

CHAPTER SIX



A Fabricated Epidemic

BY THE MID-1980S, a sinister specter had been launched. The media buildup around AIDS, combined with the 1984 announcement of an AIDS virus, had painted a picture of a twentieth-century bubonic plague capable of ravaging our nation and the planet. Now everyone was aware of the deadly disease spreading through the homosexual community.

The scientific and government experts, most prominently including Surgeon General C. Everett Koop, predicted an explosion into the heterosexual population. In early 1987, Koop and the World Health Organization were forecasting that a staggering 100 million people would be infected with the virus by early 1990.¹ Talk of casual transmission became popular once top officials at the CDC and NIH announced HIV could be found in saliva.² Evidence that the virus could survive for long periods outside the human body led to nervousness about restaurants and public toilets.³ Naturally, the fact that HIV was a blood-borne virus spurred discussion of mosquito transmission, including among top AIDS researchers.⁴

AIDS was such a new syndrome that most of its mysteries remained to be solved. Certainly no vaccine, and probably no potent therapy, would be available for several years, by which

time hundreds of thousands—or millions—of people would already have died.

In the meantime, it seemed that only public health measures could work. Authorities tried to prevent further spread of the illness by discouraging the major risk activities, those routes most easily transmitting HIV—the most obvious threat was said to be sexual intercourse. Official warnings were always accompanied by reminders that, although the virus was now transmitted by homosexual contact, it would soon follow the usual pattern of infectious diseases by spreading among heterosexuals of all walks of life. Frightening reports of the African epidemic were exploited to paint a picture of our own future; there, whole villages were apparently disappearing as the new syndrome cut a wide swath of destruction among men and women alike. In the industrial world, heterosexual intravenous drug addicts were already passing HIV around by sharing their used syringes. AIDS officials confidently reassured the public of their timely screening and protection of the nation's blood supply, but noted they were too late to save most hemophiliacs.

Ominous statistics hit the news: 50 percent to 100 percent of everyone carrying the virus would die, and the unpredictable latent period between infection and AIDS ranged from five to ten years, during which time the carriers could infect many more people. Once infected, an individual's antibody defense raised against HIV was inexplicably useless, except to alert doctors to the fatal infection. Once the virus was reactivated (for unknown reasons), it proceeded to kill off the body's entire supply of T-cells, the white blood cells regulating the immune response against all other microbes. AIDS victims suffered horribly slow, painful deaths, being eaten alive by pneumonias, yeast infections, cancers, uncontrollable diarrhea, and dementia from brain degeneration. No recovery was possible since the patient was completely defenseless against many diseases normally harmless to a healthy person.

To add a further sense of urgency, AIDS experts supplemented their official estimate of one million HIV-positive Americans with suggestions of two million to three million, plus dire predictions that the number might double every year.

The public response to such news was inevitable. Battle lines rapidly emerged between two political camps—civil rights advocates for the HIV-positives and those championing health rights for the HIV-negatives.

Under the banner call, “Fight AIDS, not people,” groups ranging from the militant AIDS Coalition To Unleash Power (ACT UP) to the federal government’s National Commission on AIDS insisted that the syndrome be treated basically as a handicap. Although acknowledging that AIDS was contagious, many political activists feared the potential backlash from widespread panic. They preferred to mobilize support for the care of AIDS patients, assiduously avoiding any hint of blame on the victims. As the National Commission on AIDS proclaimed, “HIV disease has a devastating impact on those who are already marginalized members of society... HIV disease could not be understood outside the context of racism, homophobia, poverty, and unemployment.”⁵ Likewise, President Bush admonished that “once disease strikes we don’t blame those who are suffering. We don’t spurn the accident victim who didn’t wear a seat belt; we don’t reject the cancer patient who didn’t quit smoking. We try to love them and care for them and comfort them.”⁶

The CDC and other agencies deeply involved in managing the war on AIDS continued to warn of an imminent heterosexual epidemic. Activists for HIV were therefore forced to offer some solution to halt the syndrome’s spread, but without endangering homosexual liberation; they found an answer in condoms and programs to provide heroin addicts with sterile needles. But many activists, including those in the National Commission, also saw in AIDS much opportunity:

The HIV epidemic did not leave 37 million or more Americans without ways to finance their medical care—but it did dramatize their plight. The HIV epidemic did not cause the problem of homelessness—but it has expanded it and made it more visible. The HIV epidemic did not cause collapse of the health care system—but it has accelerated the disintegration of

our public hospitals and intensified their financing problems. The HIV epidemic did not directly augment problems of substance use—but it has made the need for drug treatment for all who request it a matter of urgent national priority.⁷

Another side of the debate operated on the principle of “Better safe than sorry,” viewing AIDS in more grand and threatening terms. This alarmism created strange alliances between such individuals as California Congressman William Dannemeyer and former Marxist (head of the U.S. Labor Party) Lyndon LaRouche. Most of these people were convinced the AIDS epidemic was actually far worse than officially acknowledged. They certainly had a rich source of raw material upon which to draw, including frequent quotes and numerical projections by federal officials. A 1985 book written by an investigator at the NIH provides a typical example:

The AIDS virus shows every sign of being just as deadly as the plague during the Middle Ages. We are on a crash course with reality. This is not a practice run. There is no second chance. AIDS may be to the twentieth century what the Black Plague was to the fourteenth century.

The alarm must be sounded, loudly and persuasively. If it is not, the conclusion is inescapable: millions may die.⁸

Believing the population to be on the verge of decimation, a variety of alarmists called for strong public health measures by the government. Their reaction on behalf of the uninfected took on the strenuous tone of Gene Antonio, whose 1986 book *The AIDS Cover-Up: The Real and Alarming Facts About AIDS* became an underground bestseller: “In the pell-mell rush to identify with the plight of AIDS sufferers, compassionate concern for the rest of society has been largely ignored. Permeated with heterophobia, AIDS victim identification hysteria has dangerously impeded compassionate steps being taken to safeguard the health of the rest of society.”⁹ The alarmists generally insisted on mandatory HIV testing, particularly for health care workers and those in AIDS risk

groups, as well as infection contact tracing and reportability to government agencies, and they even discussed possible quarantine of infected persons. More than fifty countries, including the United States, adopted immigration or tourism restrictions on infected people, and the Cuban government established a quarantine detention center for its HIV-positive citizens.¹⁰ Alarmists derided the weaker proposals of their opponents, often leaping to the defense of medical workers wanting more safeguards from potentially infected patients.

Yet despite their differences, both sides of the controversy agreed on one thing: More money was needed to fight AIDS—and quickly. Federal AIDS officials were no doubt delighted to hear California Congressman Dannemeyer, in an unusual alliance with Michigan Representative John Dingell for increased medical funding on AIDS, declare enthusiastically:

The AIDS Prevention Act of 1990 is a pathbreaking piece of legislation in many respects. For the first time, the federal government would make resources available to states, hospitals, high risk clinics, and nonprofit health care facilities to provide “preventive health services” to low income individuals afflicted with a specific disease—AIDS...

This legislation breaks new ground in bringing federal resources to bear on a very specific national health problem—the epidemic of HIV infection. It includes many admirable provisions which, if enacted, would establish sound priorities and provide state and local health officials with appropriate resources to fight this horrible epidemic.¹¹

This push for larger AIDS budgets certainly succeeded. Some \$7 billion were spent by the federal government during 1994, and well over \$35 billion has been spent since the AIDS epidemic began. What are the results of this modern-day Manhattan Project? A staggering one hundred thousand scientific papers so far have been published on HIV and AIDS, a number unprecedented for any other virus. But AIDS investigators have yet to

demonstrate that even a single life has been saved by any of their programs. No vaccine exists; condom and clean-needle programs have made no measurable impact on the epidemic; the admittedly toxic drugs AZT, ddI, and ddC, which do not cure AIDS, are the only therapy substitutes available today. Despite projections of wild spread, HIV infection has remained virtually constant throughout the industrialized world ever since it could be tested in 1985, whether in the United States or Europe; the estimated incubation period between infection and disease has been revised from ten months to more than ten years; and the predicted heterosexual explosion has failed to materialize. When a disease can be neither treated nor controlled, nor its course even roughly predicted, some fundamental assumption is probably badly askew.

HIV NOT GUILTY

Twenty years of belief in dormant human viruses causing disease after long incubation periods, plus many decades of hunting animal retroviruses, rendered most biologists utterly incapable of challenging Gallo's 1984 announcement of an AIDS virus. Prestigious awards and new grant moneys awaited scientists who could apply their animal models or "slow virus" concepts to human disease. Researchers also felt insecure about venturing outside their narrow fields of specialization to raise questions in other areas. Epidemiologists assumed clinicians were accurately describing their cases; virologists trusted the statistics of the epidemiologists; the immunologists placed confidence in the virologists' lab experiments; and the computer modeling experts believed them all. Any intrusion into another scientist's domain entailed peer rejection and humiliation.

In this atmosphere of pressure to conform, the lessons of the bacteria-hunting era were easily overlooked. Virtually no one thought to test HIV according to Koch's postulates. These time-tested standards apply even more perfectly to viruses, which are nonliving parasites with no behavioral flexibility, than they do to bacteria, which can sometimes release toxins or adapt to changing environments. The growing mountains of data on HIV were

instead interpreted solely to fit the consensus virus-AIDS hypothesis, and researchers forgot the very rudiments of virology itself as they assigned increasingly bizarre properties to this virus. But Koch's postulates do indeed cut to the heart of the issue, exonerating HIV and rendering most AIDS research entirely pointless:

1. *Koch's First Postulate: The microbe must be found in all cases of the disease.* Robert Koch explicitly stated that a causal germ would be found in high concentrations in the patient and distributed in the diseased tissues in such a way as to explain the course of the symptoms. In the case of AIDS, the affected tissues include the white blood cells of the immune system, particularly the T-cells, as well as the skin cells in lesions of Kaposi's sarcoma and brain neurons in dementia. But no trace of the virus can be found in either the Kaposi's sarcomas or the neurons of the central nervous system. Since retroviruses, in fact, cannot infect nondividing cells like neurons, the absence of HIV there is hardly surprising. However, because Kaposi's sarcoma itself has long been synonymous with AIDS, the absence of virus in this cancer seriously undermines the HIV hypothesis.

If HIV were actively infecting T-cells or other members of the body's immune system, cell-free virus particles, known as *virions*, should easily be found with great ease circulating in the blood. This is the case with all classical viral diseases: In a patient suffering from hepatitis B, one milliliter of blood (about five or ten drops) contains approximately ten million free virus particles. Likewise, flu-like symptoms appear only in the presence of one million rhinovirus particles per milliliter of nasal mucous, and one to one hundred billion particles of rotavirus per gram of feces will accompany diarrhea in the patient. But in most individuals suffering from AIDS, no virus particles can be found anywhere in the body. The remaining few patients have at most a few hundred or a few thousand infectious units per milliliter of blood. One paper published in March of 1993 reported two individuals with about one hundred thousand virus particles per milliliter of blood, out of dozens of AIDS patients with little or no detectable virus.¹² Thus HIV behaves as a harmless passenger microbe, only sporadically

coming back to life long after the immune system has been destroyed by something else and can no longer suppress the virus.

