV is very rare in the non-drug-using population of the US and Europe. Only one out of 260 Americans, and one out of 900 Europeans, are HIV-positive (Figure 1A, C). Moreover, HIV does not readily spread from its established reservoir because it is very difficult to transmit horizontally (as compared to perinatally). It takes, on average, over 1000 sexual contacts with an HIV-positive individual (Peterman et al., 1988; Jacquez et al., 1994; Padian et al., 1997), or parenteral transmission via frequent injection of unsterile street drugs, or blood transfusion, to acquire HIV (Duesberg, 1992a). The inefficiency of transmission reflects the absence of infectious virus, and the very low percentage of even latently infected cells (about 1 per 1000) in antibody-positive persons (Duesberg, 1989; Duesberg, 1992a; Duesberg & Bialy, 1996).



### *Antibody against HIV and other rare microbes are markers for drug use*

The more frequently a person injects unsterile drugs, and the more sexual partners a person has, the more likely that individual is to become infected with a rare microbe (Durack, 1981; Stewart, 1989; Mullis, 1995). Because recreational drugs are typically used to achieve the average of 1000 sexual contacts that are necessary to transmit HIV sexually (Durack, 1981; Fiala & Lingens, 1997), antibody against HIV is a marker for drug use in the US and Europe, where HIV is rare. In the words of a drug addiction counselor from Washington DC, 'addiction to drugs, or at least heavy drug use, is the number one cause of HIV infection' (Bergling, 1997).

Antibodies against other rare passenger viruses are also surrogate markers for recreational drug use. These include Hepatitis B virus (Duesberg, 1992a), the human T-cell leukemia virus that was once considered the cause of AIDS (Gallo et al., 1983), a recently discovered herpes virus, termed HHV-8, which is currently considered a cause of Kaposi's sarcoma (Cohen, 1994b; Ganem, 1997), cytomegalovirus, also once considered a cause of AIDS, and many other rare viruses and microbes (Durack, 1981; Sonnabend et al., 1983; Stewart, 1989; Duesberg, 1992a).

Thus, the high incidence of antibodies against HIV and other rare passenger viruses and microbes in AIDS patients is direct confirmation of many parenteral and sexual contacts, and is indirect confirmation of long-term recreational drug use. But before we can determine whether drugs may cause AIDS, we must determine whether AIDS occurs without drugs.

### **Drug-free AIDS in America and Europe?**

As yet, there is not a single study in the huge body of AIDS literature that has ever described a significant group of American or European AIDS patients (rather than isolated anecdotal cases that fit the HIV hypothesis) who did not use recreational or anti-viral drugs or both (Table 2) (Lauritsen, 1994; Duesberg, 1995b). Although the CDC lists 'heterosexual contacts' as a 'category' of AIDS patients, it never provides evidence that the small percentage of AIDS cases from this by far largest AIDS-risk category of adults is drug-free based on conventional drug tests (Centers for Disease Control and Prevention, 1997). Indeed, the drug connection has been the Achilles' heel of the infectious-AIDS hypothesis from the very beginning.

In 1983, Curran (page 92) and other CDC officials, led by the CDC retrovirologist Don Francis, abandoned the lifestyle hypothesis in favor of 'epidemiologic evidence for a transmissible agent' (Francis et al., 1983). At this occasion, the CDC took the unprecedented step to commission an experimental study to refute the epidemiologically irrefutable nitrite-AIDS hypothesis (see Table 2) (Jaffe et al., 1983). The study was designed to test the immune system of mice after exposure of a few months to nitrite inhalants. The conclusion was published anonymously on one page in the CDC's *Morbidity Mortality Weekly Reports*: 'None of the animals exposed to IBN [isobutyl nitrite] showed any evidence of immunotoxic reactions... [Yet there was] some evidence of thymic atrophy, possibly stress-related...' (Centers for Disease Control, 1983). Considering that T-cell deficiency is the hallmark of AIDS, it is hard to understand how the CDC could have dismissed 'thymic atrophy' in mice exposed to nitrites as 'stress-related.'

In a renewed effort to dissociate AIDS from drug use, *Nature* commissioned in 1993 an epidemiological 'commentary' on an NIH-sponsored cohort of homosexual AIDS patients from San Francisco, investigated by Michael Ascher et al. (Ascher et al., 1993). Simultaneously, *The Lancet* published an epidemiological paper on such patients from Vancouver (Schechter et al., 1993b). Surprisingly, all AIDS patients enlisted in these studies reported abundant drug use: virtually all had used nitrites, most had also used cocaine and amphetamines, and most were also on DNA chain-terminators such as AZT (see Table 2) (Duesberg, 1993a; Duesberg, 1993d; Schechter et al., 1993c; Ascher et al., 1995a; Ellison et al., 1996).

The authors circumvented this confounding problem by comparing patients for the presence of HIV and only one specific drug at a time. Since the correlation with HIV is always 100% by definition (page 91) – even the most popular drug will eventually lose out: 'when controlled for HIV serostatus, there is no overall effect of drug use on AIDS' (Ascher et al., 1993). Thus, both studies refuted drugs as a cause of AIDS without offering even one drug-free AIDS patient (Ascher et al., 1993; Schechter et al., 1993c)!

Ascher et al. were indeed so determined to separate drugs from AIDS that they drew a curve of 'drug-free,' HIV-positive AIDS patients losing their T-cells over time. This curve was displayed on a blue-colored background in the *Nature* commentary (Ascher et al., 1993). But not even three published requests for the source of the 'drug-free' AIDS patients persuaded the

authors to share the documentation for their curve, giving the impression that their 'drug-free' group was an empty set (Duesberg, 1993b; Duesberg, 1993d; Duesberg, 1993a; Ellison et al., 1996). Indeed, an independent analysis of the database of Ascher et al. confirmed that impression and also revealed that Ascher et al. had omitted from their paper 45 HIV-free, but drug-using, patients with AIDS-defining diseases (Ellison et al., 1996).

Despite the highly personalized style of the *Nature* commentary (Duesberg's name was mentioned 13 times), *Nature's* editor John Maddox refused to publish Duesberg's letter inquiring about the confounding problem of the Ascher article in an editorial entitled 'Has Duesberg a right of reply?' (Maddox, 1993). But after *The Lancet* and *Science* published critical letters (Duesberg, 1993d; Duesberg, 1993a; Duesberg, 1995a), Ascher et al. replied two years later in *Science*, 'The proposed AIDS-drug use association is a classic example of confounding, that is, a suggestion of a correlation caused by the association of a spurious factor (drug use) with a factor (HIV infection) causally related to the outcome (AIDS). The standard statistical methods that we used to differentiate cause from confounding factors showed, in this case, that HIV was the cause and that drug-use association was spurious' (Ascher et al., 1995b). Thus, neither *Nature* nor *The Lancet* could find authors that were able to dissociate AIDS from drugs.

With so much encouragement from editors of leading journals, the San Francisco and Vancouver groups teamed up with Australian and Dutch colleagues to refute drugs as the cause of homosexual AIDS once more (Vcugelers et al., 1994). Again, all of 403 AIDS patients studied reported extensive drug use, particularly nitrites, and many were also on AZT (see Table 2). The 'relative [AIDS] hazard' for nitrite use was 1.03 and for AZT use was 1.04. Yet the authors concluded: 'No relation ... of alcohol, tobacco, and recreational drugs with rates of disease progression could be demonstrated,' and 'the observed relations between zidovudine [AZT] and faster progression from seroconversion to death should not be interpreted as causal' – but they could be (see page 117)!

But the CDC, Ascher et al., Schechter et al. and their 'tricontinental' alliance were not the only AIDS epidemiologists who have failed to dissociate AIDS from drugs as the following titles of their articles reveal:

1. 1987: AIDS and intravenous drug use: the real heterosexual epidemic (Moss, 1987).
2. 1988: A larger spectrum of severe HIV-I-related disease in intravenous drug users in New York City (Stoneburner et al., 1988).
3. 1989: Cocaine abuse and acquired immunodeficiency syndrome: tale of two epidemics (Lerner, 1989).
4. 1991: The Twin Epidemics of Substance Use and HIV (National Commission on AIDS, July 1991).
5. 1991: AIDS, drugs of abuse and the immune system: a complex immunotoxicological network (Pillai et al., 1991).
6. 1993: Entangled epidemics: cocaine use and HIV disease (Larrat & Zierler, 1993).
7. 1993: Recreational drugs and sexual behavior in the Chicago MACS/CCS cohort of homosexually active men (Ostrow et al., 1993).
8. 1995: New picture of who will get AIDS is dominated by addicts (Kolata, 1995).
9. 1996: Clinical features of drug use and drug use related to HIV (Brettle, 1996).
10. 1996: Tuberculosis, AIDS, and death among substance abusers on Welfare in New York City (Friedman et al., 1996).
11. 1997: The influence of drug use patterns on the rate of CD4+ lymphocyte decline among HIV-I-infected injecting drug users (Lyles et al., 1997).
12. 1998: Immunodulation by nitrite inhalants may predispose abusers to AIDS and Kaposi's sarcoma (Soderberg, 1998).

In searching for a drug-free, homosexual AIDS patient, the AIDS reporter John Lauritsen interviewed Michael Callen, a founder of the People With AIDS Coalition (Lauritsen & Young, 1997). According to Lauritsen, 'possibly no one personally knew more persons with AIDS than Michael Callen.' Indeed, Callen reports in his book, *Surviving AIDS*, his over 3000 sexual contacts and extensive drug use before he adopted a monogamous lifestyle (Callen, 1990). Asked whether "he had ever encountered a gay male person with AIDS who did not fit the profile of venereal diseases, antibiotics, drugs..., his answer was: 'No. Not in eleven years. I have gone to a great deal of trouble to find these people. I found ten, who were saying in public, 'I only had one or two contacts,' ... [but] each one ended up telling me they had been lying ... in the support group setting, they would regale us with tales of bathhouses and promiscuity and lovers on the side and drug use'" (Lauritsen & Young, 1997). Thus, neither individual investigators,

nor the CDC, nor other government statistics have ever shown that, in the HIV era, the incidence of AIDS-defining diseases among non-drug-users has exceeded long-established, national backgrounds.

Nevertheless, a few American and European drug-free AIDS patients must exist based on the CDC's AIDS definition (see page 90 and 91). They would represent the normal incidence of the 30 AIDS-defining diseases among HIV-positive Americans and Europeans under the new name AIDS (World Health Organization, 1995b; Centers for Disease Control and Prevention, 1996).

### The drug-AIDS hypothesis

In view of the epidemiological evidence that (1) AIDS is compatible with a lifestyle, but totally incompatible with infectious disease, and (2) over 95% of all American and European AIDS patients are long-term users of drugs, we propose the following hypothesis that consistently explains and predicts American and European AIDS:

*The long-term consumption of recreational drugs, such as cocaine, heroin, nitrite inhalants, and amphetamines, and prescriptions of anti-HIV drugs, such as AZT, cause all AIDS diseases in America and Europe that exceed their long-established, national backgrounds, i.e. >95%. Chemically distinct drugs cause distinct AIDS-defining diseases; for example, nitrite inhalants cause Kaposi's sarcoma, cocaine causes weight loss, and AZT causes immunodeficiency, lymphoma, muscle atrophy, and dementia. Hemophilia-AIDS, transfusion-AIDS, and the extremely rare AIDS cases that appear in the non drug-using population reflect the normal incidence plus the AZT-induced incidence of these diseases under a new name.*

This hypothesis explains why AIDS is non-contagious. If correct, the hypothesis must also be able to (1) explain why AIDS occurred in the 1980s, and (2) why it is non-random with regard to sex, sexual orientation, and age, and (3) offer proof that drugs cause AIDS. In the following we show that the drug hypothesis meets all of these criteria.

### AIDS occurred in the '80s because it is a clinical subset of the contemporary American and European drug epidemics

A brief survey of recreational drug use in America and Europe reveals why AIDS is new and why it is non-random with regard to sex and age.

