OUT OF CONTROL

AIDS and the corruption of medical science

By Celia Farber

Joyce Ann Hafford was a single mother living alone with her thirteen-year-old son, Jermal, in Memphis, Tennessee, when she learned that she was pregnant with her second child. She worked as a customer service representative at a company called CMC Call Center; her son was a top student, an athlete and musician. In April 2003, Hafford, four months pregnant, was urged by her obstetrician to take an HIV test. She agreed, even though she was healthy and had no reason to think she might be HIV positive. The test result came up positive, though Hafford was tested only once, and she did not know that pregnancy itself can cause a false positive HIV test. Her first thought was of her unborn baby. Hafford was immediately referred to an HIV/AIDS specialist, Dr. Edwin Thorpe, who happened to be one of the principal investigators recruiting patients for a clinical trial at the University of Tennessee Medical Group that was sponsored by the Division of AIDS (DAIDS)—the chief branch of HIV/AIDS research within the National Institutes of Health.

The objective of the trial, PACTG 1022, was to compare the "treatment-limiting toxicities" of two anti-HIV drug regimens. The core drugs being compared were nelfinavir (trade name Viracept) and nevirapine (trade name Viramune). To that regimen, in each arm, two more drugs were added—zidovudine (AZT) and lamivudine (Epivir) in a branded combination called Combivir. PACTG 1022 was a "safety" trial as well as an efficacy trial, which means that pregnant women were being used as research subjects to investigate "safety" and yet the trial was probing the outer limits of bearable toxicity. Given the reigning beliefs about HIV's pathogenicity, such trials are fairly commonplace, especially in the post-1994 era, when AZT was hailed for cutting transmission rates from mother to child.

The goal of PACTG 1022 was to recruit at least 440 pregnant women across the nation, of which 15 were to be enrolled in the University of Tennessee Medical Group. The plan was to assign the study's participants to one of two groups, with each receiving three HIV drugs, starting as early as ten weeks of gestation. Of the four drugs in this study, three belong to the FDA's category "C," which means that safety to either mother or fetus has not been adequately established.

Joyce Ann Hafford was thirty-three years old and had always been healthy. She showed no signs of any of the clinical markers associated with AIDS—her CD4 counts, which measure the lymphocytes that are used to indicate how strong a person's immune system is, and which HIV is believed to slowly corrode, were in the

Celia Farber is a writer based in New York City. A collection of her AIDS reporting, Serious Adverse Events, *is forthcoming from Melville House.*

Illustrations by Danijel Zezelj

normal range, and she felt fine. In early June 2003, she was enrolled in the trial and on June 18 took her first doses of the drugs. "She felt very sick right away," recalls her older sister, Rubbie King. "Within seventy-two hours, she had a very bad rash, welts all over her face, hands, and arms. That was the first sign that there was a problem. I told her to call her doctor and she did, but they just told her to put hydrocortisone cream on it. I later learned that a rash is a very bad sign, but they didn't seem alarmed at all."

Hafford was on the drug regimen for thirty-eight days. "Her health started to deteriorate from the moment she went on the drugs," says King. "She was always in pain, constantly throwing up, and finally she got to the point where all she could do was lie down." The sisters kept the news of Hafford's HIV test and of the trial itself from their mother, and Hafford herself attributed her sickness and nausea to being pregnant. She was a cheerful person, a non-complainer, and was convinced that she was lucky to have gotten into this trial. "She said to me, 'Nell' —that's what she called me—'I have got to get through this. I can't let my baby get this virus.' I said, 'Well, I understand that, but you're *awful* sick.' But she never expressed any fear because she thought this was going to keep her baby from being HIV positive. She didn't even know she was in trouble."

On July 16, at her scheduled exam, Hafford's doctor took note of the rash, which was "pruritic and macular-papular," and also noted that she was suffering hyperpigmentation, as well as ongoing nausea, pain, and vomiting. By this time all she could keep down were cans of Ensure. Her blood was drawn for lab tests, but she was not taken off the study drugs, according to legal documents and internal NIH memos.