Even those patients with some detectable virus never have more than one in every ten thousand T-cells actively producing copies of the virus; on average, only one in every five hundred or more T-cells contains even a dormant virus. The abundance of uninfected T-cells in *all* AIDS patients is the fatal, definitive argument against the many false claims for high viral “loads” or “burdens” in AIDS patients.¹³ Nothing could ever stop infectious viruses from infecting all susceptible cells in the same body (except of course antiviral immunity). If T-cells remain uninfected, there are no viruses to infect them. The absence of active, infectious virus automatically disqualifies HIV as a player in the syndrome. Microbes can cause serious damage only when infecting the host’s cells faster than the body can replace them; T-cells, the presumed target of HIV, are constantly regenerating at much, much higher rates than dormant HIV in the presence of antiviral immunity.¹⁴

To gain some perspective, one should remember that most people carry inactive forms of several viruses, none of which cause disease while the microbes remain hidden and dormant in the body. Two out of every three Americans carry the herpes virus, and an equal number harbor the herpes-class cytomegalovirus; Epstein-Barr virus, causing mononucleosis (“kissing disease”) when active, resides in dormant form in four of every five Americans; and an even higher proportion of people host the papilloma, or wart, virus. If these viruses could cause disease while latent, the absurd situation would arise in which virtually no one would be left to treat the hundreds of millions of sufferers.

HIV is not, of course, behaving differently from other viruses. Upon infecting a new host, a typical virus invades its target cells and begins replicating in large quantities, producing new virus particles that spill into the bloodstream and infect more cells; this is the period during which high levels of virus can be isolated from the patient and the symptoms are strongest. The body’s immune system responds to the threat by mobilizing to mass-produce the specific antibody proteins that attack and neutralize the virus particles. As this battle heats

up, antibodies are produced more rapidly than the virus, ultimately eliminating active virus from the body. Most viruses are thereby completely destroyed, although some herpes viruses can establish chronic infections by hiding in certain tissues.

Retroviruses, by nature, insert their genetic information into infected host cells, becoming dormant once neutralized by the host's immune system. HIV, like other retroviruses, can achieve high levels of virus when first infecting the body (up to one hundred thousand particles per milliliter of blood), but in most people HIV is then permanently inactivated by the antibodies generated against it. During this brief period of HIV activity, some newly infected people have reported mild flu-like symptoms at most—but no AIDS diseases. But all of these rare cases were male homosexuals from high-risk groups, meaning people who had used recreational drugs that can cause exactly the same symptoms.

Outside this risk group are the seventeen million HIV-positive healthy people identified by the World Health Organization¹⁵ who cannot connect any past disease with HIV infection; they are either surprised or shocked when they find out about being “positive” or are blissfully unaware of it. The reason is that HIV is one of the many harmless passenger viruses that cause no clinical symptoms during the acute infection. By contrast, most people have lasting memories of their mumps, measles, hepatitis, polio, chicken pox, and flus, after which they become “antibody positive” for the respective viruses.

AIDS patients, on the other hand, have generally been infected by HIV for years, not days, before they deteriorate and die. Thus, the virus has long since been neutralized, forcing doctors to test the patient either for the dormant virus or the antibodies against it. This is the operating principle of the “HIV test,” which identifies antibodies, and yet ironically stands as proof of the innocence of this virus.

Not all AIDS patients, however, carry even dormant HIV. Antibody-positive patients usually do have some latent virus left over from past infection. But many people dying of AIDS-like conditions, ranging from Kaposi's sarcoma to immune deficiencies and various

opportunistic infections, have never been infected by HIV in the first place. The CDC does not include most of these antibody-negative cases in its AIDS figures, rendering these people invisible.

According to the CDC's own statistics, at least 25 percent of all official AIDS cases have never been tested for antibodies against HIV, many of whom might turn out to be negative. Further, the HIV test itself often generates false-positive results, particularly in members of AIDS risk groups who have been infected with large numbers of interfering viruses.¹⁶ Thorough follow-up testing could reveal HIV-negative cases in the official AIDS tally. The scientific literature describes some 4,621 confirmed cases of HIV-free people dying of AIDS diseases, including homosexuals and heroin addicts in the United States and Europe, and central Africans.¹⁷ These dozens of studies generally found that, among any group of clinically diagnosed AIDS patients, many test negative for HIV. But because the CDC ignores virtually all HIV-negative patients, counting only those with the virus as AIDS cases, the total number of such cases may never be known.

Even a "slow virus" hypothesis of HIV cannot explain how uninfected people would develop AIDS conditions. From every angle, HIV fails Koch's first postulate.

2. Koch's Second Postulate: The microbe must be isolated from the host and grown in pure culture. This postulate was designed to prove that a given disease was caused by a particular germ, rather than by some undetermined mixture of noninfectious substances. HIV has been isolated and is now grown continuously in HIV research labs. This rule therefore has technically been fulfilled, but only in some instances.

Since free virus is rarely found in AIDS victims, HIV can be retrieved only from the great majority of them by reactivating the latent form of the virus. Millions of white blood cells must be taken from the patient and grown in culture dishes for weeks, during which time chemical stimulants that shock cells into growing or mutating are added to awaken any dormant HIV from within its host cells. Given enough patience and plenty of repetition of

such procedures, a single intact virus can eventually be activated, at which point it starts infecting the remaining cultured cells. Yet even this powerful method does not yield active virus from many AIDS cases that have confirmed antibodies against HIV. Gallo himself faced this intractable problem, a frustrating situation that may have led him to claim Luc Montagnier's virus as his own.

The situation is a mirror image of biological virus isolation that happens every time an uninfected person contracts the virus from an infected host. Natural transmission by unprotected sex has been studied in "discordant" couples, i.e., HIV-free women married to HIV-positive hemophiliacs or HIV-free male homosexuals having HIV-free sexual partners. These studies have revealed a rarely mentioned fact: After neutralizing the virus with the immune response, an HIV-positive person requires an average of one thousand unprotected sexual contacts to pass this virus along just once.¹⁸

A pregnant mother is a different story; in effect, she provides her child with a nine-month continuous exposure to her blood and therefore has at least a 50 percent chance of passing HIV to the baby. HIV, as with any retrovirus, survives by reaching new hosts perinatally (mother to child), this being five hundred times more efficient than by sexual transmission.¹⁹

This would explain why the numbers of HIV-positive people, in America as well as Africa, have remained so constant: HIV is transmitted from mother to child just like a human gene. This also reveals the reason for the virus being so widespread and equal between the sexes in Africa—HIV has been passed along from mother to child for many centuries (not through one thousand heterosexual contacts as is commonly assumed).²⁰

In the industrial world, HIV can be readily transmitted only among the most sexually active homosexuals, among needle-sharing addicts, and through blood transfusions to hemophiliacs—the routes that so easily transmit numerous other microbes. In short, the very people with tremendous health risks to begin with *also* more easily pass along HIV, making it a surrogate marker for the real cause of AIDS (see chapters 8–10). Therefore, a rough correlation exists between HIV and AIDS diseases, but it is imperfect and misleading.

The extremely low efficiency of sexual transmission explains the failures of Gallo, Weiss, and other leading AIDS researchers in isolating HIV: Even for the most experienced virus hunters, a virus that is not present is difficult to find. Only rare luck or misfortune, depending on one's purposes, and extreme persistence can extract HIV from an antibody-positive person.

The very ability of retroviruses to survive as dormant genes by attaching themselves to human chromosomes has been exploited for the most sensitive HIV assay yet—the Polymerase Chain Reaction (PCR). This incredibly sensitive technique was invented in the mid-1980s by Berkeley biochemist Kary Mullis, who was awarded the Nobel Prize for his discovery in 1993. The PCR is a technology that amplifies even the tiniest amounts of any specific DNA sequence, creating enough copies of the desired sequence for detection and analysis. This amounts to finding the proverbial needle of dormant HIV in a haystack of human DNA. But contrary to statements by some HIV scientists, this is not an isolation of the actual virus and does not fulfill Koch's second postulate. It is only the detection of dormant DNA genomes, or fractions of viral genomes, left behind from infections that occurred years earlier. Nevertheless, scientists and journalists alike sometimes mislabel such exhumations of viral fossils as “new, more sensitive techniques”²¹ that somehow prove HIV can be found in an ever-greater portion of AIDS patients. Because a few HIV molecules are technically invisible but millions of HIV molecules are visible, Mullis's PCR technique has become the only practical method to detect viral molecules in all those antibody-positive people in which no virus can be found.

3. *Koch's Third Postulate: The microbe must reproduce the original disease when introduced into a susceptible host.* The official HIV-AIDS hypothesis declares a 50 percent to 100 percent probability of death from infection. In practice, scientists and medical doctors interpret antibodies against HIV as a sure sign of imminent doom. This notion, of antibodies as a prognosis of death, defies all classical experience with viruses and bacteria. Virtually every microbe causes disease in only a minority of infected individuals,

since the majority are usually healthy enough to mount a rapid immune response. Certainly no fatal viral disease is known to cause death in nearly all infected people—except the paradoxical “AIDS virus.” Any microbe killing all its hosts would soon destroy itself, even if such could exist in the first place; any germ must be able to reach new hosts before the previous one dies, lest it go down with a sinking ship. Any universally lethal parasite would be, by definition, a suicidal organism. HIV would face even less chance of survival, being extremely difficult to transmit from one person to another, and would thus usually die with its infected host.

Traditional incubation periods, defined as the time between initial viral infection and the onset of disease symptoms, are measured in days or weeks. During this period the virus multiplies into concentrations high enough to cause disease. The process is exponential: Each virus particle infects a single cell, and eight to forty-eight hours later hundreds of new virus particles begin to be produced, each destined to infect a new cell. Flu, common colds, and herpes simplex infections develop with short incubations lasting between a few days and weeks; measles, chicken pox, and rubella have longer incubations of ten to twenty days, while extreme conditions such as hepatitis can take two to six weeks. These delays occur before the body has launched an immune response against the new virus.

Because these delays or latent periods are determined entirely by the generation time of the virus, and the generation time of HIV is about forty-eight hours, we can calculate how soon after infection AIDS should appear. Natural infection only introduces a few viruses into the body. But just one infected cell produces at least one hundred offspring within two days. These in turn will produce one hundred times one hundred within two days. Such exponential or explosive growth will produce *100 trillion* (100,000,000,000,000, or 10^{14}) viruses in just two weeks—enough to infect every single cell in the human body. Therefore, HIV should cause AIDS within a few weeks of infection.