#### *Chronology of the drug epidemic in the US*

During and after the Vietnam War, and just prior to the AIDS epidemic, the number of regular users of illicit recreational drugs in America soared from a negligible background to a high of 25 million, or about 10% of Americans in the late '70s and '80s (Clinton & The White House, 1996). The Federal Bureau of Investigation (FBI) reported an estimated 937,400 state and local drug arrests for drug law violations in the US in 1987 (White House Office of National Drug Control Policy, 1998). And the *San Francisco Chronicle* reported in 1998 that 70% of the inmates in the federal prisons are drug offenders (Carman, 1998). According to the Bureau of Justice Statistics and historians (Jonnes, 1996), this sudden epidemic of drug addiction followed a 40 year period (from before World War II until the upsurge in the 1970s) during which there was very little illicit recreational drug use (Bureau of Justice Statistics, 1992).

Since its peak in the late 1980s, the epidemic has declined to an estimated 13 million regular users in 1996, or about 5% of the 260 million Americans (Los Angeles Times, 1998; White House Office of National Drug Control Policy, 1998). Likewise, spending on illegal drugs has declined from \$91 billion in 1988 to \$53.7 billion in 1995 (Associated Press, 1997).

Based on specific drugs, the epidemic breaks down into subepidemics of cocaine, heroin, amphetamines, and other drugs.

#### *Cocaine*

The director of the NIDA wrote in 1985 that, 'Over the past 10 years, cocaine ... has evolved from a relatively minor problem into a major public health threat' (Schuster, 1985) with a 'negligible' number of users in 1973 and 9,946 non-fatal and 580 fatal medical cases in 1985 (Kozel & Adams, 1986). The numbers of cocaine patients have since increased even more to 80,355 in 1990, and to a plateau of about 140,000 in the mid 1990s (Figure 1B and Table 4). In 1996, the number of regular cocaine users had reached 3.6 million, with 28 million who had at least tried the

Table 4. Chronology of diseases and death from illicit recreational drug use in the US<sup>1</sup>

Drug	event	1990	1991	1992	1993	1994	1995	1996
cocaine	death	–	2,938	3,285	3,633	3,687	–	–
	hospital	80,355	101,189	119,843	123,423	142,878	142,494	144,180
amphetamines	death	–	252	334	566	751	–	–
	hospital	8,800	7,363	10,615	15,630	17,665	17,547	16,787
heroin	death	–	2,260	2,782	3,558	3,522	–	–
	hospital	33,884	35,898	48,003	63,232	64,013	76,023	70,463
all drugs	death	–	6,246	6,870	7,602	8,541	9,216	–
	hospital	371,208	393,968	433,493	460,910	518,521	531,827	487,564

<sup>1</sup> (U.S. Department of Health & Human Services, 1993; U.S. Department of Health & Human Services, 1994; Drug Strategies, 1995; Office of National Drug Control Policy, 1996; US Department of Health & Human Services & Public Health Service & Substance Abuse and Mental Health Services Administration (SAMHSA), 1996; White House Office of National Drug Control Policy, 1998).

Table 5. Drugs confiscated in the USA<sup>1</sup> European Union, and Germany before and after the drug explosion ('Drogen-Explosion')<sup>2</sup>

Drug	USA		European Union	Germany	
	before	after		before	after
	(kilograms)				
cocaine	500 (1980)	100,000 (1990–96)	29,000 (1994)	0.09 (1963)	1,846 (1995)
heroin	–	1,500 (1990–95)	5,900 (1994)	1.8 (1968)	933 (1995)
amphetamines	–	–	1,900 (1994)	6.6 (1972)	138 (1995)
cannabis	–	–	733,400 (1994)	5.5 (1962)	14,245 (1995)
	(doses)				
amphetamines, ecstasy	2 million (1981)	97 million (1989)	1.25 million (1994)	730 (1987)	700,000 (1996)
LSD	...	–	61,000 (1994)	10 (1967)	71,069 (1995)

<sup>1</sup> (Flanagan & Maguire, 1989; Bureau of Justice Statistics, 1991; Duesberg, 1996b; Office of National Drug Control Policy, 1996; White House Office of National Drug Control Policy, 1998); <sup>2</sup> (Deutsche Hauptstelle gegen die Suchtgefahren, 1996; Springer Verlag, 1996; Holtermann, 1997); and Section 8.

drug once in their lifetime (Bureau of Justice Statistics, 1992; Office of National Drug Control Policy, 1996; McEvoy et al., 1998). Clearly, cocaine is the most popular illegal drug in America, accounting for \$38 billion of the \$53.7 billion spent on illegal drugs in 1995 (Associated Press, 1997; White House Office of National Drug Control Policy, 1998). Another measure of the popularity of the drug is that by 1997, 78% of all one-dollar bills in the US contained chemically detectable amounts of cocaine from using these bills for drug inhalation (New York Times, 1997).

From 1980 to 1990, imports of cocaine have increased two-hundredfold, from two tons to 400 tons, and have since been kept at this level (Figure 1B, Table 5) (Duesberg, 1992a; Clinton & The White House, 1996; Office of National Drug Control Policy, 1996). These data are based on cocaine seizures that increased from 500 kg in 1980 to 100 tons in

1990 and have since remained at this level (Flanagan & Maguire, 1989; Bureau of Justice Statistics, 1991; Office of National Drug Control Policy, 1996; White House Office of National Drug Control Policy, 1998), and on estimates that no more than 10-20% of the imported cocaine is confiscated (Anderson, 1987; Clinton & The White House, 1996; Associated Press, 1997). This corresponds to about 110 g for each of the 3.6 million regular users per year, which is rather close to the estimated daily consumption of 1g per day per addict (Schnoll et al., 1985; Volkow et al., 1997).

### Heroin

Between 1992 and 1995 about 1,500 kg of heroin were confiscated annually (Table 5) (Office of National Drug Control Policy, 1996). In view of these statistics, the *San Francisco Chronicle* warned in 1996 that 'a growing segment of the population [is] attracted

by its [heroin] deadly mystique and encouraged by its low prices ...' (Evenson & Whiting, 1996). According to the Drug Abuse Warning Network (DAWN) of the Department of Health and Human Services (HHS), heroin deaths climbed from 2,260 in 1991 to 3,522 in 1994 (Table 4). Heroin-related hospital emergencies increased over fivefold from 12,000 in 1978 to a plateau of about 70,000 in the mid 1990s (Table 4 and Figure 1B). The heroin epidemic accounted for \$10 billion of the \$53.7 billion spent on drugs in 1995 (Associated Press, 1997).

### *Amphetamines*

Based on US government statistics, the amounts of amphetamine confiscated have spiraled fiftyfold in the 1980s, from 2 million doses in 1981 to 97 million doses in 1989 (Table 5) (Flanagan & Maguire, 1989). According to the US Department of HHS, 'amphetamine-related emergency room episodes... [presenting with] violent paranoid behavior as well as stroke, seizure and death ...' (Drug Strategies, 1996) increased from 8,800 in 1990 to about 17,000 in the mid 1990s (Table 4). In California, amphetamine or 'speed' hospitalizations rose even faster: from 1,466 in 1984 to 10,167 in 1994 (Russell, 1996; Wallace, 1996).

It is apparent from the data shown in Figure 1 A, B that the chronology of the American drug epidemic closely paralleled that of the American AIDS epidemic.

### *Chronology of the drug epidemic in Europe*

In the 1970s, Europe was also hit by a drug explosion ('Drogen-Explosion') (Springer Verlag, 1996). For example, German consumption of cocaine, heroin, amphetamines, LSD, and cannabis increased one thousand to ten thousand fold from the 1960s to the 1990s based on the amounts confiscated by the Bundeskriminalamt (Table 5). These drugs are consumed by 2 million regular users, or 5% of the 80 million Germans, including 120,000 heroin addicts and 80,000 users of inhalants ('Schnueffelstoffe'), and 5.8 million who have tried drugs at some time in their life (Deutsche Hauptstelle gegen die Suchtgefahren, 1996; Springer Verlag, 1996). *The Lancet* confirmed that 'in the UK [cocaine] abuse has also reached epidemic proportions, with the abuse of Ecstasy in particular being associated with the 'rave culture'' (McEvoy et al., 1998).

Drug consumption by the combined European Union almost matches, and in the case of heroin, even exceeds the American epidemic, based on the amounts confiscated (Table 5). For example, 5.9 tons of heroin were confiscated in Europe in 1994, compared to 1.5 tons in the US. At the same time, 29 tons of cocaine, 1.9 tons of amphetamines, 733 tons of cannabis and 61,000 doses of LSD and 1.25 million doses of ecstasy were seized in the European Union in 1994 (Springer Verlag, 1996). In the first five months of 1995, 8.7 tons of heroin, 21 tons of cocaine, 769 tons of cannabis, and 1.87 tons of amphetamines were confiscated in Europe (Deutsche Hauptstelle gegen die Suchtgefahren, 1996).

The European drug epidemic chronologically paralleled the AIDS epidemic: there were 1312 drug deaths in 1981; 1424 in 1985; 4506 in 1990; and 4566 in 1995 (Figure 1C), (Deutsche Hauptstelle gegen die Suchtgefahren, 1996), and there were a few dozen AIDS cases in 1981; about 1000 in 1985; 12,000 in 1990; and about 20,000 in 1995 (Figure 1C), (World Health Organization, 1995b; World Health Organisation, 1996). Thus, both the European and American drug epidemics parallel the respective AIDS epidemics.

### **Both drug use and AIDS are non-random and spread non-exponentially**

#### *Sex and age of the AIDS epidemic match those of the drug epidemic*

*The National Drug Control Strategy: 1996* from the White House reports that 78% of the hard recreational drug users are males (Clinton & The White House, 1996). Earlier studies have reported similar non-random sex distributions, that is, that between 70 and 80% were males (Table 6). As in America, 66% of the German drug users are males (Deutsche Hauptstelle gegen die Suchtgefahren, 1996; Fietz, 1997). Thus, male drug users outnumber females between two and fiftyfold (e.g. nitrites, page 95) depending on the drug used.

All American consumers of hard recreational drugs such as cocaine, heroin, and amphetamines are over 18 and under 54 years of age (Table 6). *The National Drug Control Strategy: 1996* reports that 74% of the drug users are 21–44 years old (Clinton & The White House, 1996). Almost all drug decedents are also over 18 and under 54 years of age, and most are over 25

Table 6. Drug deaths and diseases in the USA by age and sex 1990–1994<sup>1</sup>

drugs		% male	% 18–54 years
cocaine	users	70, 82 <sup>2</sup>	99
	hospitals	67	
	deaths	80	
heroin	users	69, 83 <sup>2</sup>	99
	hospitals	71	
	deaths	83	
amphetamines	users	80	97
	hospitals	61	
	deaths	79	
nitrites	users	98 <sup>3</sup>	>98
	hospitals		
	deaths		
all combined	users	75, 78 <sup>4</sup> , 86 <sup>5</sup>	>75
	hospitals	71	
	deaths	77	

<sup>1</sup> (Bureau of Justice Statistics, 1992; U.S. Department of Health & Human Services, 1993; U.S. Department of Health & Human Services, 1994; Office of National Drug Control Policy, 1996)

<sup>2</sup> (Wesson & Smith, 1985)

<sup>3</sup> (San Francisco Department of Public Health & Lesbian & Gay Substance Abuse Planning Group, 1991; Ascher et al., 1993)

<sup>4</sup> (Clinton & The White House, 1996)

<sup>5</sup> (Bureau of Justice Statistics, 1988).

(Office of National Drug Control Policy, 1996) (Table 6). Germans who have used illegal drugs were also between 18 to 59 years old (*Deutsche Hauptstelle gegen die Suchtgefahren*, 1996). And 83% of the 15,000 first-time users in 1995 were over 21 years old (Springer Verlag, 1996).