Eight days later, Hafford went to the Regional Medical Center "fully symptomatic," with what legal documents characterize as including: "yellow eyes, thirst, darkening of her arms, tiredness, and nausea without vomiting." She also had a rapid heartbeat and difficulty breathing. Labs were drawn, and she was sent home, still on the drugs. The next day, July 25, Hafford was summoned back to the hospital after her lab reports from nine days earlier were finally reviewed. She was admitted to the hospital's ICU with "acute and sub-acute necrosis of the liver, secondary to drug toxicity, acute renal failure, anemia, septicemia, premature separation of the placenta," and threatened "premature labor." She was finally taken off the drugs but was already losing consciousness. Hafford's baby, Sterling, was delivered by C-section on July 29, and she remained conscious long enough not to hold him but at least to see him and learn that she'd had a boy. "We joked about it a little, when she was still coming in and out of consciousness in ICU," Rubbie recalls. "I said to her, 'You talked about me so much when you were pregnant that that baby looks *just* like me.'" Hafford's last words were a request to be put on a breathing tube. "She said she thought a breathing tube might help her," says Rubbie. "That was the last conversation I had with my sister." In the early morning hours of August 1, Rubbie and her mother got a call to come to the hospital, because doctors had lost Hafford's pulse. Jermal was sleeping, and Rubbie woke her own daughter and instructed her not to tell Jermal anything yet. They went to the hospital, and had been there about ten minutes when Joyce Ann died.

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Rubbie recalls that the hospital staff said they would clean her up and then let them sit with her. She also remembered a doctor who asked for their home phone numbers and muttered, "You got a lawsuit." (That person has not resurfaced.) They hadn't been sitting with Hafford's body long when a hospital official came in and asked the family whether they wanted an autopsy performed. "We said yes, we sure do," she says. The hospital official said it would have to be at their expense—at a cost of $3,000. "We said, 'We don't have $3,000.' My sister didn't have any life insurance or anything," says Rubbie. "She had state health care coverage, and we were already worried about how to get the money together to bury her." Consequently, no autopsy was done. There was a liver biopsy, however, which revealed, according to internal communiqués of DAIDS staff, that Hafford had died of liver failure brought on by nevirapine toxicity.

And what was the family told about the cause of Hafford's death?

"How did they put it?" Rubbie answers, carefully. "They told us how safe the drug was, they never attributed her death to the drug itself, at all. They said that her disease, AIDS, must have progressed rapidly." But Joyce Ann Hafford never had AIDS, or anything even on the diagnostic scale of AIDS. "I told my mom when we were walking out of there that morning," Rubbie recalls, "I said, 'Something is wrong.' She said, 'What do you mean?' I said, 'On the one hand they're telling us this drug is so safe, on the other hand they're telling us they're going to monitor the other patients more closely. If her disease was progressing, they could have changed the medication.' I knew something was wrong with their story, but I just could not put my finger on what it was."

When they got home that morning, they broke the news to Jermal. "I think he cried the whole day when we told him," Rubbie recalls. "My mom had tried to prepare him. She said, 'You know, Jermal, my mom died when I was very young,' but he was just devastated. They were like two peas in a pod those two. You could never separate them." Later on, Jermal became consumed with worry about how they would bury his mother, for which they had no funds and no insurance. The community pitched in, and Hafford was buried. "I haven't even been able to go back to her grave since she passed," says Rubbie.

Rubbie King is haunted by many questions, including whether her sister

was really infected with HIV,[1] and also what the long-term damage might be to Sterling, whom Rubbie is now raising, along with Jermal and her own child. Sterling, in addition to the drugs he was exposed to in the womb, was also on an eight-week AZT regimen after birth. One of the reasons the family suspects Hafford may have been a false positive is that St. Jude's Children's Research Hospital has not released Sterling's medical records, and although they have been told that he is now HIV negative, they never had any evidence that he was even born positive. (All babies born to an HIV-positive mother are born positive, but most become negative within eighteen months.)

Hafford's family was never told that she died of nevirapine toxicity. "They never said that. We never knew what she had died of until we got the call from [AP reporter] John Solomon, and he sent us the report," says Rubbie King. "It was easier to accept that she died of a lethal disease. That was easier to handle." The family has filed a $10 million lawsuit against the doctors who treated Hafford, the Tennessee Medical Group, St. Jude's Children's Research Hospital, and Boehringer Ingelheim, the drug's manufacturer.[2]

Rubbie King made a final, disturbing discovery when she was going through Hafford's medical records: In addition to discovering that her sister had only ever been given a single HIV test, she also came across the fifteen-page consent form, which was unsigned.

On August 8, 2003, Jonathan Fishbein, who had recently taken a job as the director of the Office for Policy in Clinical Research Operations at DAIDS, wrote an email to his boss, DAIDS director Ed Tramont, alerting him that "there was a fulminant liver failure resulting in death" in a DAIDS trial and that it looked like "nevirapine was the likely culprit." He said that the FDA was being informed. He was referring to Joyce Ann Hafford. Tramont emailed him back, "Ouch. Not much wwe can do about dumd docs!"