But borrowing from their cancer research, virus hunters officially give HIV ten years between infection and the onset of AIDS—years

after antibodies have neutralized the virus. Such latency periods have been invented solely to circumvent Robert Koch's third postulate. But any germ not causing symptoms before being cleared by the immune system should be ruled out as causing disease.

Koch's third postulate insists on reproducing the disease in at least some cases by injecting the allegedly dangerous microbe into a number of uninfected and otherwise healthy hosts. This condition can be tested in one of three ways: infection of laboratory animals, accidental and natural infection of humans (deliberate infection would be unethical), or by vaccination experiments. HIV fails all three tests:

(a) Blood from AIDS patients was injected into several chimpanzees in 1983, before the availability of HIV tests. The animals were infected by HIV, as later evidenced by antibodies against the virus, but in ten years none has yet developed any sickness. Roughly 150 other lab chimpanzees, injected with purified HIV since 1984, have proved that antibodies against the virus are generated within a month of inoculation just as in humans; but again, none has developed symptoms to this very day.²²

In short, no animal becomes sick from HIV, although monkeys and other test animals do suffer disease from human viruses causing polio, flu, hepatitis, and other conditions.

By the end of 1992 the CDC had reported some thirty-three medical workers as most likely having received HIV accidentally, of whom seven were diagnosed with AIDS symptoms. None of these reports has been confirmed with published medical case histories, although in a 1989 issue of the *New England Journal of Medicine* an informal editorial entitled, "When a House Officer Gets AIDS" was written by a doctor infected by a patient. The article describes only minor weight loss of ten pounds and a "bit" of fatigue as being the doctor's AIDS "complications."²³ This hardly counts as evidence for Koch's third postulate. Nor has the CDC stated whether any of these medical workers have taken the dangerously toxic AZT, the official AIDS treatment, which itself causes immune deficiency (see chapter 9).

(b) During the past decade, more than four hundred thousand AIDS patients have been treated and investigated by a system of five million medical workers and AIDS researchers, none of whom have been vaccinated against HIV. Doctors who have treated AIDS patients were initially admired by their peers and the press for their courage to face a fatal, contagious condition for which there was no cure, no drug, and no vaccine.

But ten years later there is *not even one* case in the scientific literature of a health care worker who ever contracted presumably infectious AIDS from a patient. Imagine what it would have been like if four hundred thousand cholera, hepatitis, syphilis, influenza, or rabies patients had been treated by health care workers for ten years without protection from vaccines and antimicrobial drugs—thousands would have contracted these diseases. This is exactly why we consider these diseases infectious. The complete failure of four hundred thousand AIDS patients to transmit their diseases to even one of their unvaccinated doctors in ten years can mean only one thing: AIDS is not infectious.

However, several thousand health care workers have by now been diagnosed with AIDS, but these individuals belong to the same AIDS risk groups as 90 percent of all AIDS cases—homosexuals and intravenous drug users. And although three-quarters of all health care workers are female, more than 90 percent of these AIDS patients are male, the exact same ratio as with all other AIDS cases.²⁴ *In other words, medical accidents are not producing the expected AIDS epidemic among unvaccinated personnel in that industry.*

Nor has HIV affected the recipients of blood transfusions, most notably hemophiliacs. Some fifteen thousand hemophiliacs in the United States—about three-quarters of the total—were infected with HIV before screening of the blood supply began in 1984. But also during the past fifteen years, improved medical treatment has doubled their median life expectancy. The virus-AIDS hypothesis would have predicted that now, ten and more years later, more than half of them would have died from AIDS. Instead fewer than 2 percent of these HIV-positive hemophiliacs develop AIDS each

year. According to several dozen small studies, this matches the rate of immune deficiencies and death among HIV-negative hemophiliacs, a phenomenon apparently related to hemophilia itself.²⁵

(c) The third postulate can be tested in humans through a reverse method. If vaccines or other techniques can be used to provoke the body into neutralizing the microbe with antibodies and the disease is thereby prevented, the germ has been proven guilty experimentally. But since AIDS is found in each patient only after the immune system has already suppressed HIV, the virus plays no role. Most AIDS researchers have conveniently forgotten this important principle and continue to blame the virus when only antibodies against it can be found; others blatantly reverse the logic of the vaccination test, declaring antibodies useless because they do not prevent AIDS.

(d) The acid test of Koch's third postulate would be to infect newborn babies with HIV, because newborns are immunotolerant and thus much more susceptible to a virus than adults. It is known from experiments with animals that a virus is totally harmless if it does not cause a disease in newborns.

It would, of course, be unthinkable to inject HIV experimentally in human babies to test whether it causes AIDS. Yet, exactly this experiment has already been done millions of times by nature to generate most of the seventeen million healthy, but HIV-positive, people living on this planet.²⁶ Most of these people picked up HIV by natural infection from their mothers.

Indeed, all animal and human retroviruses, including HIV, depend on mother-to-child (perinatal) transmission for survival. Since sexual transmission is extremely inefficient, depending on one thousand sexual contacts in the case of HIV, retroviruses could never survive by sexual transmission. They can only survive by perinatal transmission, which is about 50 percent efficient.²⁷ Therefore perinatal transmission must be harmless or else the baby, the mother, and the virus would not survive; HIV would be a kamikaze killer—it would kill itself together with its host.

If that were true, one would expect thousands of healthy young American men or women to have HIV but not AIDS. That is exactly what the U.S. Army reports. The U.S. Army tests all applicants and all its young men and women annually and identifies thousands of HIV-positives who are totally healthy. While some of these might have acquired their virus sexually, it is impossible that thousands would have had the 1,000 sexual contacts with HIV-positives or the 250,000 sexual contacts with average Americans (of which only 1 in 250 is HIV-positive) that are necessary to pick up HIV by sexual transmission.²⁸ Therefore, most of these HIV-positive young men and women must have acquired HIV from their mothers sixteen to twenty years prior to their application to the U.S. Army. The same must be true for most of the remaining seventeen million humans who are healthy and HIV-positive.

The fact that millions have acquired HIV at birth yet are healthy adults is the most devastating argument against the HIV-AIDS hypothesis. It proves that HIV, like all other microbes that are transmitted perinatally or sexually, cannot be fatally pathogenic. Indeed no fatally pathogenic microbe exists in animals or humans that depends either on perinatal or sexual transmission for survival.

No matter how one looks at the HIV hypothesis, it is flawed either in terms of facts or in theory or in both.

(e) Koch's third postulate can also be tested provisionally on human cells in culture. If HIV cannot induce disease in whole organisms, one might at least expect it to kill T-cells grown in laboratory culture dishes, where the concentrations of actively replicating virus are enormously high. Robert Gallo, however, has been able to patent the virus by growing it continuously in immortal T-cell cultures since 1984. The French discoverer of the virus, Luc Montagnier, reported occasional cell death in infected cultures that was stopped by adding antibiotics, which do not affect virus replication but do kill undetected bacterial contaminants. Indeed, the HIV antibody test is made from virus that is mass-produced in T-cells, which grow continuously rather than die. The reports from other labs and biotechnology companies are consistent: HIV grows harmoniously

with the cells it infects. The failure to kill T-cells, even under optimal conditions, is the Achilles' heel of the supposed AIDS virus.²⁹

HIV typifies a retrovirus in every measurable way. It has the same biochemical structure and infective properties, benignly stimulating some cells to produce more copies of the virus. It has the same amount of genetic information and the same three basic genes as all other retroviruses. It also has six smaller genes, themselves a normal feature of other retroviruses. Although many HIV researchers focus their efforts on studying these "extra" genes as possible AIDS genes, no one gene is unusual and all are needed for virus survival. HIV contains no special "AIDS gene" expressed during the syndrome. However, this does not stop industrious AIDS scientists from endlessly reexamining the genetic sequences for some magical clue to explain AIDS.

HIV clearly fails Koch's postulates. However, virologists should have expected this from the beginning. HIV is, after all, a retrovirus, precisely the kind of virus so benign to its host cells that it had inspired such hope in the War on Cancer, since cancer cells grow and behave uncontrollably rather than die. Retroviruses have never been known to inhibit or kill billions of rapidly dividing cells and could hardly be expected to affect T-cells or otherwise destroy the immune system.

To be the cause of AIDS, the virus would require still more miracles. A number of the AIDS indicator diseases are not opportunistic infections preying on an immune-deficient host, including dementia, wasting syndrome, and the various AIDS cancers—Kaposi's sarcoma, the lymphomas, and, as of 1993, cervical cancer. Altogether these non-immunodeficiency AIDS diseases made up 39 percent of all American AIDS diseases in 1992, and, owing to a new definition of AIDS, 20 percent of all AIDS diseases in 1993 (see Table 1).

HIV would have to kill T-cells while destroying brain neurons it cannot infect and at the same time induce white blood cells and skin cells to grow malignantly. To reconcile these non-immunodeficiency diseases with HIV, AIDS scientists would like to blame even these diseases on immune suppression. But despite years of research, no evidence can be found that the immune system fights cancer cells, which,

TABLE 1

AIDS-defining diseases in the United States in 1992^a and 1993^a fall into two classes: immunodeficiency diseases and non-immunodeficiency diseases

Immuno- deficiencies	1992 (in %)	1993 (in %)	Non-immuno- deficiencies	1992 (in %)	1993 (in %)
<200 T-cells	—	79	wasting disease	20	10
pneumonia	42	22	Kaposi's sarcoma	9	5
candidiasis	17	9	dementia	6	3
mycobacterial (including tuberculosis)	12	11	lymphoma	4	2
cytomegalovirus	8	4			
toxoplasmosis	5	2			
herpesvirus	5	3			
Total =	61 ^b	80 ^b	Total =	39	20

^a The data are from the Centers for Disease Control (Centers for Disease Control, 1993; Centers for Disease Control and Prevention, 1994).

^b Over 61 percent and 80 percent are due to overlaps.

In the United States 39 percent of all AIDS cases were non-immunodeficiency diseases in 1992. Owing to the third re-definition of AIDS by the Centers for Disease Control in 1993, that included less than 200 T-cells per microliter of blood as an AIDS disease, about 20 percent of all American AIDS diseases were non-immunodeficiency diseases in 1993. The distribution of AIDS diseases in 1994 was nearly the same as in 1993, since the AIDS definition was not changed that year.

after all, are part of the host's own body. In fact, dozens of AIDS patients with Kaposi's sarcoma or dementia have been reported to have normal immune systems.³⁰ So HIV would indeed have to accomplish many incredible tasks at once. Stranger still, infants with AIDS suffer immune suppression from deficiencies in B-cells, a subgroup of white blood cells altogether different from T-cells.

Since there are no precedents for cell-killing retroviruses and no laws other than Koch's for convicting viruses for a disease, even the HIV orthodoxy admits that their hypothesis stands unproven.³¹ However, they insist that Koch's not-guilty verdict of HIV does not prove HIV innocent and that further work will eventually prove HIV guilty.