A breakdown according to sexual persuasion is offered by Kim Gilliam, the drug addiction counselor from Washington, DC cited above: 'Addiction knows no boundaries, but in the gay community you find addicts of all ages. It's just not like that in the heterosexual population, where the addicts are generally younger' (Bergling, 1997). Indeed, according to a continental European investigation, the median age at death of mostly heterosexual, intravenous drug users is 30 years (Lockemann et al., 1995). The average age of a small group of English drug patients was 31 (McEvoy et al., 1998). Their American counterparts die between 25 and 44 years (Stoneburner et al., 1988; Hayes et al., 1994).

Thus, sex and age of those dying from drugs matches that of those dying from AIDS.

### *The dynamics of the AIDS and drug epidemics coincide*

The dynamics of the AIDS and drug epidemics also closely overlap as the following two examples show: (1) In 1997 the CDC reported that annual AIDS cases had continued to drop since 1993, with a 19% drop from 1996 (Centers for Disease Control and Prevention, 1997; Stolberg, 1997). The department gave the credit for the decline to the new anti-HIV drug cocktails (Centers for Disease Control and Prevention, 1997; Stolberg, 1997; Russel, 1998).

However, the CDC offered no evidence that dying AIDS patients were untreated and those on the new cocktails were the ones living and free of AIDS. On the contrary, the *New York Times* reported that AIDS patients died 'despite new AIDS drugs' (Stolberg, 1997), and the *San Francisco Examiner* reported that the cocktails had just begun to fail up to 50% of those treated (Krieger, 1997) (see page 121). Indeed a national survey just confirmed that all American AIDS patients are dying on anti-HIV treatments (Perlman, 1998). Moreover, the CDC failed to explain that their own AIDS statistics had started to decline in 1994, whereas the new anti-HIV cocktails were only introduced in 1996. This discrepancy was even pointed out by an AIDS correspondent from *Science* magazine (Cohen, 1997a). It follows that anti-HIV drugs are not a plausible explanation for the recent decline in AIDS cases.

The CDC also failed to consider information from another federal agency. The White House's *National Drug Control Strategy: 1998* reported that hard recreational drug use had declined from 25 million regular users in the 1980s to a low of 13 million in 1996, and spending for drugs had declined from \$91 billion in 1988 to \$53.7 billion in 1995 (see page 103) (*Los Angeles Times*, 1998; White House Office of National Drug Control Policy, 1998). Thus, the less than twofold decline of AIDS cases appears to be a product of two competing factors: (1) a drop in recreational drug use, and (2) a steady increase in the use of AIDS-causing anti-HIV drugs (see below).

(2) In an effort to close the 9:1 gender gap of its AIDS statistics, the CDC has reported since the early '90s that women had become the fastest-growing AIDS risk group (Centers for Disease Control, 1994), sometimes even at the cost of the truth (Bennett & Sharpe, 1996), suggesting each time that heterosexual 'transmission' was the cause. Again, the CDC failed to reconcile its publications with the fact that 'women

account for the fastest-growing population in jails and prisons, in large part because of drug offenses' (Drug Strategies, 1995).

Thus, the epidemiology and chronology of AIDS even mirrors the dynamics of the drug epidemic exactly, confirming the view that the AIDS epidemic is a clinical subset of the drug epidemic. To prove this we need to investigate whether drugs cause AIDS.

### Mechanisms of drug pathogenesis

Before we investigate whether drugs cause AIDS, it is helpful to consider mechanisms of drug pathogenesis. In view of the popular hypothesis that AIDS is caused by a virus, it is important to clarify that drugs differ from viruses as a cause of diseases in dose-dependence, and in time-dependence.

#### *Dose-dependence*

It was 500 years ago that Paracelsus von Hohenheim first defined the primary criterium of drug toxicity: *the dose makes the poison*. A pathogenic dose of recreational or medical drugs is typically achieved cumulatively by long-term consumption over many years. But it can also be achieved in a single application as an overdose.

By contrast, viruses and microbes are largely independent of the input concentration as causes of diseases, because viruses and microbes are self-replicating poisons. Once introduced into a susceptible host, they grow exponentially to pathogenic titers within days or weeks, provided they are not inhibited by antiviral immunity (Stewart, 1968; Fenner et al., 1974; Mims & White, 1984; Duesberg, 1996d; Duesberg, 1996c). Thus, viruses and most microbes are typically dose-independent pathogens.

#### *Time-dependence*

Because drug toxicity is dosage dependent, drugs typically take a long time to cause disease if taken at recreational or medical doses. This 'latent' or grace period is a function of the chemical toxicity and the concentration of the drug, and the ability of the body to repair drug damage over time. As a result, it takes decades of smoking before irreversible diseases such as lung cancer, emphysema, and heart disease occur, and it takes decades of alcoholism before the liver becomes cirrhotic. Likewise, it took months of

clinoquinol (Enterovioform<sup>®</sup>) prescriptions against intestinal parasites before blindness, termed severe myelo-optic neuropathy (SMON), occurred in Japan in the 1960s (Duesberg, 1996d). The carcinogenic effects of diethylstilbestrol prescriptions against premature delivery were also only discovered years after the prescriptions were taken (Pitot, 1986). Thus, drugs taken at recreational and medical doses are slow pathogens because their toxicity is dosage dependent, and microbes are fast pathogens because their toxicity is dosage independent.

Therefore, one would expect only a small fraction of drug users to develop diseases at a given time, namely those who have persisted in using drugs for the longest time. Statistics confirm this point. There are currently 50 million smokers in the US (Associated Press, 1995), but only 200,000 per year develop lung cancer (Parker et al., 1996). Many smokers never develop lung cancer because they discontinue to smoke, do not smoke enough, or die from other causes before they reach a critical threshold of toxicity.

On the basis of their interactions with the body, the recreational and anti-HIV drugs of AIDS patients fall into two categories, those that are (1) indirectly toxic by functioning catalytically, and those that are (2) cytotoxic, mutagenic, and carcinogenic via chemical reactions.

#### *Indirectly toxic drugs*

Cocaine, amphetamines, and heroin are indirectly toxic. All three function as catalysts of neurotropic reactions (Wilson et al., 1996; Volkow et al., 1997). Cocaine and heroin are natural compounds and amphetamines are synthetic adrenalinines that were used in Germany during World War II to suppress fatigue and anxiety in pilots and tank commanders (Weil & Rosen, 1983). A typical daily dose of 1-2 g of cocaine (Schnoll et al., 1985; Volkow et al., 1997) or heroin (Wesson & Smith, 1985) or amphetamine (Wilson et al., 1996) consists of about  $10^{21}$  molecules, or  $10^7$  molecules for every one of the  $10^{14}$  cells of the human body. At that concentration these catalysts are so active that recipients forget to eat, to drink, and to sleep, and lose many of the inhibitions that control undrugged life – the reason for their popularity and eventual pathogenicity.

Weight loss, malnutrition, and insomnia are consequences of drug-induced suppression of appetite and fatigue (Layon et al., 1984; Lerner, 1989; Pillai et al., 1991; Duesberg, 1992a; Larrat & Zierler, 1993;

Mientjes et al., 1993; Sadownick, 1994; Bergling, 1997). According to Maurice Seligmann and Anthony Fauci, now leading proponents of the HIV hypothesis in France and the US, malnutrition is the world's leading cause of immunodeficiency (Seligmann et al., 1984). These drug effects are compounded by poverty due to the enormous costs of illicit drugs. For example, an average cocaine habit of 1g per day costs \$800 per week (Schnoll et al., 1985).

*Cytotoxic, immunotoxic, mutagenic, and carcinogenic drugs*

Nitrite inhalants react with all biological macromolecules, mutating and inactivating DNA and RNA, diazotizing proteins, killing vitamins and oxidizing hemoglobin to inactive methemoglobin (Lauritsen & Wilson, 1986; Haverkos & Dougherty, 1988b; Duesberg, 1992a). As a result, they are cytotoxic and immunotoxic (Goedert et al., 1982; Haverkos & Dougherty, 1988b; Ratajczak et al., 1995), and able to function as carcinogens in animals and humans (National Research Council, 1982; Pitot, 1986). At the recreational dose of 1 ml per day (Dax et al., 1988; Haverkos & Dougherty, 1988b), the user introduces about  $10^{21}$  molecules into the lungs, or  $10^7$  molecules for every cell in the human body – enough for abundant toxicity. The cytotoxicity of nitrites on the epithelial cells of the lung is enhanced at least twofold by cigarette smoke, which also suppresses the immune system (Nieman et al., 1993).

A standard daily prescription of 0.5 g AZT, ddI, and other DNA chain-terminators as anti-HIV drugs corresponds to about  $10^{21}$  molecules per body, or  $10^7$  per human cell. If taken up, this is more than enough to kill growing cells by terminating DNA synthesis. The fast growing immune cells, red cells, and epithelial cells of the gut are especially vulnerable (Merck Research Laboratories, 1992; Chiu & Duesberg, 1995). Stopping the regeneration of these cells over several days causes anemia, lymphocytopenia, hepatitis, nausea, and wasting disease (Richman et al., 1987; Duesberg, 1992a; McLeod & Hammer, 1992; Freiman et al., 1993; Retrovir, 1994). AZT also prevents mitochondrial DNA synthesis in non-proliferating cells, causing muscle atrophy, hepatitis, and dementia (Duesberg, 1992a; Bacellar et al., 1994; Parker & Cheng, 1994; Chiu & Duesberg, 1995).

The National Cancer Institute (NCI) first reported in 1990 that AZT, at doses used for AIDS therapy, increases the annual lymphoma risk fifty-fold com-

pared to untreated controls (Pluda et al., 1990). Several unpublished studies by the NCI have confirmed AZT to be carcinogenic in mice (Table 8) (Cohen, 1987; Yarchoan & Broder, 1987; Cohen, 1997b). It is conceivable that such studies have remained unpublished until after AZT was licensed and firmly established because the Delaney Amendment prohibits the sale of drugs with carcinogenic potential in the US (Pitot, 1986). If AZT were confirmed to be carcinogenic, which seems to be the case, the NCI's first anti-HIV drug, and possibly all other DNA chain-terminators now licensed by the Food and Drug Administration, would be illegal (Yarchoan & Broder, 1987; Nussbaum, 1990; Duesberg, 1996d).

**Scientific, legislative, and non-scientific evidence that recreational drugs cause AIDS-defining and other diseases**

There are four elements of proof that recreational drugs cause AIDS-defining and other diseases: (1) long-term drug users develop diseases, (2) experimental animals become sick, (3) drug-specific diseases, and (4) patients recover from drug diseases if they stop taking drugs in time.

*Long-term drug users develop fatal diseases*

The first scientific paper on diseases caused by long-term morphine addiction was published in Paris, France, in 1909 (Achard et al., 1909). The paper reported immunodeficiency and several corresponding opportunistic infections as consequences of morphine addiction. Since then at least 63 other studies, summarized in Table 7, have confirmed that recreational drugs, including heroin, cocaine, amphetamines, and nitrite inhalants, cause AIDS-defining and other diseases. As a result of these diseases, and also of overdoses, intravenous drug users typically die at an average age of only 30 years from AIDS-defining, and other diseases – regardless of the presence of HIV (see Tables 4 & 7) (Stoneburner et al., 1988; Duesberg, 1992a; Hayes et al., 1994; Lockemann et al., 1995; Wilson et al., 1996; McEvoy et al., 1998; Baldwin et al., 1997).

Details of how some AIDS era-specific drugs, such as nitrites and amphetamines, cause diseases are briefly summarized:

1. The first five AIDS cases ever reported were male homosexuals with *Pneumocystis pneumonia* and



Table 7. Drug diseases diagnosed before the AIDS era, and in HIV-free addicts

Disease	Drugs used*	AIDS defining	References
immunodeficiency	C, H, N, A	YES	1
Kaposi's sarcoma	N	YES	2
candidiasis	C, H	YES	3
pncumonia	C, H, N	YES	4
lymphadcnopathy	C, H	YES	5
tuberculosis	C, H	YES	6
weight loss	C, H, A	YES	7
dementia, oncephalopathy	C, H, A	YES	8
diarrhea	C, H	YES	9
fever	C, H	YES	10
thrombocytopenia	C, H	YES	11
spontaneous abortion,	C, H	YES	12
premature birth, congenital abnormalities			
night sweats	C, H		13
impotence	C, H		14
severe atherosclerosis	A		15
tooth loss, caries	C, H		16
dermatitis	C, H		17
hepatitis	C, H		18
epileptic seizures	C, H		19
endocarditis	C, H		20
bronchitis	C, H		21

\* A, amphetamines; C, cocaine; H, heroin; N, nitrites.

- (Achard et al., 1909; Terry & Pellens, 1928; Briggs et al., 1967; Sapira, 1968; Harris & Garret, 1972; Geller & Stimmel, 1973; Pillari & Narus, 1973; Brown et al., 1974; Louria, 1974; McDonough et al., 1980; Gottlieb et al., 1981a; Marmor et al., 1982; Jaffe et al., 1983; Tubaro et al., 1983; Layon et al., 1984; Newell et al., 1985a; Newell et al., 1985b; Culver et al., 1987; Donahoe et al., 1987; Bureau of Justice Statistics, 1988; Haverkos & Dougherty, 1988b; Selwyn et al., 1988; Novick et al., 1989; Mientjes et al., 1991; Pillai et al., 1991; Larrat & Zierler, 1993; Mientjes et al., 1993; Sadownick, 1994; Ratajczak et al., 1995; Brettle, 1996; Baldwin et al., 1997)
- (Jaffe et al., 1983; Newell et al., 1984; Haverkos et al., 1985; Haverkos, 1988; Haverkos & Dougherty, 1988a; Archer et al., 1989; Friedman-Kien et al., 1990; Marquart et al., 1991; Safai et al., 1991)
- (Pillari & Narus, 1973; Stoneburner et al., 1988; Rogers et al., 1989)
- (Gottlieb et al., 1981a; Jaffe et al., 1983; Mathur-Wagh et al., 1984; Mathur-Wagh et al., 1985; Selwyn et al., 1988; Stoneburner et al., 1988; Ettinger & Albin, 1989; Mientjes et al., 1993; Hayes et al., 1994; Brettle, 1996)
- (Geller & Stimmel, 1973; Pillari & Narus, 1973; Espinoza et al., 1987; Des Jarlais et al., 1988; Brettle, 1996)
- (Firooznia et al., 1973; Courtwright, 1982; Layon et al., 1984; Stoneburner et al., 1988; Braun et al., 1989; Brudney & Dobkin, 1991; Hayes et al., 1994)
- (Pillari & Narus, 1973; Des Jarlais et al., 1988; Brettle, 1996; Bergling, 1997; McEvoy et al., 1998)
- (Stoneburner et al., 1988; Koch, 1990; Aylward et al., 1992; Larrat & Zierler, 1993; Hayes et al., 1994; Brettle, 1996; McEvoy et al., 1998)
- (Des Jarlais et al., 1988; Muñoz et al., 1992; Brettle, 1996)
- (Des Jarlais et al., 1988; Ettinger & Albin, 1989; Brettle, 1996)
- (Savona et al., 1985)
- (Fricker & Segal, 1978; Lifschitz et al., 1983; Alroomi et al., 1988; Rogers et al., 1989; Toufexis, 1991; Finnegan et al., 1992; Larrat & Zierler, 1993)
- (Des Jarlais et al., 1988; Brettle, 1996)
- (Larrat & Zierler, 1993; Brettle, 1996)
- (Wilson et al., 1996)
- (Pillari & Narus, 1973)
- (Pillari & Narus, 1973; Brettle, 1996)
- (Dismukes et al., 1968; Pillari & Narus, 1973; Layon et al., 1984)
- (Brettle, 1996)
- (Layon et al., 1984; Stoneburner et al., 1988; Mientjes et al., 1993; Brettle, 1996)
- (Ettinger & Albin, 1989; Lerner, 1989; Brettle, 1996)

cytomegalovirus infections who had all consumed nitrite inhalants (Gottlieb et al., 1981a). The report even cites nitrites as the possible cause of their diseases. HIV was not even a suspect because it was only discovered in 1983 (Barre-Sinoussi et al., 1983).

2. In 1985, Haverkos et al. from the CDC analyzed the AIDS risks of 87 male homosexual AIDS patients, 47 with Kaposi's sarcoma, 20 with pneumonia, and 20 with Kaposi's sarcoma plus pneumonia (Haverkos et al., 1985; Haverkos, 1988). All the men had used several sexual stimulants; 98% had used nitrites. Those with Kaposi's sarcomas reported two times more sexual partners and 4.4 times more receptive anal intercourse than those with only pneumonia. The median number of sexual partners in the year prior to the illness was 120 for those with Kaposi's and 22 for those with pneumonia only. The Kaposi's cases reported six-times more amyl nitrite and ethylchloride use, four times more barbiturate use, and two times more methaqualone, lysergic acid and cocaine use than those with pneumonia only. The authors concluded that the nitrites and other drugs had caused Kaposi's sarcoma because no statistically significant differences were found for sexually transmitted diseases among the patients.
3. A 4.5 year tracking study of 42 homosexual men with lymphadenopathy, but not AIDS, reported that eight had developed AIDS within 2.5 years (Mathur-Wagh et al., 1984) and 12 within 4.5 years of observation (Mathur-Wagh et al., 1985). All of these men had used nitrite inhalants and other recreational drugs, including amphetamines and cocaine, but they were not tested for HIV. The authors concluded that 'a history of heavy or moderate use of nitrite inhalant before study entry was predictive of ultimate progression to AIDS' (Mathur-Wagh et al., 1984).
4. Other studies also investigated the dose-response relationships between nitrites and AIDS: (i) one compared 20 homosexual AIDS patients to 40 AIDS-free controls (Marmor et al., 1982); (ii) another compared 31 patients to 29 controls (Newell et al., 1985b). Each study reported that multiple 'street drugs' were used as sexual stimulants and concluded that drugs were 94% to 100% consistent risk factors for AIDS (Newell et al., 1985b). Newell et al. derived a direct 'dose-response gradient': the higher the nitrite usage, the greater the risk for AIDS. The Kaposi response was estimated

to take a dose equivalent of 7 to 10 years of nitrite use (Newell et al., 1985a; Beral et al., 1990; Lifson et al., 1990; Duesberg, 1992a).

*Animals demonstrate that cocaine and nitrites cause AIDS-defining diseases*

1. Surprisingly, in view of the official disregard of the nitrite-AIDS hypothesis, the National Institutes of Environmental Health Sciences reported in 1995 that nitrite inhalants cause immunodeficiency in mice. Based on exposure of the animals to isobutyl nitrites (IBN) for 15 weeks, the Institute concluded that, 'in the absence of impaired pulmonary host defenses, IBN produces significant and partially reversible suppression of systemic humoral immunity' (Ratajczak et al., 1995). This conclusion directly contradicts that reached previously by the CDC in 1983 in exactly the same system, 'None of the animals exposed to IBN showed any evidence of immunotoxic reactions...', although 'thymic atrophy' was acknowledged (see page 101) (Centers for Disease Control, 1983).
2. In 1998, Lee Soderberg reviewed his experiments with mice showing that nitrite inhalants are 'depleting many cells of the immune system'. Going beyond the data of his experiments, Soderberg proposed that nitrites are a 'cofactor' of HIV in causing AIDS, because they 'stimulate HIV replication and can also stimulate the growth of Kaposi's sarcoma cells' (Soderberg, 1998). Clearly, both the popularity and fundability of investigations on the pathogenicity of nitrites are well served by involving HIV. But, considering that only one in 1000 T-cells that are lost in AIDS patients is latently infected by HIV, the cofactor hypothesis is biochemically unlikely. It may be for this reason that Soderberg did not mention a simple control of the nitrite-HIV cofactor hypothesis: Compare the immune system of a group of HIV-positive nitrite users to those of an otherwise matched HIV-free group.
3. An article entitled 'acute and chronic effects of cocaine on the immune system and the possible link to AIDS' points out in 1998 that 'human and animal studies document that cocaine alters the function of ... T-cells, neutrophils and macrophages.' In view of this, the authors propose a 'wide-ranging capacity for cocaine to suppress the immune system.' Again, cocaine is proposed to be just a 'cofactor' of HIV in the 'pathogenesis

of AIDS' without suggesting a control of the co-factor hypothesis by testing the effects of cocaine on HIV-free addicts (Baldwin et al., 1998).

4. Yet another review describes in 1998 the '*in vivo* effects of cocaine on immune cell function' (Pellegrino & Bayer, 1998). The article carefully avoids a decision whether cocaine is immunosuppressive on its own, but acknowledges that immune suppression in animals is dose-dependent. It proposes animal studies to investigate 'decreased immune responsiveness in cocaine addicts,' which is thought to be responsible for an increased risk of HIV infection. However, the risk of viral or microbial infection is independent of immune function, but the possible consequences of an infection are not. Again, the question whether the immunodeficiency diseases of cocaine addicts depend on HIV is not asked.

Despite their scientifically uncontrolled loyalty to HIV, each of the last three reviews confirm that nitrites and cocaine are at least 'cofactors' of immune deficiency in animals and man.

### Government statistics and legislation about drug diseases

The US government regularly confirms that recreational drugs cause diseases and death with emergency department and medical examiner statistics from the Office of the National Drug Control Policy and the DAWN. These statistics show that hundreds of thousands of patients are hospitalized annually for drug diseases and that several thousands die from drugs. In 1995 alone, 531,827 Americans were hospitalized for drug diseases and 9,216 died on drugs (Table 4). The European drug deaths have shot up from about 1000 per year in the mid-eighties to almost 6000 in the early '90s, and have declined slightly to about 5000 annual cases now (Figure 1C) (Deutsche Hauptstelle gegen die Suchtgefahren, 1996).

Governments have even confirmed the nitrite-AIDS hypothesis by legislation and convictions; for example, after publication of the NIDA monograph 'Health Hazards of Nitrite Inhalants' in 1988 (Haverkos & Dougherty, 1988b), the US Congress banned in 1990 the sale of nitrites with the Public Law (100-690) of the 'Crime Control Act' (Haverkos, 1990; Duesberg, 1992a; Hodgkinson, 1996). However, the nitrite ban is virtually unknown to the American public, and not a single violation is ever reported

by the press. In the summer of 1996, the Royal Pharmaceutical Society also banned the sale of nitrites in the UK citing: 'Our primary concerns were the health risks associated with the drug, including the suggestive links between poppers and Kaposi's sarcoma' (Pink Paper, 1996). In the same year, a Swiss court convicted a sex offender for popper use because poppers cause 'headache, arrhythmia, vertigo, fainting, paralysis, and unconsciousness' (Ineichen, 1996). But an official of the Swiss public health office, BGA, stated to the gay interest journal *aK* that it was not possible yet to predict the health effects of popper use ('noch keine Risikoabschätzung des Poppers-Gebrauchs möglich'), although he acknowledged that a man had just died after inhaling two grams of amyl nitrite (Ineichen, 1996).

### Non-scientific descriptions of how drugs cause diseases and death

The non-scientific AIDS literature provides independent evidence that recreational drugs cause AIDS and death. For example, a drug treatment specialist from St. Vincent's Hospital in New York has described amphetamine diseases: 'We are just starting to see heavy usage types in our emergency rooms in New York City. What's troubling about this drug isn't just the way it destroys the body – life expectancy for those intravenously injecting crystal is two years – but the bizarre psychotic symptoms that develop' (Sadownick, 1994). An official from AIDS Project Los Angeles, now director of an AIDS foundation in France, acknowledges the pathogenicity of amphetamines, although coded in HIV-jargon: 'there is ample evidence to suggest that crystal accelerates premature progression to full-blown AIDS in people dealing with HIV infection. Studies have shown that crystal eats T-cells for breakfast, lunch and dinner.' (Sadownick, 1994).