No matter how convincing the HIV-AIDS paradoxes should be, official AIDS scientists cannot be dissuaded from their virus hypothesis. When forced to answer the above arguments, their imaginations run wild in designing ever-new variations of the same experiments to prove their hypothesis.³² According to HIV advocate John Maddox, “The remedy is not, of course, to pander to wish-fulfillment, but to redouble effort in the laboratory and the clinic.”³³ But these experiments have only proven to this date that the HIV hypothesis is impossible to prove.

INNOCENT VIRUS

According to Koch’s postulates, HIV is “not guilty” of AIDS. But this not-guilty verdict is not perceived as innocence by most scientists, particularly by nonscientists, for two reasons:

1. The term *virus* (the Latin word for poison), just by itself, inspires fear. Therefore HIV must be bad. This general prejudice that all viruses are bad is based on the fact that some viruses actually are bad. These pathogenic viruses and microbes are to researchers and to the press what criminals are to detectives—the focus and justification of their existence.

But only a few people know that the great majority of all viruses and microbes cause no disease at all. Such viruses are called *passenger* viruses.³⁴ They are the most uninteresting of all viruses to virologists, because the standing of virologists in the scientific community depends on the pathogenic potential of the viruses they study. Since passenger viruses do not advertise their presence by causing a disease, most of them go unnoticed, riding with their hosts like a passenger in an airplane. Passengers are the silent majority of animal and human viruses; pathogenic viruses are just the tip of the iceberg.

Passenger viruses infect just enough cells of the host to survive without ever causing a disease. Since passenger viruses keep such a low profile, virologists could not easily detect them until recently, when the technology was developed to detect needles in

a haystack. Because a passenger virus neither hurts nor kills, it is the most efficient survivor and hence the most common virus in animals and man.

2. The second reason even scientists consider HIV not innocent in AIDS is the much cited “overwhelming correlation between HIV and AIDS.” However, the HIV-correlation argument is not just misleading; it is deceptive on three counts:

First, the overwhelming correlation is not with HIV but with an antibody against it—a difference like day and night. A virus is a potential pathogen, an antibody is a certain antidote.

Second, American and European AIDS risk groups have one common microbial denominator: They have many more microbes and many more antibodies against microbes than the rest of the population.³⁵ This is because from a microbiologist’s point of view, “AIDS risk behavior” is collecting microbes in the process of many sexual contacts with different persons (promiscuity), sharing needles during intravenous drug use, consumption of unsterile drugs, prostitution for drugs, or receiving transfusions for hemophilia. No matter what microbe one chooses—toxoplasma, bacteria-causing syphilis, genital wart virus, human T-cell leukemia virus, cytomegalovirus, one of the many herpes viruses, hepatitis virus, or HIV—it correlates overwhelmingly with risk behavior. In fact, three of these microbes, namely syphilis, HTLV-I, and cytomegalovirus, were considered AIDS causes before HIV, because of “overwhelming” correlations with antibodies against them.³⁶ However, since HIV was chosen, rather than proved, to be the cause of AIDS in 1984, the correlation with HIV and AIDS became 100 percent—the definition of AIDS. Therefore, the overwhelming correlation is one of the purest examples of circular logic.³⁷

Third, the literature includes more than 4,621 clinically diagnosed AIDS cases that are all HIV-free (see appendix C). To cover up this discrepancy with the overwhelming correlation, HIV-free AIDS cases were renamed in 1992 as *idiopathic CD4-lymphocytopenia* (ICL) cases by the CDC and Anthony Fauci, the director of the National Institute of Allergy and Infectious

Diseases.³⁸ Thus, the “overwhelming correlation” between antibodies against HIV and AIDS is a mere consequence of risk behavior and of the definition of AIDS. It is irrelevant for causation.

The scientific method offers three unambiguous criteria on how to tell a virus that is potentially “guilty by association” from one that is an innocent passenger virus:

1. The time between infection by a passenger virus and the occurrence of any disease, if one occurs, is entirely unpredictable. It could be anywhere from a day to the lifetime of the patient. Since the passenger virus does not cause a disease, the time of infection is irrelevant to the onset of a disease.
2. A passenger virus can be active or passive, rare or abundant, during any disease. Since the passenger does not cause disease, its activity is irrelevant to it.
3. The passenger virus can be present or absent during any disease. Since the virus is not pathogenic, disease can occur in the absence of the passenger virus.

In short, a virus that has been in its host for years before a disease occurs, that is typically inactive and rare during a disease, and that is not present in every case of that disease is not a credible suspect for viral disease. It is an innocent bystander or a passenger virus. HIV meets all of these criteria. Since HIV also fails Koch’s postulates, there is no rational basis for the HIV-AIDS hypothesis. In the courts of science HIV must be acquitted of all charges for AIDS—it is an innocent virus.

AIDS NOT INFECTIOUS

In December 1994 *Science* wrote a surprising editorial blaming a newly discovered herpes virus for Kaposi’s sarcoma.³⁹ The surprise was that the AIDS orthodoxy had adopted the view that another virus could cause AIDS. Although this article should have

registered as a major heresy among AIDS scientists, it did not. It was received instead only as a “minor sin” because it did not question the central, although tacit, dogma of the AIDS orthodoxy: infectious AIDS. Questioning infectious AIDS is without doubt the ultimate heresy in the AIDS orthodoxy.

The fear of questions about the orthodoxy’s most carefully cultivated dogma is understandable, because AIDS does not meet the classic epidemiological criteria of an infectious disease:

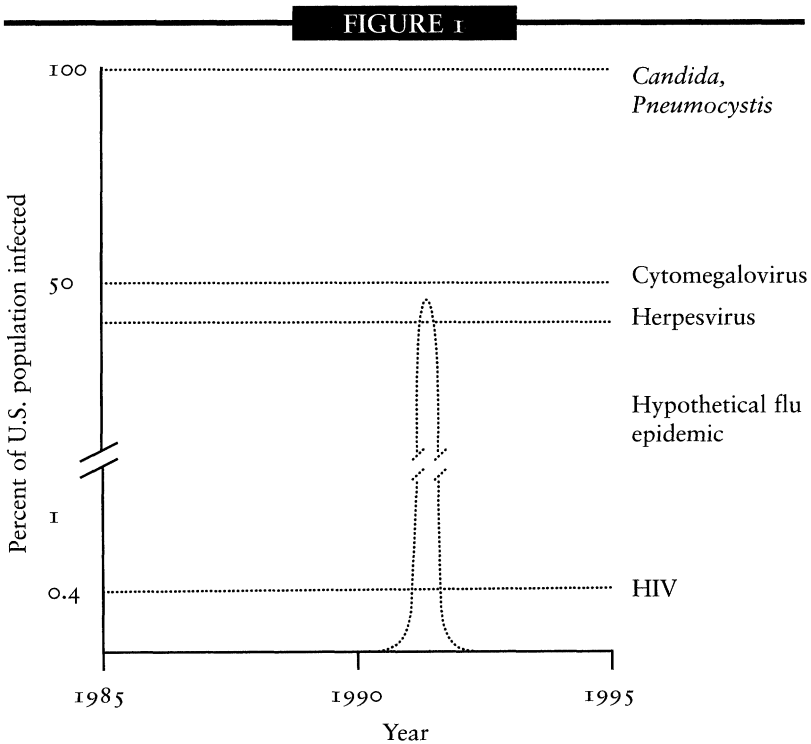
1. *Infectious diseases do not discriminate between sexes.* The first epidemiological law of viral and microbial diseases holds that men and women are affected equally, because no virus or microbe discriminates between the sexes. This law applies to all known infectious diseases affecting large populations. Examples are flu, polio, syphilis, hepatitis, tuberculosis, pneumonia, and herpes—all of which do not discriminate between the sexes nor do they select their victims only from specific risk groups.

By contrast, AIDS selects all its victims from a few, newly established AIDS risk groups: long-term intravenous drug addicts and their babies, male homosexuals using recreational drugs, and hemophiliacs under long-term treatment with commercial clotting factor VIII. Breaking with the sexual equality displayed by conventional infectious diseases, AIDS attacks men ten times more often than women in Europe and the United States. Among men it decidedly prefers homosexuals to heterosexuals. Thus, American and European AIDS is not distributed between the sexes like an infectious disease. (Chapter 8 explains why African AIDS does not discriminate between men and women.)

2. *Farr’s law: Infectious diseases spread exponentially.* Early in the last century the British epidemiologist William Farr first recognized the seasonal rise and fall of microbial epidemics.⁴⁰ A new infectious disease rapidly explodes in a population—just as rapidly as microbes are transmitted from person to person. Then it declines within months because it is stopped by the elimination of susceptible victims either by death or more often by natural

immunization. In accordance with Farr's law the Hawaiian natives, the California Indians, and the Eskimos were all quickly decimated by European microbes once they had been introduced to them by their European discoverers. But survivors soon became as resistant to these microbes as the Europeans. Likewise, contemporary Americans and Europeans suffer from new, seasonal flu epidemics, following Farr's law to the letter.

Figure 1 shows the exponential rise and fall of a new, seasonal flu epidemic against the backgrounds of several long-established



The distribution over time of a new hypothetical flu epidemic, against the background of several parasites long-established in the United States. The long-established parasites differ from a new one in their distribution over time: According to Farr's law the new one rises and falls (or equilibrates) exponentially—the old ones remain steady. Its unchanging incidence in the United States identifies HIV as an old American virus!

microbes. Since the percentage of Americans with herpes virus, cytomegalovirus, and the fungal parasites *Pneumocystis* and *Candida* is constant over time, these are “old” American microbes. Surprisingly, HIV is one of them, because 1 in 250 Americans (0.4 percent) have been “positive” ever since HIV could be detected in 1984. Thus, contrary to its reputation, HIV is an old American virus.

Figure 2A compares the time course of the American AIDS epidemic with that of the American HIV epidemic. The comparison offers another surprise: The HIV epidemic is constant and thus old, but the AIDS epidemic is increasing and thus new. Since the two epidemics follow totally different time courses, the HIV epidemic cannot possibly be the cause of the AIDS epidemic.

In sharp contrast to the bell-shaped curve of a conventional new infectious epidemic, like the flu epidemic shown in Figure 1, the AIDS epidemic increased steadily for fifteen years (Figure 2A). American AIDS gradually spread from a few dozen cases annually in 1981 to more than eighty thousand cases in 1994. It did not explode, as the HIV orthodoxy predicted; neither did it decline, as would be expected from antiviral immunity.⁴¹ Instead of resembling an infectious disease, the time course of the AIDS epidemic resembles the slow progressing epidemics of lung cancer and emphysema in industrialized nations, building up over the years in step with tobacco consumption. These noninfectious epidemics neither rose exponentially nor affected all groups of the population or both sexes equally, nor did they disappear as a result of antiviral immunity or natural resistance.