In 1997, *The Signorile Report on Gay Men: Sex, Drugs, Muscles, and the Passages of Life* describes medical consequences of amphetamine use:

In the long term, Ecstasy is possibly more damaging than any of the others – which are all pretty nasty to begin with. ... Ecstasy seems to kill off a significant part of the serotonin cells in your brain in large quantities, maybe for a long time, maybe forever. Crystal meth [also amphetamine] is the only other drug that in relatively small doses can also cause long-term changes in the brain. ... [The report quotes a psychiatrist of the Columbia

University in New York for further symptoms] ‘I have seen eating disorders, jaw-clenching, panic disorder, unusual bingeing disorder, flashbacks, cognitive deficits, derealization [out of touch with reality], sleep disorders, and chronic fears ... These symptoms might not show up for many years. ... well many of my colleagues are used to saying that all you need to do to eat up T-cells quickly is do lots of crystal’ (Signorile, 1997b).

Regarding steroids, the report warns of ‘danger ... on the brain,’ including ‘aggression, hypertension, the mythic ‘roid rages,’ violent sex, [and] shrinking testicles,’ and complains that ‘long-term effects of cosmetic steroid use, even at small doses, are simply not known. ... Studies have been inconclusive and few and far between’ (Signorile, 1997a; Signorile, 1997b).

One of the first observers to blame AIDS among male homosexuals on nitrites was Lauritsen (see 6), author of *Death rush, poppers and AIDS* (Lauritsen & Wilson, 1986). In 1993, Lauritsen says in *The AIDS War*:

Every Saturday night an estimated 2,000 gay men attend a dance club where drug consumption is the main activity. According to London sources, virtually 100 percent of the men are on drugs, from 3:00 in the morning, when the club opens, until it closes many hours later. Especially popular is a variety of Ecstasy, whose ingredients are claimed to include heroin. Poppers are sold legally in London [not since 1996]. No one seems to think they even count as drugs, as gay physicians, writing in the gay press, have said that poppers are harmless.

None of the major AIDS organizations have properly warned about the dangers of drugs. At most, their risk-reduction literature has urged people to use alcohol and drugs in moderation, so as not to affect the ‘judgment.’ Drugs are portrayed as risky only to the extent that they might facilitate a lapse into ‘unsafe sex.’ Poppers – which cause genes to mutate, which cause severe anemia, which can kill through heart attacks, which suppress the immune system – are depicted as bad only if they cause someone to forget condoms (Lauritsen, 1993).

Three years later, the British magazine *Gay Times* cites the concerns of a first aid officer from a London gay club:

I see some faces in the same dire state every week for years and I personally think there’s gonna be an awful lot of very ill people in a few year’s time.

Taking all these substances on such a regular basis cannot be good for you. Medically it can’t. Sooner or later, something’s got to give (Gibbons, 1996).

And an article in the *The Advocate* with the title ‘A deal with the devil’ asked philosophically in 1996:

So why is it that in the gay world, where almost half the urban male population is dead or sick from an epidemic closely associated with substance use, there is such ambivalence about drugs that AIDS organizations profess to see nothing wrong with raising money from events that glamorize drug use? Why, despite the bitter legacy of AIDS, do we continue assuring ourselves that being gay means we have to be totally non-judgmental about the very things that have wiped us out (Rotello, 1996)?

In the summer of 1997, the San Francisco based gay-interest magazine *Frontiers* directly addressed the long-term effects of methamphetamine addiction because they are only ‘begrudgingly and rarely discussed’:

Crystal is, at face value, a great bargain. At an estimated cost of \$40, it will create a ‘high’ that can last the entire weekend. It makes its user incredibly sexual, energetic, euphoric and confident. It is very easy to obtain (and equally easy to manufacture). In research studies using low doses of the drug in animals, scientists were able to induce epileptic-like seizures in the brain. Those frightening responses in animal studies have already occurred in humans. Remember Robert, the young television executive? ‘He was incredibly talented and headed for a major career,’ says a Southern California therapist under the pseudonym Dr. Clayton who treated dozens of crystal addicts during a 10-year period. ‘Robert started doing crystal at age 25, a few years after he’d begun his career ascent. He was handsome, worked out Monday through Friday, was blue-eyed, blond and tanned.’ Positive and asymptomatic for many years, Robert’s addiction to crystal began affecting his life. Everything outside of work revolved around the drug.

Dr. Clayton recalls one episode in which Robert missed his flight for the East Coast where he was headed for a major circuit party on Fire Island. Robert was so desperate to be there that he chartered a private jet. On another party weekend, this one in Key West, he had his first seizure and was hospitalized for three weeks. He began to go blind and ultimately died at his parents’ home

at the age of 32 last year. Doctors confirmed that crystal abuse caused his death (Zachary, 1997).

According to the above cited (page 97) drug counselor from Washington DC: ‘... there is a distinct line that travels from the bar to the bedroom to the hospital’ (Bergling, 1997).

#### *Drug-specific diseases*

The drug-AIDS hypothesis predicts that chemically distinct drugs cause distinct diseases. Indeed, the drug literature offers many examples, the most specific of these being the evidence that nitrite inhalants cause Kaposi’s sarcoma (Table 7).

#### *Kaposi’s sarcoma from nitrites*

Kaposi’s sarcoma is at least 20 times more common among homosexual than among non-homosexual AIDS patients (page 90) (Haverkos & Dougherty, 1988b; Beral et al., 1990; Chamberland & Curran, 1990). Based on the correlation between the new epidemic of homosexual Kaposi’s sarcomas and the almost exclusive use of nitrites by male homosexuals, it was originally proposed in the early 1980s that nitrites were the cause (Durack, 1981; Marmor et al., 1982; Newell et al., 1984; Haverkos et al., 1985; Newell et al., 1985b).

Moreover, the nitrite-Kaposi hypothesis is directly supported by clinical distinctions between the Kaposi’s of the AIDS era and those originally described by Moritz Kaposi in the last century (Kaposi, 1872). The ‘HIV-associated’ sarcomas are ‘aggressive and life-threatening’ (Sloand et al., 1993), and fatal within 8-10 months after diagnosis (Meduri et al., 1986; Garay et al., 1987; Gill et al., 1989; Irwin & Kaplan, 1993). In contrast, the classic ‘indolent and chronic’ Kaposi’s sarcomas hardly progress over many years (Meduri et al., 1986; Drotman & Haverkos, 1992; Cohen, 1994a). Further, Kaposi had diagnosed sarcomas only on the skin, mostly of the lower extremities, but up to 32% of the Kaposi’s sarcomas of homosexual men are lung cancers (Gill et al., 1989; Irwin & Kaplan, 1993). Pulmonary Kaposi’s sarcoma had never been observed by Moritz Kaposi in the last century, nor by anyone else prior to the AIDS epidemic (Kaposi, 1872). Because the lungs are the primary site of exposure to nitrite inhalants, pulmonary Kaposi’s sarcoma is further support for the nitrite-Kaposi hypothesis.

Sensing the clinical novelty, Meduri et al. point out that the ‘pulmonary involvement by the neo-

plasma has been an unusual clinical finding’ compared to all ‘classic’ Kaposi’s sarcomas (Meduri et al., 1986). Nevertheless, the distinction between classic and AIDS-Kaposi’s sarcoma is rarely acknowledged and may escape many observers due to the ‘difficulty in pre-mortem diagnosis’, and because ‘pulmonary Kaposi’s sarcoma was indistinguishable from opportunistic pneumonia...’ (Garay et al., 1987).

#### *Intracerebral haemorrhage and AIDS from cocaine and amphetamines*

According to a study published in *The Lancet*, ‘it has been recognised for many years that abuse of amphetamine, cocaine, or Ecstasy, can give rise to intracerebral haemorrhage (ICH)...’ In addition, the study points out that, ‘despite their young age, the mortality and morbidity of patients presenting with ICH following substance abuse appears to be substantially greater than similar patients without this aetiological factor... presenting in a dehydrated state and often have serious underlying illness such as AIDS and malnutrition’ (McEvoy et al., 1998).

#### *AIDS patients recover if they stop taking drugs*

Curing a disease with an anti-microbial vaccine or antibiotic is considered one of the strongest elements of proof that the respective disease was caused by a microbe. Likewise, a toxic drug is proved to be a cause of a disease if patients recover once the drug is removed. For example, if termination of smoking stops bronchitis, tobacco smoke is proved to cause that disease. Several examples of recovery from AIDS-defining diseases by discontinuation of drug use have also been recorded:

1. In France, 71 babies, who shared intravenous drugs with their mothers prior to birth, were born with immunodeficiency and AIDS-defining, as well as other diseases, and were HIV antibody-positive (Blanche et al., 1989; Blanche et al., 1994). However, one to two years after birth and after discontinuation of the maternal recreational drug supply, 61 had recovered. Their T-cell levels had even gone up to normal, despite the continued presence of HIV. Some of the 10 who did not recover were among the first to be treated with AZT.
2. A collaborative study from Europe reports that 60% of babies born to drug-addicted mothers recovered from pneumonia, bacterial infections, candidiasis, and cryptosporidial infection and

were healthy at 6 years of age, despite the presence of HIV (The European Collaborative Study, 1994). About 40% of the HIV-positive children died — exactly the percentage that was ‘treated with zidovudine [AZT]’; 10% before 6 months of age and 40% by 4 years.

Although this study of pediatric AIDS does not even mention maternal drug use (focusing instead only on HIV), earlier reports from the European Collaborative Study group did. One of these documented in 1987 that ‘nearly all children were born to mothers who are intravenous drug users’ (Mok et al., 1987). In 1991, the European Collaborative Study group reported that 80% of the children with pediatric AIDS were born to mothers who were intravenous drug users (page 97, 98) (European Collaborative Study, 1991).

3. The risk of AIDS-defining diseases to HIV-positive intravenous drug users dropped fourfold after giving up drug injection for treatment with synthetic methadone. Only 5% (5/93) of those on methadone, compared to 19% (23/124) of those who continued illicit drugs, developed such diseases over 16 months of observation (Weber et al., 1990). Since methadone is immunosuppressive (Klimas et al., 1991), the difference could have been even bigger if no drugs had been used.
4. The T-cell counts of HIV-positive intravenous drug users from New York had stabilized after they had stopped injecting intravenous drugs. By contrast, that of controls, who continued drug injection, dropped 35% over the 9 months of observation (Des Jarlais et al., 1987).
5. The T-cells of 29% of 1,020 HIV-positive male homosexuals and intravenous drug users in the placebo arm of an AZT trial increased over the span of two years, despite the continued presence of HIV (Hughes et al., 1994). The probable reason is that those whose T-cells increased had given up or reduced immunosuppressive recreational drug use under the clinical surveillance of the drug trial.

*Conclusion: recreational drugs cause AIDS-defining and other diseases*

In sum, three lines of evidence show that long-term consumption of recreational drugs causes AIDS-defining and other diseases: (1) 63 scientific publications, government statistics and legislation, and case reports from the non-professional literature document drug diseases, (2) the identity of the disease is de-

termined by the chemistry of the drug, (3) disease is cured if drug use is stopped in time.

**Evidence from scientists, manufacturers, and the press show that anti-HIV drugs cause AIDS-defining and other diseases**

AZT and other DNA chain-terminators, prescribed as anti-HIV drugs, were first licensed in 1987 as reported by *Science* under the ominous title ‘Imminent marketing of AZT raises problems; marrow suppression hampers AZT use in AIDS victims’ (Kolata, 1987). Indeed, AZT was synthesized over 30 years ago for cancer chemotherapy, long before AIDS was known (Horwitz et al., 1964). Because the operating principle of cancer chemotherapy is to kill growing human cells by terminating cellular DNA synthesis at micromolar concentrations, AZT was predictably pathogenic. To hide the emerging tragedy, HIV/AIDS doctors have tried to trivialize or rationalize the fatal toxicity of their drugs.

*Diseases caused in animals by AZT and other anti-HIV drugs*

Years after AZT was safely licensed and established as an anti-HIV drug, or anti-AIDS drug, in 1987, the first animal experiments on its pathogenic properties were published. Contrary to the antecedent human experiments, the animal experiments all appeared in specialized journals (Table 8). All of these animal tests demonstrate that DNA chain termination causes AIDS-defining and other fatal diseases in a dose dependent fashion. According to McKallip et al., AZT causes immunodeficiency in mice even after short-term treatment of one to two weeks: ‘It was observed that the thymus was highly sensitive to AZT treatment. Also, AZT when present at the time of T-cell differentiation was found to play a critical role in inducing immunosuppression’ (Table 8) (Benedict, 1972). On this basis they warn ‘that AZT may affect the immune response to HIV antigens’ rather than function as an anti-HIV drug. Another study points out that ‘AZT [treatment] has a potent[ial] to transit into leukemias’ (Table 8) (Inoue et al., 1997).

According to several studies, the pathogenic effects of short-term AZT treatment of several weeks on adult animals are largely reversible (see references in Table 8). However, one also reports on death from anti-HIV drugs. Combined treatment with ddC and

Table 8. Diseases caused by AZT in animals

Animal	dose	Diseases		Ref
		AIDS-defining	Other	
<i>adult:</i>				
rat, mouse	1 mg/ml H <sub>2</sub> O 7wks	neutropenia lymphopenia thrombocytopenia bone marrow depletion weight loss	anemia	1
mouse	0.5–1 mg/ml H <sub>2</sub> O 7 wks	thrombocytopenia myelodysplasia	anemia leukemia	2
mouse	500 mg/kg 7–14 days	T-cell depletion thymic atrophy		3
mouse	400 mg/kg 17 days	lymphotoxicity	anemia nephrotoxicity mitochondrial necrosis	4
mouse, rat, dog, monkey	18–250 mg/kg 2–52 wks	cytostatic leukopenia	anemia	5
mouse, rat	500 mg/kg + 500mg/kg ddC 29 days	lymphocytopenia, thrombocytopenia, marrow depletion, death of 25/30 mice	'nonregenerative' anemia	6
<i>embryo:</i>				
mouse	12–25 mg/day 12–18 days of gestation		cancer: lung liver	7
monkey	10 mg/day last 10 wks of gestation		vaginal	
mouse	1 μM		preimplantation: lethal postimplantation: retarded development	8

1) (Cronkite & Bullis, 1990), 2) (Inoue et al., 1997), 3) (McKallip et al., 1995), 4) (Omar et al., 1996), 5) (Ayers et al., 1996), 6) (Thompson et al., 1991), 7) (Olivero et al., 1997), 8) (Toltzis et al., 1993)

AZT – as is now the standard for cocktails including protease inhibitors – at 500 mg/kg each for 29 days killed 25 out of 30 mice (Thompson et al., 1991). The survivors suffered severe 'nonregenerative' anemia, lymphocytopenia, and thrombocytopenia (Table 8). On this basis, the authors warned, 'The overall similarities in hematopoietic effects between rodents and human beings, especially for AZT, indicate that these studies can and do provide important information relevant to the toxicity of these drugs.'

'Because of widespread projected use of AZT in human pregnancy,' the effects of AZT on animal embryos are also relevant (pages 99, 118, 120) (Olivero et al., 1997). These effects fall into two groups: (1) Preimplantation embryos are killed by AZT at 1 μM, which is only about 1/20 of the concentration of the

daily dose of 500 mg prescribed to a pregnant mother in the last two trimesters (Connor et al., 1994). (2) AZT treatment of postimplantation embryos results in retarded development and cancer (Table 8).

Based on these results, Olivero et al. conclude that 'AZT is unequivocally a transplacental genotoxin and carcinogen.... When given transplacentally to mice, benzo[a]pyrene produced lung and liver tumor multiplicities similar to those observed [with AZT]' (Olivero et al., 1997). Therefore, the authors warn: 'our results suggest that the current practice of treating HIV-1-positive women and their infants with high doses of AZT could increase cancer risk in the drug-exposed children when they reach young adulthood or middle age' (Olivero et al., 1997).

With regard to the protease inhibitors that were licensed in 1996, the Merck Company published in 1997 that a coded 'HIV protease inhibitor' is 'rapidly' hepatotoxic in dogs and rats 30 min after administration. Histological examination revealed single cell necrosis (Grossman et al., 1997).

#### *Diseases caused by anti-HIV drugs in humans*

In the following, we analyze some of the few placebo-controlled AZT-licensing studies in which the occurrence of diseases in AZT-treated persons was compared to that in untreated HIV-positive control groups. Because of the perceived benefits that AZT was said to show in the licensing studies, it became unethical to use untreated control groups after the licensing of AZT. Therefore, most recent studies are uncontrolled. Some of these uncontrolled studies are also analyzed here based on what is generally or specifically known about untreated HIV-positive people.

Reviews of these studies indicate that the recreational drug use of AZT recipients and controls is never reported, probably because HIV is thought to be the only cause of their ailments. These reviews also suggest that there is a hierarchy of openness toward AZT toxicity in AIDS papers: AZT is accepted as a red cell killer, causing anemia. It is less readily accepted as a killer of neutrophils, because neutropenia is immunodeficiency which AZT is said to prevent. AZT is least likely to be described as a lymphocyte or even T-cell killer, because these are the presumed targets of the hypothetical T-cell killer HIV, which AZT is supposed to inhibit. Therefore, evidence for lymphocytopenia and T-cell depletion is usually not mentioned or is hidden in the raw data of AIDS papers. The following analyses of AZT toxicity were made with these experiences in mind:

1. The licensing study of AZT, sponsored by the NCI and Burroughs Wellcome, the manufacturer of the drug, investigated 289 patients from AIDS risk groups with prior *Pneumocystis pneumonia* and 'unexplained' weight loss, fever, oral candidiasis, night sweats, herpes zoster, and diarrhea (Fischl et al., 1987; Richman et al., 1987). All but 13 of these patients were males, but there was no information about their sexual orientation or recreational drug use. The study was planned for six months but was interrupted after four months because by then the therapeutic benefits of AZT were claimed to be too obvious to continue the placebo control. By that time only 1 out of 145 in the AZT group, but 19

out of 137 in the placebo group, had died. Therefore the study concluded that AZT can 'decrease mortality'.

However, during the same time period the lymphocyte counts had decreased over 50% in 34% of the AZT recipients, but in only 6% of the control group. In the AZT group, 66 suffered from severe nausea, compared to only 25 in the control group; muscle atrophy was observed in 11 AZT recipients but in only 3 from the control group. Moreover, 30 out of the 145 in the AZT group depended on multiple transfusions to survive anemia, compared to only 5 out of the 137 in the placebo group. Thus, the number of subjects in the AZT group who would have died from severe anemia if untreated was larger (i.e., 30) than the AIDS deaths and anemias of the control group combined, namely 19 + 5.

The 'decreased mortality' claim is further compromised by numerous 'concomitant medications' other than transfusions for AZT-specific diseases and failure to match the AZT and placebo groups for the cumulative effects of prior and parallel recreational drug use. In addition, some of the AZT-specific AIDS diseases observed in the placebo group appear to be due to patient-initiated 'drug sharing' between AZT and placebo recipients (Lauritsen, 1990; Duesberg, 1992b; Freestone, 1992) and falsification of the case report forms (Lauritsen, 1992; Hodgkinson, 1996).

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

The possibility that recreational drugs were the cause of AIDS, in which case anti-viral AZT would only be a fatal poison – like chemotherapy without cancer – was not even mentioned by the many authors of the AZT study. However, one of them, Durack, was the first to propose in 1981 that 'recreational drugs [are] immunosuppressive' (Durack, 1981). Another antiviral therapist, Donna Mildvan, had just published that 'a history of heavy or moderate use of nitrite inhalant before study entry was predictive of ultimate progression to AIDS' (Mathur-Wagh et al., 1984), and that



- the T-cells of intravenous drug users dropped in proportion to drug use (Des Jarlais et al., 1987). Yet another anti-HIV therapist, Michael Gottlieb, had reported in 1981 that all of five homosexual *Pneumocystis pneumonia* patients, later described as the first AIDS patients ever diagnosed, had used nitrite inhalants (Gottlieb et al., 1981a).
2. In 1994, the British-French Concorde trial, the largest controlled study of its kind, including 1749 subjects compared the onset of AIDS and death in AZT-treated, healthy, HIV-positive male homosexuals to untreated controls. It found that AZT is unable to prevent AIDS and increases the mortality by 25% compared to the untreated controls. The conclusion was: 'The results of Concorde do not encourage the early use of zidovudine [AZT] in symptom-free HIV-infected adults' (Seligmann et al., 1994).
  3. An independent British study found that AZT prophylaxis of mostly male homosexuals reduced survival from three to two years and also observed AZT-specific AIDS diseases such as 'wasting syndrome, cryptosporidiosis, and cytomegalovirus infection ... almost exclusively' in AZT-treated AIDS patients (Poznansky et al., 1995). This result confirmed Concorde's observation, in particular the 25% higher mortality of those on AZT. The authors' interpretation: 'The study of patients who progress from primary HIV infection to AIDS without receiving medical intervention gives insights into the effects of medical intervention on presentation and survival after developing an AIDS-defining illness' (Poznansky et al., 1995). But the nature of these 'insights' was not revealed.
  4. An analysis of American homosexual men from the MAC study revealed that AZT treatment increased the risk of pneumonia two to fourfold (Saah et al., 1995). The AIDS researchers explain their observation as follows: 'Zidovudine was no longer significant after T-helper lymphocyte count was considered, primarily because non users had higher cell counts...' (Saah et al., 1995). The fact that an inhibitor of DNA synthesis, designed to kill human cells, would reduce lymphocyte counts was not considered.
  5. According to HIV/AIDS researchers James Goedert et al. from the NIH, AIDS prophylaxis by AZT also proved to be negative for American hemophiliacs. The AIDS risk of hemophiliacs on AZT was 4.5 times higher and their mortality was 2.7 times higher than that of untreated controls (Goedert et al., 1994). The authors' interpretation of the increased morbidity and mortality was that this was 'probably because zidovudine was administered first to those whom clinicians considered to be at highest risk' (Goedert et al., 1994). In other words, 'clinicians' would be able to recognize hemophiliacs in which HIV would strike first and prescribed AZT accordingly. AZT-specific mortality of homosexual AIDS patients has been excused with the same argument (see 6) (Veugeliers et al., 1994).
  6. Since the licensing of AZT in 1987, the mortality of British HIV-positive hemophiliacs has increased tenfold (Darby et al., 1995) and that of American hemophiliacs has increased sharply (Chorba et al., 1994). This has been blamed on HIV (Chorba et al., 1994; Darby et al., 1995; Horton, 1995; Maddox, 1995). However, the coincidence that the mortality of hemophiliacs started to increase in exactly the same year they began taking AZT suggests that AZT was responsible (Duesberg, 1995c; Duesberg, 1995d). In view of this, Darby et al. acknowledge that 'treatment, by prophylaxis against *Pneumocystis carinii* pneumonia or with zidovudine [AZT] has been widespread' in HIV-positive hemophiliacs, and that 'HIV-associated mortality has not been halted by these treatments' (Darby et al., 1995). Challenged for an explanation by a published commentary (Duesberg, 1996a), the British hemophilia researchers Sabin et al. offered the same explanation as Goedert and Veugliers et al: 'Observational studies often show that patients given zidovudine have a worse prognosis than untreated patients. Patients receiving zidovudine are selectively treated because they are ill. The interpretation of findings from these studies should not therefore be that zidovudine increases the risk of AIDS' (Sabin et al., 1996). 'Should not,' but could.
  7. An American study of intravenous drug users observed in 1993 that 'the rate of CD4 lymphocyte depletion did not appear to slow after the initiation of zidovudine therapy ... [and] results suggested that zidovudine users in this sample may have experienced more rapid CD4+ cell depletion' (Alcibes et al., 1993). The authors' interpretation was that 'The results of this analysis provide evidence for a mechanism by which the clinical factors that predict more rapid progression to AIDS, such as bacterial infection, might work, and why other factors, such as drug injection, are unrelated to

AIDS risk' (Alcabes et al., 1993). But no control is offered for drug-free AIDS.