Thus, AIDS does not meet the classical epidemiological criteria of an infectious disease. The failure of AIDS to meet these criteria destroys not only all hopes of the HIV orthodoxy ever to prove that HIV causes AIDS, but also any other viral or bacterial theories of AIDS.

Despite all these violations of the fundamental principles of virology and epidemiology, the virus-AIDS hypothesis has remained the sole basis for our unproductive war on AIDS. This is as much a scientific as a human tragedy. The reckless rule of the

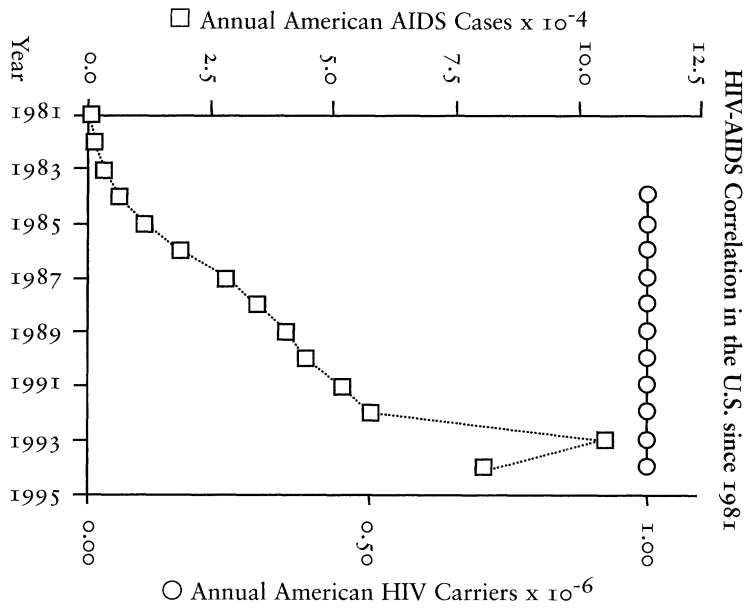


FIGURE 2A

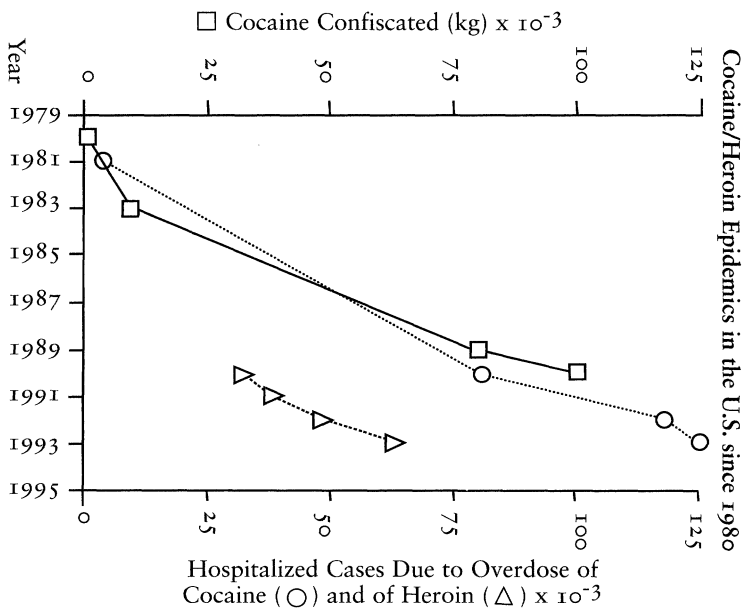


FIGURE 2B

HIV-AIDS monopoly breaches the most fundamental principle of disease control, “First find the cause, then fight the cause,” and closes the door for alternative hypotheses that might be productive.

DEFENDING THE LOW GROUND

After the polio epidemic ended, no new diseases and no fundamentally different viruses were being discovered. To maintain a medical relevance, virologists began connecting known viruses to unexplained diseases—such as cancer or multiple sclerosis. Because these diseases in no way behave as traditional infectious diseases, the virus hunters had to invent new properties for the germs. First, the incubation period of viruses—typically anywhere between one day and three weeks—was allowed to stretch into years. Then antibodies had to be abandoned as a sign of immunity against the microbes. And since the viruses never reappeared during disease, indirect methods of damage had to be postulated.

Nevertheless, all these creative maneuvers merely delayed the inevitable. By the early 1980s, virology was withering from lack of public interest—a fatal weakness when trying to attract new recruits, research money, and federal programs. The public was losing faith in wars on cancer that were never won or wars on diseases that rarely affected the average person.

But AIDS has changed everything, reviving virus hunting as the most glamorous and rewarding branch of biomedical research. To blame HIV for AIDS, virologists had to employ every invention at their disposal, including an ever-expanding latent period, an antibody test, and plenty of paradoxes to keep tens of thousands of investigators busy for many years. The evolution toward these false assumptions had been so gradual, so favored by consensus politics within science, and so shaped by the increasing sensitivity of biotechnology, that most researchers had been lulled into thinking of such rationalizations as normal science. By the time Robert Gallo and other virus hunters had engraved the HIV hypothesis in stone, anyone who dared to raise serious questions appeared truly radical to the rest of the research establishment.

Peter Duesberg first began to ask his colleagues questions about the HIV hypothesis shortly after Gallo's 1984 press conference. The HIV dissidents could see two fundamental problems: HIV was a retrovirus, meaning it should not kill the cells it infected, and the virus could barely be detected even in late-stage AIDS patients. The following year, the NIH awarded Duesberg its Outstanding Investigator Grant, a special seven-year award officially designed to allow free inquiry and latitude for exploring risky new research directions. He took this mandate to heart. As the discussions over HIV continued quietly, he began exploring the issue as a potentially important shift from his usual work on cancer genes and animal retroviruses.

Upon hearing of Duesberg's doubts about whether retroviruses could cause cancer in humans or most animals, the editor of *Cancer Research* invited him to write a special review paper in 1985. Duesberg spent many months compiling the evidence from the scientific literature. While he was working on this piece, the questions about HIV began intruding into his thinking ever more prominently. He finally decided to add a section arguing that HIV could not cause AIDS, citing data that showed HIV was inactive in the body, did not kill T-cells, and could not possibly have a long latent period before inducing AIDS.

He was still writing the paper in 1986 when he took nine months' leave from Berkeley to work in another retrovirus lab at the NIH facility in Bethesda, Maryland. As chance would have it, he worked in the building that housed Gallo's laboratory, though on a different floor. This afforded him many opportunities to test his growing suspicions of the virus-AIDS hypothesis. Not yet realizing Duesberg's intentions, Gallo invited him to be the featured speaker at one of his regular lab seminars. Gallo seemed to enjoy most of Duesberg's talk, which questioned the importance of cancer genes, and did not even become upset when Duesberg threw in a short criticism of the HIV-AIDS hypothesis at the end. Apparently, Gallo thought Duesberg was not really serious, merely dabbling for fun.

But the following weeks brought increasingly tense conversations between them in which Duesberg would constantly raise

new questions. One day such a discussion took place in the elevator, on the way to Gallo's lab. Gallo burst into such anger over Duesberg's persistence that he left the elevator on the wrong floor—missing the lab where he had worked for many years! Although Gallo increasingly resisted talking about HIV, several researchers in his lab privately admitted to Duesberg the enormous problem of not finding the virus active in the body. They knew perfectly well something had to give. Rather than abandon HIV, however, they told Duesberg they hoped to explain the problem using “cofactors” or other rationalizations. Naturally, these experiences began confirming Duesberg's suspicion that he had stumbled onto something profound.

Duesberg's twenty-two-page review paper appeared in the March 1987 issue of *Cancer Research*. Colleagues found the section on AIDS especially shocking, privately admitting the importance of the questions about HIV. To this very day, not one scientist has come forward to answer the paper. Traditionally, such deafening silence has been interpreted as a victory for the author, indicating the arguments to be irrefutable. However, despite being unable to find any flaws in the article, no researcher could afford to take on the powerful HIV-AIDS establishment. Unwilling to risk status and career by challenging the growing AIDS research structure, but having no arguments to defend the virus hypothesis, scientists chose the safety of continuing their studies of HIV, claiming that it was at least an “interesting” virus. Some researchers became quite sensitive about the virus hypothesis, reacting angrily to any criticisms.

The *Cancer Research* paper nevertheless generated some interest, and upon invitation Duesberg wrote a guest editorial in *Bio/Technology* that November. Again, no answer. The wide-circulation *Science* soon ran an article on the emerging controversy, placing Duesberg in a rather unsympathetic light. Prompted by Duesberg's letter in response, the editor decided to set up an official debate in this journal, which appeared in July of 1988. Duesberg was on one side, opposing Gallo, Howard Temin, and the epidemiologist William Blattner. Each side offered an opening

page and a rebuttal to the opposition's opening page; that was all. *Science* has thereafter refused to publish anything but an occasional letter on the topic, declaring it received as much coverage as it deserved.

Although before this exchange Duesberg still had doubts, he became thoroughly convinced the virus was harmless after seeing this faltering inability to answer his arguments. As he further immersed himself in the AIDS literature, the sheer volume of damning evidence became overwhelming. In a response to the short *Science* debate, he wrote an extended update paper, which after months of fighting he managed to publish in the *Proceedings of the National Academy of Sciences* in 1989. This paper was printed on the express condition that another virologist would respond with an equal rebuttal. Gallo himself promised such but has not delivered as of this date. Once again, no scientist has ever chosen to answer that piece nor to answer Duesberg's subsequent review papers in *Research in Immunology* or the *Proceedings*.

Only a few short, general responses to Duesberg have appeared in other journals: the brief debate forum in *Science*, short exchanges in some 1989 issues of the *Journal of AIDS Research*, terse letters in a May 1990 issue of the *New England Journal of Medicine*, a blatantly *ad hominem* attack in the pages of *Nature* during June of 1990, and a few editorials in 1993. But in December 1994, *Science* published an eight-page article on the "Duesberg phenomenon" by the journal's foremost AIDS journalist. The article acknowledges that "the Duesberg phenomenon has not gone away and may be growing."⁴² Although tendentious for the HIV hypothesis, the article made some telling concessions: "(i) According to some AIDS researchers [not all] HIV now [but not earlier, when it was named the AIDS virus] fulfills the classic postulates of... Koch," and (ii) "AZT and illicit drugs, which Duesberg argues can cause AIDS, don't cause the [*sic*] immune deficiency characteristic of that disease," knowing full well that about thirty different diseases are said to be "characteristic of the disease."⁴³

From these and excerpts of Gallo's own writings, the standard defense of the HIV-AIDS hypothesis can be reconstructed. None

of the most influential AIDS scientists has ever published a definitive defense of HIV, yet when confronted with the paradoxes they all answer with similar arguments. Otherwise, they prefer to ignore the questions.