8. In 1994, it was reported that among 104 pregnant women treated with AZT, eight aborted spontaneously, eight were aborted 'therapeutically', and another eight delivered babies with serious birth defects, including cavities in the chest, abnormal indentations at the base of the spine, misplaced ears, triangular faces, heart defects, extra digits, and albinism (Kumar et al., 1994). The conclusion of the authors was that their results were 'not proving safety, thus lending tenuous support to the use of this drug' (Kumar et al., 1994). However, 'spontaneous' or therapeutic abortion as a result of AZT was not an unforeseeable accident. A review in *The Lancet* on 'non-surgical abortion' documents that chemotherapeutic drugs, like methotrexate, have been used to abort normal and ectopic pregnancies since 1952 (Potts, 1995). The article concedes 'early concerns over teratogenicity,' but concludes: 'used correctly, the method could bring great benefits' (Potts, 1995). Moreover, abortion of developing mouse embryos by AZT had been published a year before HIV-positive, pregnant women were treated with AZT (see Table 8) (Centers for Disease Control and Prevention, 1997).
9. In 1996, a study from the American National Institute of Child Health and Human Development concluded that AIDS prophylaxis with AZT also harms children: 'In contrast with anecdotal clinical observations and other studies indicating that zidovudine favorably influences weight-growth rates, our analysis suggests the opposite' (Moye et al., 1996).
10. An American study on AIDS prophylaxis of 107 three-month to six-year-old HIV-positive children with AZT reported depletion of 350 out of an average of 1044 T-cells per year, and neutropenia of less than 400 cells per microliter in 20%. Since the study did not include an untreated control, it is not directly obvious whether AZT or HIV was the cause (Kline et al., 1998). However, because HIV is not known to infect neutrophils and the children were free of neutropenia prior to AZT, the authors concluded that the neutropenia was due to AZT. In view of this, they warned that 'neutropenia of the magnitude observed in the present study (<400/ $\mu$ L) can be an important limitation to the success of antiretroviral therapy, requiring temporary or even permanent discontinuation of an otherwise effective agent.'
- By contrast, the authors blamed the dramatic loss of T-cells on natural, age-dependent reduction: 'As expected in this population of young children, median absolute CD4+ lymphocyte counts decreased ....' However, it is unlikely that children naturally lose 35% of their T-cells per year. It is also unlikely that the hypothetical T-cell killer HIV was the reason for the T-cell depletion because the study included up to six year-old children with perinatally acquired HIV whose T-cell counts averaged 1044 per microliter before AZT treatment. Thus, AZT remains the only plausible cause for the T-cell depletion in the AZT-treated children.
11. The damage caused by AZT prescriptions is compounded by many of the concomitant medicines taken by most, if not all, HIV-positive Americans with AIDS and at risk for AIDS (Table 3). For example, some of the antiviral drugs such as ganciclovir and acyclovir are also DNA chain terminators that are nearly as toxic as AZT (Douglas, 1990). As expected, combination therapy of ganciclovir and AZT produces 'pancytopenia' (Jacobson et al., 1988) by killing hemopoietic cells and has 'direct [toxic] effects on myeloid and erythroid progenitor cells' (Fogelman et al., 1994; Retrovir, 1994). Thus, DNA chain-terminators cause 'immunodeficiency.'
12. Anti-HIV/AIDS polypharmacies have 'nephrotoxic, cytotoxic, and myelosuppressive [effects], such as amphotericin B, co-trimoxazole, dapsone, interferon, pentamidine, vincristine, flucytosine, adriamycin, vinblastine, and others [which] could potentially increase the risk of hematologic toxicities in patients being treated with ZDV [AZT]' (Fogelman et al., 1994). In other words, these drugs are immunosuppressive because they intoxicate and kill immune cells.
13. Three articles in *The Lancet* describe 'abnormal fat accumulation in patients with HIV-1 infection' (Lipsky, 1998) who were treated with HIV protease inhibitors. These accumulations have been termed 'buffalo humps' and 'Crix belly' in various studies (Lipsky, 1998; Lo, 1998; Miller et al., 1998). One of these studies showed a direct dose response relationship between the lengths of protease inhibition and fat accumulation (Miller et al., 1998).

### *AIDS diseases caused by blocking mitochondrial DNA with AZT*

By terminating mitochondrial DNA synthesis, AZT can also destroy non-growing cells, such as neurons and muscle cells (Parker & Cheng, 1994). This process generates muscle atrophy (Richman et al., 1987; Bessen et al., 1988; Gorard & Guilodd, 1988; Helbert et al., 1988; Dalakas et al., 1990; Hitchcock, 1991; Yarchoan et al., 1991) and dementia (Smothers, 1991). Several examples illustrate this point:

1. The American MAC study of 5000 male homosexual men observed that 'HIV dementia among those reporting any antiretroviral use (AZT, ddI, ddC, or d4T) was 97% higher than among those not using this antiretroviral therapy' (Bacellar et al., 1994). The result is interpreted by its authors with little concern for percentages: 'This effect was not statistically significant' (Bacellar et al., 1994).
2. Two weeks after discontinuing AZT, four out of five AIDS patients recovered from myopathy (Till & MacDonnell, 1990).
3. Anecdotal cases of muscle atrophy and general wasting from AZT and other DNA terminators confirm these results: (i) After years on and off AZT, the ballet star Rudolf Nureyev was unable to stand up for ovations for his new production of *La Bayadere* at the Palais Garnier shortly before his death at 54 years of age in Paris on January 6, 1993 (Lauritsen, 1993). (ii) After several years on AZT and ddI, the tennis star Arthur Ashe had wasted to a skeleton and passed away at 49 years of age in New York in 1993 (Ashe & Rampersad, 1993; Duesberg, 1996d). (iii) After less than a year on AZT, Kimberly Bergalis, who was famous for the hypothesis that her HIV was derived from her dentist, lost over 30 pounds and required a wheelchair because of muscle atrophy prior to her death at 23 years of age (Duesberg, 1996d).

In view of this, it may not be just a coincidence that the CDC added dementia and wasting from muscle atrophy to its list of AIDS-defining diseases in 1987, just when AZT was first prescribed to hundreds of thousands of HIV-positive people (Centers for Disease Control, 1987; Kline et al., 1998).

### *Anti-HIV drug patients recover after they stop taking drugs*

1. Ten out of 11 HIV-positive, AZT-treated AIDS patients recovered cellular immunity after discontinuing AZT in favor of an experimental vaccine (Scolaro et al., 1991).

2. Three of four AIDS patients recovered from severe pancytopenia and bone marrow aplasia four to five weeks after AZT was discontinued (Gill et al., 1987).
3. After treatment with protease inhibitors for only 4 to 10 days at 800 mg every 8 hrs in conjunction with 6 to 12 other anti-HIV/AIDS drugs, three HIV-positive AIDS patients developed severe hepatitis, jaundice, nausea, vomiting, and abdominal pain (Braeu et al., 1997). Two weeks after discontinuation of the protease inhibitors, two of the three AIDS patients had recovered (Braeu et al., 1997).
4. Five HIV-positive patients were hospitalized one to three weeks after starting Indinavir (protease inhibitor) treatment with fever and AIDS-defining mycobacterial infection (see Table 1). These symptoms disappeared either after Indinavir was stopped or upon additional medication. In one of these patients, the symptoms reappeared after starting Indinavir again and disappeared again after stopping the drug (Racc et al., 1998).
5. Another 'good example that medicines hurt more than they help' is the story of Roger Cobb, co-chairman of the consumer caucus for the Commission on AIDS Care, Service and Treatment for Philadelphia and Nine Surrounding Counties:

'Sixty days after I started substance abuse treatment, I learned that I was HIV-positive,' recalls Cobb, who had used crack and cocaine, among a smorgasbord of other drugs, for more than 21 years. 'A little while later I started treatment with AZT for about 14 months.' It was during this time that he developed what he calls 'the look.' 'I had the sunken face, the ashy skin; I lost weight – everything. Against my doctor's advice, I decided AZT was not for me, so I decided to try something else.' And 'the look'? 'The look is fabulous now,' says the 40-year-old, who is working on his master's degree in social work. 'I'm back to me', (Freeman, 1996).

6. After AZT treatment for about six months prior to birth and 1.5 months after birth because of maternal HIV infection (pages 99, 118) (Connor et al., 1994), two HIV-free babies developed AIDS-defining Pneumocystis pneumonia and high fever at the age of two months (Heresi et al., 1997). Because both babies were HIV-free, there was

no further AZT treatment. Both babies fully recovered and remained healthy for the period of observation of over a year. The authors entitled their report 'Pneumocystis pneumonia in infants... exposed to HIV but...not infected: an exception to the AIDS Surveillance case definition,' but acknowledged in the text that 'it is unclear if ... zidovudine treatment set the stage for PCP.' Had the babies had antibodies to HIV, they would have been AZT-treated to their deaths.

*The medical orthodoxy and the press, confirm that anti-HIV drugs cause AIDS-defining and other diseases – but only informally*

*The medical orthodoxy*

Recently, some AIDS clinicians have openly registered concerns about the medical consequences of anti-HIV treatments, although not in the form of dedicated articles. Says Jay Levy, professor of medicine at the University of California at San Francisco (UCSF), 'With all the hoopla about antiviral drugs, and you get any virologist aside and they'll say this is not how we are going to win, it's high time we look at the immune system' (Pennisi & Cohen, 1996). In 1998 Levy confirmed this view, and even pointed out that all 'long-term survivors of HIV' are those who did not take the anti-HIV drugs (Levy, 1998).

Lecturing his students, another professor of medicine at UCSF, Donald Abrams, is even more direct, according to a university magazine:

'In contrast with many of my colleagues at SFGH [San Francisco General Hospital] in the AIDS program, I am not necessarily a cheerleader for anti-retroviral therapy. I have been one of the people who's questioned, from the beginning, whether or not we're really making an impact with HIV drugs and, if we are making an impact, if it's going in the right direction.

Despite the promising evidence, definitive proof of protease inhibitors' efficacy can be provided only by randomized clinical trials with placebo. Because new antiviral drugs are continuously being developed, conducting such trials is virtually impossible due to the reluctance of patients to continue treatment with an 'old' drug.' Abrams spent the first half of his lecture describing analogous problems during the testing and approval of AZT, the first drug used in AIDS therapy.... Although the study was originally intended to last 24 weeks, it was cut short and unblinded

half way through because of statistically significant differences in deaths between the two groups. Abrams lamented that although '18 more people made it to this arbitrary milestone of four to eight months after pneumocystis... I didn't feel that this was showing that we were prolonging survival' (page 116).

Abrams blamed the 'very powerful rhetoric' of the emerging community of AIDS activists, who demanded an end to clinical trials. 'Somebody should write a book about the impact of that decision on HIV clinical trials history,' added Abrams, 'because everything changed because of that demand.' Abrams recounted his early misgivings about AZT, which loses its effect after a year or two because the virus becomes resistant [a euphemism for a lethal dose of AZT (Martinez, 1997)]. He was also disturbed by findings demonstrating that a high dose of AZT resulted in a smaller rise of CD4 cells than a lower dose. 'Maybe if we just stop it altogether people will be better off,' he said. Members of the audience were surprised to learn of the paucity of solid, clinical research behind AZT and other nucleic acid chain terminators.... Abrams exposed the tragic farce of past AIDS research and therapy – people who thought they were doing something useful were actually wasting time and valuable resources.

'How should the clinician apply the new therapies? I have a large population of people who have chosen not to take any antiretrovirals since I've been following them since the very beginning.... They've watched all of their friends go on the antiviral bandwagon and die, so they've chose to remain naive [to therapy]. More and more, however, are now succumbing to pressure that protease inhibitors are 'it'.... We are in the middle of the honeymoon period, and whether or not this is going to be an enduring marriage is unclear to me at this time, so, I'm advising my patients if they still have time, to wait' (Tanaka, 1996).