The arguments for HIV fall into four categories.

1. Arguing for HIV by Ignoring the Facts

The case for HIV as an AIDS virus depends first on bypassing Koch's postulates. The most complete rationale for this is presented by Gallo in his 1991 book *Virus Hunting—AIDS, Cancer, and the Human Retrovirus: A Story of Scientific Discovery*, where he coolly disposes of these time-tested standards:

Rules were needed then, and can be helpful now, but not if they are too blindly followed. Robert Koch, a great microbiologist, has suffered from a malady that affects many other great men: he has been taken too literally and too seriously for too long. We forget at times that we have made great progress in the last century in developing tools, reagents, and diagnostic techniques far beyond Koch's wildest fantasies...

Koch's Postulates, while continuing to be an excellent teaching device, are far from absolute in the real world outside the classroom (and probably should not be in the classroom anymore except in a historical and balanced manner). They were not always fulfilled even in his time. Certainly, they did not anticipate the new approaches available to us, especially in molecular biology, immunology, and epidemiology, or the special problems created by viruses. They were, after all, conceived only for bacterial disease, and even here they often fail. Sometimes they are impossible to fulfill; many times one would not even want to try to do so; and sometimes they are quite simply erroneous standards.⁴⁴

But Koch's postulates consist of elementary logic. Whereas technology is continually being outdated, logic is permanent. Koch's rules, after all, simply restate the germ theory itself in

experimental terms. Gallo never tries to explain how logic would change over time; indeed, in this age of ultrasensitive biotechnology, such rules take on more importance than ever in sorting out relevant data from mere trivia. *Nor does Gallo offer any rigorous scientific rules to replace Koch's postulates, leaving HIV science with no standards at all.*

Gallo continues by misstating Koch's postulates, falsely claiming that a germ is required to cause a disease every single time it infects a new host. With most microbes, the majority of infected people or animals experience no symptoms; Koch's test only requires that some animals become sick when injected with a disease-causing germ or that vaccination prevents the illness. Gallo then cites false or misleading examples of germs that supposedly fail the postulates despite causing disease, pretending, for example, that the hepatitis and flu viruses cause no disease in animals. Gallo misses the point that the failure of a given germ to meet Koch's postulate does not call the postulate into question, but rather the germ as the cause of a disease. Or he draws examples from the "slow virus" hypotheses, including measles/SSPE, papilloma/cervical cancer, HTLV-I/leukemia, and Feline Leukemia Virus (see chapters 3 and 4). Or he cites diseases erroneously thought to result from bacteria, such as neurosyphilis (see chapter 2). In reality, all truly viral diseases do fulfill Koch's standards perfectly—yellow fever, measles, polio, chicken pox, herpes, hepatitis A and B, and flu, among others.

Gallo's "these postulates are too old" argument is repeated by English retrovirus hunter Robin Weiss and American CDC official Harold Jaffe: "What seems bizarre is that anyone should demand strict adherence to these unreconstructed postulates 100 years after their proposition."⁴⁵ Weiss and Jaffe also forget to explain how logical rules could become outdated and again proceed to misquote Koch and use misleading examples of disease-causing microbes supposedly failing the postulates.

It is generally assumed that stardom in a given field is directly proportional to knowledge: the more famous a person is, the more he knows about his field. However, a star is often born by a

coincidence in which the most desirable solution to problems is delivered to the best-prepared audience. To deliver such a popular solution requires a complete knowledge of the politics of science but not of science itself. As we shall see, Gallo and Montagnier fit the formula for scientific stardom in this regard exactly.

Both had studied retroviruses as causes of cancer for more than a decade when AIDS appeared. But neither one had studied other noninfectious causes of diseases, not even other viruses, nor have they treated AIDS patients after AIDS appeared. Retroviruses were their primary investment and their exclusive expertise.

Having persuaded himself to ignore the traditional rules of Robert Koch, Gallo joins with Luc Montagnier in substituting a previously unknown “postulate”:

That HIV is the cause of AIDS is by now firmly established. The evidence for causation includes the fact that HIV is a new pathogen, fulfilling the original postulate of “new disease, new agent.”⁴⁶

Superficially, it appears logical to postulate that a new virus would cause a new disease. However, Gallo and Montagnier’s argument fails because it ignores a multiplicity of facts:

(i) AIDS is not a disease. Instead, the AIDS syndrome is a steadily growing collection of (currently) about thirty “previously known” (old) diseases (see below). Surprisingly, in view of their notoriety for AIDS, neither Gallo nor Montagnier know the AIDS definition.

It is true, however, that the incidence of AIDS diseases has increased dramatically in the 1980s (Figure 2A) as intravenous drug use has increased and as both the consumption of recreational drugs used as sexual stimulants and the use of AZT as antiviral drug have increased in male homosexuals.

(ii) HIV is not a “new agent.” According to Farr’s law, a virus is new if the percentage of infected people increases rapidly over time—or “explodes” as the CDC predicted in the early days of

AIDS. A virus is old if the percentage of infected people is stable over time (Figure 1). Since the number of HIV-infected Americans has been an unchanging 1 million since HIV was able to be tested in 1985, HIV is an old virus in the United States (Figure 2A). In order to misjudge the age of HIV so grossly, Gallo and Montagnier must have been unaware of the epidemiology of HIV in the United States and unaware of Farr's law.

Gallo and Montagnier probably assumed HIV is new because it was newly discovered by them. But since the technology used to detect HIV is just as new as the discovery of HIV, there is another interpretation: Gallo and Montagnier discovered a previously unknown but old virus with a new technique. Their claim that HIV is new is just as naive as the claim of an astronomer that a previously unknown star is new because it became detectable with a new telescope.

Since HIV is old in the United States and the epidemic of AIDS diseases is new, HIV is not a plausible cause for a "new" rise of AIDS diseases in the United States.

(iii) AIDS is not an infectious, viral epidemic as Gallo and Montagnier assume. AIDS fails all epidemiological criteria of an infectious disease. Gallo and Montagnier completely ignore the evidence that the new AIDS epidemic could well be the consequence of the new recreational drug use epidemic that started in America after the Vietnam War. Apparently, neither Gallo nor Montagnier were aware of the "lifestyle hypothesis," which originally proposed that AIDS patients were suffering from drug diseases because all early AIDS patients were recreational drug users.⁴⁷

To distinguish between toxic drugs and toxic microbes, Gallo and Montagnier should have investigated whether AIDS is infectious or not. But Gallo and Montagnier completely ignored that AIDS does not meet even one of the classical epidemiological criteria of infectious diseases—possibly because they never considered nonviral causes of disease.

(iv) Considering that hundreds of known retroviruses are harmless

passenger viruses, one would have expected that the “leading” retrovirologists Gallo and Montagnier would have explained why they believe that HIV is fatally pathogenic. Yet all that Gallo and Montagnier had to offer in support of HIV pathology was their own credibility.

Indeed Gallo’s and Montagnier’s reasoning fits their narrow expertise exactly. Two leading retrovirologists agreeing on a retrovirus as the cause of AIDS and ignoring all competing retroviral and nonretroviral explanations. And for the leaders, ignorance is bliss.

2. Arguing for HIV Based on Inappropriate Models

When confronted with the paradoxes of HIV, its defenders simply reach for their bag of virus hypotheses, pulling out on demand a mixture of invented or misinterpreted models. They usually cite viral precedents of three types.

The first comes from the supposed “slow viruses,” which are used to justify the long latency period of HIV, but which fall apart in light of the evidence presented above.

The second model suggests HIV reactivation based on authentic prototypes. Herpes simplex virus, for example, can cause lesions even long after the first antibodies against the virus have been produced. However, this can happen only if the virus is reactivated because the original antibodies and anti-viral T-cells have dropped below a safe threshold level. After reactivation the virus multiplies into large numbers just as in the original infection. Using this model, HIV scientists justify both the latent period and antibody test in one breath. But herpes produces the same lesions upon first infecting the body as it does upon reactivation, and antibodies neutralize it both early and late. Herpes can only recur because it hides in certain nerve cells, waiting until some future opportunity when the host’s immune function is temporarily reduced. Once the immune system regains strength, the virus is again suppressed and the sores disappear. HIV, on the other hand, is alleged to kill its host only years after being neutralized, and even without reactivating. There is no HIV reactivation and no HIV in most AIDS patients.

The third virus model has been created only since the appearance of AIDS. Some animal retroviruses will cause “AIDS” when injected into hosts of the appropriate species. Simian immunodeficiency virus (SIV), a monkey retrovirus, attracts most of the attention. But these animal diseases can be called “AIDS” only by stretching the definition to extremes. They do not include most of the human AIDS conditions such as Kaposi’s sarcoma or dementia. Rather, the animal symptoms usually resemble the flu: The animals become sick within days or not at all, without long latent periods; some animals recover by raising an immune response and never suffer a relapse; and those that die must be injected with large quantities of the virus while very young, before they have developed any immune system at all. In the wild, their cousins retain antibodies against SIV all their lives without ever becoming sick from the virus. These laboratory diseases are, in all respects, very traditional viral flu-like diseases, but HIV scientists rename them “AIDS.”⁴⁸

3. Arguing for HIV Based on Evasion

Lacking answers to Koch’s postulates and authentic virus precedents, AIDS scientists resort to a variety of excuses. The standard evasions fall into four general categories: the arguments from unknowns, from speculation, from authority, and from irresponsibility.

The argument from unknowns makes the obvious point that scientists never know everything and implies that the HIV-AIDS question is therefore somehow unimportant now, since it eventually will be resolved through more research. According to this argument, the issue is not whether, but how, HIV causes AIDS; paradoxes therefore merely prove that further research is needed and that scientific knowledge will consequently expand, not that the virus is itself in question. William Blattner and Robert Gallo of the National Cancer Institute joined with fellow retrovirologist Howard Temin in using typical arguments from unknowns:

Biology is an experimental science, and new biological phenomena are continually being discovered... Thus, one cannot conclude that HIV-1 does or does not cause AIDS from Duesberg's "cardinal rules" of virology...

Duesberg's descriptions of the properties of viruses [are] in error and [provide] no distinction between knowing the cause of a disease, that is, its etiology ["whether"], and understanding the pathogenesis of this disease ["how"]. There are many unanswered questions about the pathogenesis of AIDS, but they are not relevant to the conclusion that HIV causes AIDS.

The CDC definition of AIDS has been revised several times as new knowledge has become available and will undoubtedly be revised again.⁴⁹

Likewise, Robin Weiss and Harold Jaffe assert:

It is unwise to conclude that because we do not understand the pathogenesis of HIV in molecular detail, it is therefore harmless... So Duesberg is right to draw attention to our ignorance of how HIV causes disease, but he is wrong to claim that it does not.

One need not harp upon molecular quibbles, important though these are for directing research to the prevention or amelioration of HIV infection. To deny the role of HIV in AIDS is deceptive.⁵⁰

It should be clear by now that the questions surrounding the alleged pathogenesis of HIV are too many and too substantial to be dismissed as mere "quibbles." To assert the role of HIV in AIDS is unscientific, particularly since the guardians of the HIV hypothesis have never suggested which standards could prove the virus harmless. Until they propose a scientific experiment that could disprove the HIV hypothesis, they convey the implicit message that they will accept no evidence against it whatsoever.

The argument from speculation is used more often than any other. It uses specialized terms that make it difficult for outsiders to understand, responding to any paradox with one untested

assumption after another. For instance, if little or no HIV can be found in the body, scientists propose hidden reservoirs and special routes of infection. If only antibodies against HIV can be found, researchers call them “nonneutralizing” (or ineffective) antibodies and assert that the virus mutates too fast for the antibodies to keep up. If the virus does not make animals sick or kill cells in culture, then researchers claim that the virus somehow makes fine distinctions between humans and chimpanzees, something no other virus can do. All these hypotheses are constantly being disproved or shown to be irrelevant, but the reservoir of new evasions is inexhaustible.

The argument from authority cites the “overwhelming evidence” for HIV, without becoming too specific. In another form, it rebuffs inquisitive epidemiologists for lacking clinical experience while bypassing medical critics for having no epidemiological training. In other words, unless one is an expert in everything, one may not question anything. This response alludes to esoteric scientific data as a reason for critics to remain silent. Blattner, Gallo, and Temin provide perfect examples: “In summary, although many questions remain about HIV and AIDS, a huge and continuously growing body of scientific evidence shows that HIV causes AIDS,” and “Thus, we conclude that there is overwhelming evidence that HIV causes AIDS.”⁵¹

The argument from irresponsibility serves as the answer of last resort. In the vein of a “better safe than sorry” warning, such HIV defenders as Weiss and Jaffe assert the weapon of fear:

If he [Duesberg] and his supporters belittle “safe sex,” would have us abandon HIV screening of blood donations, and curtail research into anti-HIV drugs and vaccines, then their message is perilous.⁵²

The irony, as will be reviewed later, lies in the danger of the officially approved measures to combat HIV, which are themselves costing lives.

4. Arguing for HIV Based on Antibody Correlations

The three basic arguments outlined above clearly answer no questions. The only positive evidence in favor of the virus-AIDS hypothesis is found in epidemiology, the study of disease epidemics. This field operates entirely by correlation: According to AIDS officials, where HIV goes, AIDS follows. Despite all the sophisticated biotechnology and vast investment in virology, the best evidence for HIV is only by correlation with antibodies present against it. Ironically, the point is made by retrovirologists Blattner, Gallo, and Temin: “The strongest evidence that HIV causes AIDS comes from prospective epidemiological studies that document the absolute requirement for HIV infection for the development of AIDS.”⁵³ Or, as stated by Weiss and Jaffe, “The evidence that HIV causes AIDS is epidemiological and virological, not molecular.”⁵⁴ Gallo again emphasizes the point in his book, declaring correlation to be “one hell of a good beginning.”⁵⁵

What sort of correlations seem so convincing to AIDS officials? The one usually cited first might be called the “geographic overlap.” According to Blattner, Gallo, and Temin, “epidemiological data show that AIDS and HIV infection are clustered in the same population groups and in specific geographic locations and in time. Numerous studies have shown that in countries with no persons with HIV antibodies there is no AIDS, and in countries with many persons with HIV antibodies there is much AIDS. Additionally, the time of occurrence of AIDS in each country is correlated with the time of introduction of HIV into that country; first HIV is introduced, then AIDS appears.”⁵⁶ The three HIV advocates fail to mention, however, that a disease is only recorded as AIDS if antibodies to HIV are also found.

Second, a tighter association is recorded for individual people: Every victim of AIDS has antibodies against HIV, whereas most healthy people do not. This apparently perfect correlation exists in selected surveys that follow people at risk for AIDS. But no national AIDS statistics exist that even document how well HIV compares with AIDS.⁵⁷ Clearly, most of the seventeen million healthy HIV-positive humans have yet to develop AIDS.

Altogether fewer than 6 percent (about one million) have developed AIDS in the past ten years.⁵⁸ Furthermore, thousands of clinically diagnosed AIDS patients are HIV-free.

A third argument evokes powerful emotional sentiments without much substance and works surprisingly well not only on the lay public, but on scientists as well. When challenged that only people with serious health risks develop AIDS, experts answer with anecdotes, even though the same medical officials will consider anecdotes a worthless type of evidence in any other debate. An anecdotal story is one individual case chosen to prove the absence of other health risks, implying HIV was the only factor that could have led to disease. So, for example, epidemiologists will describe a baby contracting HIV and subsequently developing AIDS. But in a nation of 250 million people, a few anecdotal cases can always be found to support any medical view.

Fourth, AIDS epidemiologists point to their prospective studies, in which the supposedly conclusive proof of the HIV hypothesis can be found. These studies monitor two groups of people over time, one of HIV-positive patients and the other of HIV-negative people in the same age group. According to such reports, the infected people develop AIDS while their uninfected counterparts do not. But all the reports that have also investigated drug use and other noncontagious AIDS risks have found that AIDS correlates with those factors just as well, if not better, than HIV (see chapters 8–10).⁵⁹

Yet, these HIV-AIDS correlations have proven to be the most powerful arguments to scientists and laymen alike. Only a more complete picture can expose the misleading nature of this sloppy epidemiology.

THE OTHER STATISTICS

In one strange sense, officials do refer to some genuine correlations between HIV and AIDS. The syndrome, for example, is rarely found in any nation or individual apart from HIV infection. Indeed, the virus and the syndrome correlate with near-textbook perfection, ironically illustrating the most fundamental problem

with the entire virus-AIDS hypothesis—the connection was artificially constructed.

AIDS is a syndrome of about thirty diseases, not a disease. It displays no unique combination of diseases in the patient. Clinically, it is identified by the diagnosis of specific diseases known to medical science for decades or centuries. The CDC has several times increased—but never decreased—the official list of AIDS indicator diseases, most recently on January 1, 1993 (See Table 2). The list now includes brain dementia, chronic diarrhea, cancers such as Kaposi's sarcoma and several lymphomas, and such opportunistic infections as *Pneumocystis carinii* pneumonia, cytomegalovirus infection, herpes, candidiasis (yeast infections), and tuberculosis. Even low T-cell counts in the blood can now be called "AIDS," with or without real clinical symptoms. Cervical cancer has recently been added to the list, the first AIDS disease that can affect only one gender (in this case, women). The purpose behind adding this disease was entirely political, admittedly to increase the number of female AIDS patients, creating an illusion that the syndrome is "spreading" into the heterosexual population.⁶⁰ Originally, the AIDS diseases were tied together because they were all increasing within certain risk groups, but today they are assumed to derive from the common basis of immune deficiency. The overlap between AIDS and certain risk groups still holds true but, as pointed out in Table 1, a significant number of these diseases are not products of weakened immune systems.

According to Blattner, Gallo, and Temin, "The CDC definition of AIDS has been revised several times as new knowledge has become available and will undoubtedly be revised again."⁶¹ However, neither the CDC nor other advocates of the HIV hypothesis ever identify the "new knowledge" about HIV that mandates these revisions. It is also remarkable that such "new knowledge" always drives the list of AIDS-defining illnesses upward. Not once has an AIDS-defining disease been subtracted in the light of "new knowledge" about HIV. Irrespective of the undisclosed gains in knowledge about HIV, one thing is clear—the repeated upward adjustments in the definition of AIDS have substantially increased

TABLE 2

Chronology of the CDC's AIDS definitions

Year	Diseases	HIV antibody
1983	<i>Protozoal and helminthic infections</i>	
	1 Cryptosporidiosis, intestinal, causing diarrhea for more than a month	
	2 <i>Pneumocystis carinii</i> pneumonia	
	3 Strongyloidosis, causing pneumonia, central nervous system infection, or disseminated infection	
	4 Toxoplasmosis, causing pneumonia or central nervous system infection	
	<i>Fungal infections</i>	
	5 Candidiasis, causing esophagitis	
	6 Cryptococcosis, causing central nervous system or disseminated infection	not required
	<i>Bacterial infection</i>	
	7 "Atypical" mycobacteriosis, causing disseminated infection	
	<i>Viral infections</i>	
	8 Cytomegalovirus, causing pulmonary, gastrointestinal tract, or central nervous system infection	
9 Herpes simplex virus, causing chronic mucocutaneous infection with ulcers persisting more than one month or pulmonary, gastrointestinal tract, or disseminated infection		
10 Progressive multifocal leukoencephalopathy (presumed to be caused by a papovavirus)		
<i>Cancer</i>		
11 Kaposi's sarcoma in persons less than 60 years of age		
12 Lymphoma, primary, of the brain		
1985	13 Histoplasmosis	
	14 Isosporiasis, chronic intestinal	
	15 Lymphoma, Burkitt's	
	16 Lymphoma, immunoblastic	required
	17 Bronchial or pulmonary candidiasis	
	18 Chronic lymphoid interstitial pneumonitis (under 13 years of age)	
1987	19 Encephalopathy, dementia, HIV-related	

	20	Mycobacterium tuberculosis any site (extrapulmonary)	
	21	Wasting syndrome, HIV-related	required
	22	Coccidiomycosis, disseminated or extrapulmonary	
	23	Cryptococcosis, extrapulmonary	
	24	Cytomegalovirus, other than liver, spleen, or nodes	
	25	Cytomegalovirus retinitis	
	26	Salmonella septicemia, recurrent	
<hr/>			
1993	27	Recurrent bacterial pneumonia	
	28	Mycobacterium tuberculosis any site (pulmonary)	
	29	Pneumonia, recurrent	
	30	Invasive cervical cancer	required
	31	T-cell count is less than 200 cells per microliter or less than 14 percent of the expected level	
<hr/>			

References

Selik, R. M., Haverkos, H. W., and Curran, J. W. (1984). "Acquired Immune Deficiency Syndrome, (AIDS) in the United States, 1978-1982." *The American Journal of Medicine* 76: 493-500.

Institute of Medicine and National Academy of Sciences (1986). *Confronting AIDS*. National Academy Press, Washington, DC.

Institute of Medicine (1988). *Confronting AIDS-Update 1988*. National Academy Press, Washington, DC.

Centers for Disease Control and Prevention (1992). Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS among Adolescents and Adults. *Morb Mort Weekly Rep* 41 (No. RR17): 1-19.

Duesberg, P., and Yiamouyiannis, J. (1995). *AIDS*. Health Action Press, Delaware.

the American AIDS statistics while HIV infections have remained completely flat since 1985 (see Figure 2A).