Others echoed Abrams' concerns (Phillips & Smith, 1997, Karpas et al., 1997). For example, Jerome Groopman, from the Beth Israel Medical Center in Boston told *Newsweek* in December 1996 that 'some patients have been 'showing signs of the benefits wearing off' – an effect that is termed 'crashing' (Leland, 1996). Doctors from the National Institutes of Child Health and Development even published in a medical journal that 'zidovudine use is confounded by progression of HIV disease' (Moye et al., 1996). And British

researchers just wrote a letter to *Science*: 'It is, unfortunately, quite possible that all of the current regimens are, to use Lange's phrase, 'suboptimal' and the best strategy at present for asymptomatic individuals may be to take none of the existing cocktails, but to wait for improved treatments' (Breckenridge et al., 1997).

#### *The drug-manufacturers' warnings*

Even the drug manufacturers warn about drug-specific diseases. Sigma, a non-medical provider of AZT, warns with skull and crossbones on the label that AZT is toxic to the bone marrow, the very source of T-cells (Figure 3). Glaxo Wellcome, the producer of medical AZT, states in the *Physician's Desk Reference* that 'it was often difficult to distinguish adverse events possibly associated with zidovudine [AZT] administration from underlying signs of HIV disease...' (Retrovir, 1994).

Merck & Co., the manufacturer of the protease inhibitor Crixivan, warns 'about kidney pains' and the 'chances of forming a kidney stone ... in about 4% of patients; [and] pain, fatigue, nausea, diarrhea in 2% or more of patients' and that, 'It is not yet known whether taking CRIVAN will extend your life or reduce your chances of getting other illnesses associated with HIV' (Merck & Co., 1997).

Hoffmann-La Roche says in the UK about its protease inhibitor Invirase/saquinavir: 'Patients should be informed that saquinavir is not a cure for HIV infection and that they may continue to acquire illnesses associated with advanced HIV infection, including opportunistic infections' (Shenton, 1998). The American *Physician's Desk Reference* also has a 'WARNING – The indication for Invirase for the treatment of HIV infection is based on changes in surrogate markers [lab tests for HIV]. At present there are no results from controlled clinical trials evaluating the effect of regimens containing Invirase on survival or the clinical progression of HIV infection, such as the occurrence of opportunistic infections or malignancies' (Physician's Desk Reference, 1997).

#### *Anti-HIV drug diseases in the press*

In December, 1995, *The Advocate* published an anecdotal case against anti-viral therapy. The story describes a 47-year-old, HIV-positive man, who 'for at least 17 years doesn't have AIDS – and no one knows why':

Leoutsakas, 47: A former IV-drug user who last shared a needle in 1978 ... first tested positive in 1987. He has a T-cell count ... between 650

and 950. In addition, Leoutsakas has had none of the opportunistic infections that define AIDS – no pneumonia, no Kaposi's sarcoma, no fungal infections. Leoutsakas says doctors have attempted to explain his case by theorizing that, like the Australians (Learmont et al., 1992), he is infected with a weakened form of HIV – but it's really just speculation. ... Leoutsakas has no theory of his own – and no special formula for his well-being. He's never taken AZT or any other antiretroviral drugs (Simmons, 1995).

No more IV-drugs, no antiretroviral drugs – but *The Advocate* failed to see the 'formula for his well-being.'

Neville Hodgkinson, science correspondent of the *London Sunday Times*, has recorded in his book *AIDS: the Failure of Contemporary Science* the observations of the homosexual activist Callen (page 102):

In researching his 1990 book *Surviving AIDS*, Callen interviewed nearly fifty people who had lived for many years not just after being pronounced HIV-positive, but after an AIDS diagnosis. He found that only four had ever used AZT; three of those had since died, and one was dying of AZT-induced lymphoma. But the overwhelming majority of long-term survivors had somehow managed to resist the enormous pressure to take AZT.

The pressure did not just come from doctors, Callen told the Amsterdam meeting (Maddox, 1992; Hodgkinson, 1996), but from a certain segment of AIDS activism that seemed driven by a 'drugs-into-bodies' mentality. 'I feel many AIDS activist friends who are in the forefront of this frenzy are very misleading to people with AIDS, who are frightened and desperate. They only seem to talk about two possible outcomes of taking experimental drugs: one is that it works and one that it does not work. There is a third, apparently much more common possibility, which is that you will be worse off than if you did nothing at all. And nobody likes to talk about that because it is so unpleasant.' He had seen the devastation wreaked by AZT, watching with horror as friends with AIDS 'turn the colour of boiled ham from AZT poisoning, endure the melting away of their muscles, become transfusion dependent, and experience drug-induced psychosis.' Yet, 'they would sell their grandmother into slavery to get a slot in the latest drug-of-the-month clinical trial.' (Hodgkinson, 1996).

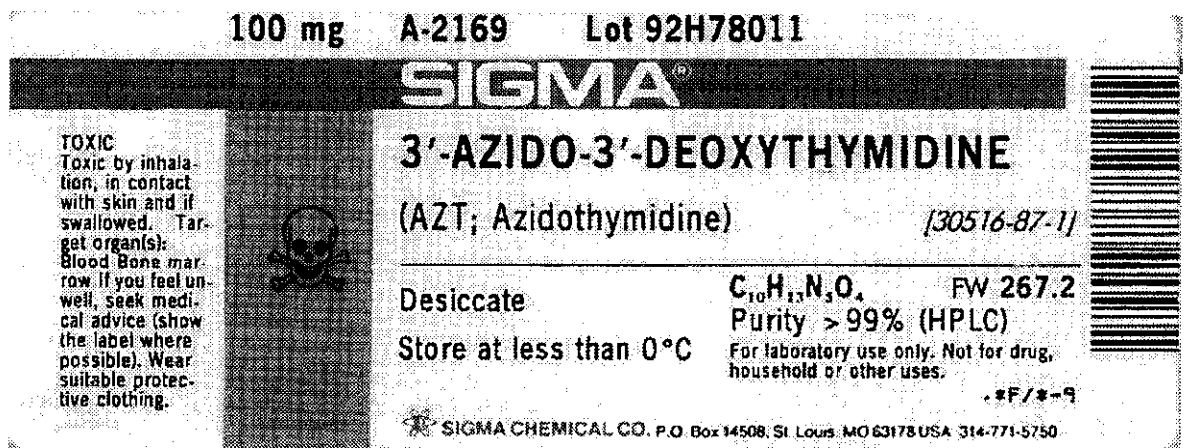


Figure 3. The advisory on the label reads: 'TOXIC. Toxic by inhalation, in contact with skin and if swallowed. Target organ(s): Blood bone marrow. If you feel unwell, seek medical advice (show the label where possible). Wear suitable protective clothing.'

Callen was on a polypharmacy of 56 AIDS drugs composed by his friend Sonnabend (page 99), but not on AZT, when he died in December 1993 from pulmonary Kaposi's sarcoma (Maddox, 1992; Lauritsen & Young, 1997). According to his biography, Callen had achieved over 3000 sexual contacts using nitrites and other drugs before his AIDS diagnosis (Callen, 1990; Farber, 1994).

In August 1997, about a year after the protease inhibitors had been introduced to the United States as a 'miracle cure,' *The New York Times* issued a first drug alert: 'Despite powerful new AIDS drugs many are still losing the battle' (Stolberg, 1997). According to the *Times*, 'medications seem to be failing 25 percent to 30 percent of the 150,000 people who are using them. For some the complex regimen of three drugs does not work from the onset, for reasons doctors do not understand. Some people get sick from the combination therapy, which has side effects ranging from diabetes to diarrhea. "There is an increasing percentage of people in whom, after a period of time, the virus breaks through," said Dr. Anthony Fauci, director of the National Institute of Allergy and Infectious Diseases in Bethesda.... The failure rate may eventually run as high as 50%.'

Indeed, by September 1997, the 'failure rate' already did 'run as high as 50%.' At that time the San Francisco General Hospital released cocktail treatment results from 'real world' patients, 'unlike the patients selected for well-controlled, industry-sponsored clinical trials.' According to this trial, the new 'AIDS drug cocktails fail 53%. ... The 47 percent of patients who obtained lasting benefit from the drugs ... were

those who were more recently infected and who had not been treated with earlier generations of anti-viral medicines' (Krieger, 1997). In other words, healthy 'patients' can tolerate the new cocktails longer than those already rendered sick by AZT.

But even 'lasting benefit' seems not to last. According to a meeting report from Chicago in February 1998, 'people taking advanced drug treatments for HIV infection ...' now 'develop hardened fat deposits in stomachs and on necks [termed] Buffalo hump [and] Protease Paunch' (Garrett, 1998). One study reports that 11% of drug takers have the 'abdominal fat problem.' Another reports that '60 percent of patients developed chemical changes akin to a pre-diabetes state after 15 months on the drugs' (Garrett, 1998). The report even acknowledges that 'none of the drugs [protease inhibitors] were originally tested on more than a handful of people for more than a year, so the long-term effect is unknown.' Neither the press nor the medical profession seem even to wonder anymore why there are no animal tests prior to the licensing of the drug for humans—just human experiments.

But anti-HIV drugs are not the only medical drugs that take life prematurely. A recent study from Canada has concluded that over two million Americans experience adverse reactions and over 100,000 die prematurely from prescription drugs annually (Lazarou et al., 1998).

### The solution to the AIDS dilemma

According to our analysis, drugs qualify both correlatively and functionally for AIDS causation:

1. All American and European AIDS-defining diseases that exceed their long-established national backgrounds correlate with long-term recreational drug use, or anti-HIV drug use, or both. The huge AIDS literature has yet to offer the first epidemiological study that presents a significant group of 'previously healthy' (Gottlieb et al., 1981b) people who have developed AIDS without drug use (Duesberg, 1995b).
2. The drugs used by AIDS patients have been shown in at least 63 scientific papers, in statistical analyses, and in numerous non-scientific reports to cause AIDS-defining and other diseases. In addition, specific drugs have been shown to cause AIDS risk group-specific AIDS diseases; that is, nitrites causing Kaposi's sarcoma, cocaine causing weight loss, and AZT causing muscle atrophy and dementia. Moreover, some AIDS patients have recovered by terminating drug use. Certainly further tests of the pathology of AIDS drugs could be obtained easily, either experimentally in animals or epidemiologically in humans (Duesberg, 1995b). But these studies are not done because of the prevailing bias against the drug-AIDS hypothesis (Weiss & Jaffe, 1990; Cohen, 1994a; Lang, 1996; O'Brien, 1997; Lang, 1998).

According to the drug hypothesis, AIDS would be entirely preventable and at least partially curable, if:

1. AZT and other anti-HIV drugs were banned,
2. recreational drug use were controlled by advertising that drugs may cause AIDS,
3. AIDS patients were treated for their specific diseases with proven medications, e.g. tuberculosis with antibiotics, Kaposi's sarcoma with conventional cancer therapy, and weight loss with good nutrition.

The drug hypothesis also answers all 12 questions that could not be answered by the virus-AIDS hypothesis (Introduction). And it predicts that the war on AIDS and the war on drugs could be won simultaneously if they were based on the health consequences of long-term drug use, as the success of the federal anti-smoking program already demonstrates. Education that smoking causes lung cancer, emphysema, and heart disease has reduced the percentage of smokers in the US from 42% of the adult population in 1965 to 25% in 1995 (Associated Press, 1995).

In addition to saving over 50,000 lives per year from AIDS, the drug hypothesis could save American taxpayers up to \$25 billion annually. Eight of the \$25 billion are currently spent on AIDS treatment, re-

search, and education based on the unproductive HIV hypothesis (AIDS Weekly, 1995; Gutknecht, 1995), and another \$17 billion are currently spent on the War on Drugs (Los Angeles Times, 1998; White House Office of National Drug Control Policy, 1998). There is but one obstacle to the solution of AIDS: the current AIDS establishment.

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