The increasing numbers of new AIDS cases until 1993 have largely been products of the artificial AIDS definitions (see Figure 2A). Each alteration in that definition has added, not subtracted, diseases to the diagnostic list. Every time the CDC needs higher rates of new AIDS cases, it expands that definition once again, and more diseases are reclassified into the syndrome. With the stroke of a pen an illusion of the spread of AIDS is created, prominent officials explain the revisions as products of our growing scientific knowledge, and the lay public feels reassured that federal efforts are justified—or perhaps even a little too slow.

One might ask how a doctor would distinguish between an AIDS-related tuberculosis and a traditional one. Clinically, the symptoms are identical, so the CDC has stipulated in its current definition that the tuberculosis must be renamed “AIDS” if antibodies against HIV are also found in the patient. In the absence of previous HIV infection, the disease is classified under its old name, in this case “tuberculosis,” and treated accordingly. AIDS, therefore, can never be found apart from HIV infection—entirely by definition!

AIDS officials neglect to mention this crucial fact partly from ignorance, most never having read the definition carefully and in some cases precisely because it shines a disturbing light onto their supposedly perfect epidemiological coincidence between the virus and AIDS. *The observation that AIDS always follows HIV in each nation becomes trivial, since testing for antibodies is followed by a renaming of indigenous diseases.*

The real epidemiological questions, then, must be shifted away from any “correlation” between antibodies against HIV and AIDS to a correlation between HIV and the separate AIDS-diagnostic diseases. Does infection with the virus, independently of any other health risks, lead to an increased risk of contracting pneumonia, cancer, or other diseases? Is HIV new and found in all recent outbreaks of these diseases? Is HIV infection nearly always fatal?

The latter question can be answered most easily. Since the HIV test was made available in 1985, the CDC has officially estimated about one million Americans to be HIV positive, a figure that has not changed with the accumulation of testing data or the passage of ten years (see Figure 2A). Of these, only about four hundred thousand had been diagnosed with AIDS by the end of 1994. But this statistic does not subtract the normal incidence of the thirty AIDS-defining diseases in one million people over ten years. Two-thirds of HIV-positive Americans have not developed any of the AIDS diseases since 1985 (even including the most recent expansion in the AIDS definition).

Nor will most of them do so. The numbers of new AIDS cases have clearly been leveling off for some time now, although

different analysts will place the peak at different times. Michael Fumento, the Colorado-based lawyer who gained some media notoriety with his 1989 book *The Myth of Heterosexual AIDS*, draws a curve with its peak in 1987;⁶² two epidemiologists, in a 1990 paper in the *Journal of the American Medical Association*, suggest 1988 as the year of leveling.⁶³ The CDC observed a leveling off in 1994.⁶⁴ In any case, a slowly increasing forty thousand to fifty thousand new cases of AIDS—4 percent to 5 percent of the infected subpopulation—have appeared before the 1993 revision of the AIDS definition—hardly the “explosion” that AIDS, as a new infectious disease, was once predicted to show. The enormous gap between HIV-infected people and AIDS patients has induced the CDC to play more tricks with the numbers; at the time of this writing, the CDC is considering lowering its official estimate of one million HIV-positive Americans to a new total of six hundred thousand to eight hundred thousand.⁶⁵

Part of the AIDS scare results from the way the numbers are reported. Rather than giving the numbers of new AIDS cases each year, CDC and other officials use the cumulative total for the current year added to the figures for all years previous, including those victims already dead. So where the annual numbers would remain constant in the first case, the number actually reported to the public grows with each passing year. Such calculation gives the overwhelming but false impression that AIDS is spreading, since the cumulative numbers can only go up. Given enough time, such accounting methods will boost the total AIDS count higher than the number of HIV-positive people. If this method were applied to count the American population, the cumulative number of newborns over several decades would eventually exceed the total number of Americans alive.

The commonly cited 50 percent to 100 percent death rate from HIV has been derived not from national statistics, but from studies on carefully selected cohorts of people. Several ongoing epidemiological studies have for years been observing hundreds, or at most thousands, of homosexual men at high risk for AIDS. Large proportions of the men in these studies have already been infected

with HIV. But virtually all the subjects also admit to years of heavy drug abuse, extremely promiscuous sexual activity, and long histories of venereal diseases. Indeed, one major study was specifically organized around homosexual men with repeated bouts of hepatitis B. Researchers calculate the high fatality rate of HIV infection from these health risk groups, casually extrapolating these numbers to average, heterosexual HIV-positives—thus the discrepancy with the higher survival rate among the nation's one million HIV-positives.

The national AIDS figures fall well short of a virus with a nearly 100 percent fatality rate. But rather than abandon the hypothesis, the experts have chosen to revise the parameters of HIV infection. The latency period was originally calculated in 1984 on the basis of tracing sexual contacts, finding homosexual men developing AIDS an average of ten months after their last sexual contacts with other AIDS patients.⁶⁶ This “incubation period” has since been stretched to ten to twelve years between HIV infection and disease. For each year that passes without the predicted explosion in AIDS cases, approximately one more year is added to this incubation time. Even this is insufficient; with only 5 percent of infected Americans developing AIDS each year, the average latent period would have to be revised up to some twenty years for 100 percent to become sick.

A deeper look at the disease risk of infected populations reveals stranger paradoxes yet. The probability of developing AIDS varies radically between different HIV-positive populations. Sub-Saharan Africa, with infection rates approaching 30 percent of the population in some areas, has reported only approximately 250,000 AIDS cases to the World Health Organization in the past decade. This stands against six million to eight million Africans infected with HIV since the mid-1980s, whereas more Americans (now over 400,000) have contracted AIDS in a country with only one million HIV-positives. AIDS patients in Zaire, with about three million HIV-infected people, number only in the hundreds; Uganda, internationally considered a model for accurate testing and reporting, had by 1990 only generated some 8,000 AIDS cases out of one

million HIV-positives. Roughly 360,000 infected Haitians have produced only a few hundred AIDS patients. In the industrial nations, homosexuals, heroin addicts, and hemophiliacs face greater probabilities of developing AIDS than do HIV-positive individuals without extraordinary health risks. And infants have a much shorter average latent period—two years, as opposed to the ten years in adults. No virus, including HIV, could possibly discriminate so enormously based on such subtle distinctions between its hosts.

HIV would need to perform other miracles to cause AIDS. Virtually all diagnoses of Kaposi's sarcoma are made in homosexuals, not in the other AIDS risk groups. Intravenous drug addicts disproportionately suffer from tuberculosis, Haitians from toxoplasmosis, and hemophiliacs from pneumonias. African AIDS diseases are basically different, manifesting as tuberculosis, fever, diarrhea, and a slim disease, unlike our wasting syndrome. A homosexual with HIV who may develop Kaposi's sarcoma can donate blood for a hemophiliac. But no hemophiliac has ever developed Kaposi's sarcoma from a blood transfusion. Instead he is more likely to develop pneumonia, if he contracts anything at all. Only HIV is common to both victims.

No virus could possibly make such distinctions between its hosts. A more likely hypothesis would blame the health risks specific to each group for their different diseases. If the same diseases can be found on the rise in the same risk groups, but also in people without HIV, then the virus would appear to be a harmless passenger.

The evidence bears this out. Hemophiliacs without HIV develop progressive immune degeneration just like the infected ones.⁶⁷ HIV-negative babies of infected mothers develop the same dementia-related symptoms as their HIV-positive siblings. Heroin addicts contract the same pneumonias, herpes infections, weight loss, and tuberculosis with or without the virus, and uninfected homosexuals with Kaposi's sarcoma are now being reported. Outbreaks of pneumonias or tuberculosis in recent years have included as many people without the virus as those with it.

Thousands of central Africans with “slim disease” have now been tested for HIV, and over half are completely negative; given the relatively high cost of HIV antibody tests, most African cases must be diagnosed by symptoms and remain untested for the virus.⁶⁸ In the industrial world, upward of one-quarter of all AIDS patients remain untested for the antibodies against HIV, with their doctors merely assuming the virus is present. The existing scientific literature records more than forty-six hundred cases of AIDS-defining conditions in people never infected by HIV.⁶⁹ With various AIDS-type diseases increasing in the risk groups even apart from HIV, the virus appears ever less relevant.

All circumstantial evidence aside, the ultimate epidemiological test for HIV would be a case-controlled comparison. In such a study, a large number of infected people would be monitored over time and compared with a large number of uninfected people. They would be matched for age, sex, income, and all other health risks such as drug use. Hemophilia and other medical complications would be excluded. If HIV were truly harmful, the infected group would develop AIDS and the uninfected would not. Scientists would conduct this type of study even before testing Koch’s postulates. But no such study testing HIV as an AIDS virus can be found in the more than one hundred thousand studies to date on this virus!⁷⁰

When confronted with the whole of the evidence against them, defenders of the HIV hypothesis will sometimes cite studies comparing notorious AIDS risk groups, with and without the virus, to show that only those infected will degenerate and die. But none of the vast number of such prospective studies has actually matched two groups for the health risks that might cause AIDS. They have been designed merely to compare the symptoms of AIDS patients with normal people in the same age group, not to determine the cause of the syndrome. Such studies, their marginal and questionable value notwithstanding, are too often quoted by some researchers as proof of the virus-AIDS hypothesis.⁷¹

NO AIDS VIRUS AT ALL

Given that HIV fails all standards of scientific evidence as an “AIDS virus,” could another, possibly unidentified, virus cause AIDS instead? Such a microbe would have to possess amazing and unprecedented qualities, for AIDS does not behave as a contagious disease at all.

The sexual revolution of the past twenty years has caused increases in all the major venereal diseases, including syphilis, gonorrhea, chlamydia, and genital warts. The same has occurred with hepatitis B. All of these infectious diseases have spread far beyond their original reservoirs into the general population and affect men and women nearly equally.

AIDS, however, has remained absolutely fixed in its original risk groups. Today, a full decade after it first appeared, the syndrome is diagnosed in homosexuals, intravenous drug users, and hemophiliacs some 95 percent of the time, just as ten years ago. Nine out of every ten AIDS patients are male, also just as before. Even the very existence of a “latent period” strongly suggests that years of health abuse are required for such fatal conditions. Among most AIDS patients in the United States and Europe, one extremely common health risk has been identified: the long-term use of hard drugs (the evidence for this new AIDS hypothesis will be presented in chapters 8 and 11). AIDS is not contagious nor is it even a single epidemic.

Tragic deaths, time and money wasted, hysterical public debate over a harmless virus—these have been the fruits borne of a scientific establishment grown too large for genuine science. The creative pursuit of knowledge has been swallowed to satisfy careerism and its voracious appetite for job security, grant money, financial benefits, and prestige. But the monster is twice guilty, for it also destroys or marginalizes those few scientists daring to ask questions. These dissidents against the HIV hypothesis are the subject of the next chapter.