This email exchange came to light in December 2004, when AP reporter John Solomon broke the story that Fishbein was seeking whistle-blower protection, in part because he had refused to sign off on the reprimand of an NIH officer who had sent the FDA a safety report concerning the DAIDS trial that launched the worldwide use of nevirapine for pregnant women. The study was called HIVNET 012, and it began in Uganda in 1997.

The internal communiqués from DAIDS around the time of Hafford's death made it clear that doctors knew she had died of nevirapine toxicity. Tramont's reply to Fishbein suggests that he thought blame could be placed squarely with Hafford's doctors, but it

[1] HIV tests detect footprints, never the animal itself. These footprints, antibodies, are identified by means of molecular protein weights, and were limited to two in 1984, when the first test was developed and patented, but over the years expanded to include many proteins previously not associated with HIV. Like most Americans, Hafford thought that a single HIV-positive test meant that she "had" HIV—a surefire death sentence. But a majority of HIV-positive tests, when retested, come back indeterminate or negative. In many cases, different results emerge from the same blood tested in different labs. There are currently at least eleven different criteria for how many and what proteins at which band density signal "positive." The most stringent criteria (four bands) are upheld in Australia and France; the least stringent (two bands), in Africa, where an HIV test is not even required as part of an AIDS diagnosis. The U.S. standard is three reactive bands. It has been pointed out that a person could revert to being HIV negative simply by buying a plane ticket from Uganda to Australia.

[2] Dr. Thorpe declined to comment, citing ongoing litigation, as did the Tennessee Medical Group, the Regional Medical Center at Memphis, and St. Jude's Children's Research Hospital.

was the NIH itself that had conceived of the study as one that tested the "treatment-limiting toxicities" of HIV drugs in pregnant women.

The conclusion of the PACTG 1022 study team was published in the journal *JAIDS* in July of 2004. "The study was suspended," the authors reported, "because of greater than expected toxicity and changes in nevirapine prescribing information." They reported that within the nevirapine group, "one subject developed fulminant hepatic liver failure and died, and another developedStevens-Johnson syndrome." Stevens-Johnson syndrome is skin necrolysis—a severe toxic reaction that is similar to internal third-degree burns, in which the skin detaches from the body. Another paper, entitled "Toxicity with Continuous Nevirapine in Pregnancy: Results from PACTG 1022," puts the results in charts, with artful graphics. A small illustration of Hafford's liver floats in a box, with what looks like a jagged gash running through it. Four of the women in the nevirapine group developed hepatic toxicity.

As Terri Schiavo lay in her fourteenth year of a persistent vegetative state, and the nation erupted into a classically American moral opera over the sanctity of life, Joyce Ann Hafford's story made only a fleeting appearance—accompanied by a photo of her holding a red rose in an article that was also written by the AP's John Solomon. But soon a chorus of condemnation was turned against those who were sensationalizing Hafford's death and the growing HIVNET controversy to condemn nevirapine, which had been branded by the AIDS industry as a "life-saving" drug and a "very important tool" to combat HIV in the Third World.

So-called community AIDS activists were sprung like cuckoo birds from grandfather clocks at the appointed hour to affirm the unwavering AIDS cathechism: AIDS drugs save lives. To suggest otherwise is to endanger millions of African babies. Front and center were organizations like the Elizabeth Glaser Pediatric AIDS Foundation, which extolled the importance of nevirapine. Elizabeth Glaser's nevirapine defenders apparently didn't encounter a single media professional who knew, or cared, that the organization had received $1 million from nevirapine's maker, Boehringer Ingelheim, in 2000.[3] This was no scandal but simply part of a landscape. Pharmaceutical companies fund AIDS organizations, which in turn are quoted uncritically in the media about how many lives their drugs save. This time the AIDS organizations were joined by none other than the White House, which was in the midst of promoting a major program to make nevirapine available across Africa.[4]

America is a place where people rarely say: *Stop*. Extreme and unnatural things happen all the time, and nobody seems to know how to hit the brakes. In this muscular, can-do era, we are particularly prone to the seductions of the pharmaceutical industry, which has successfully marketed its ever growing arsenal of drugs as the latest American *right*. The buzzword is "access,"

[3] *"Our mission of eradicating AIDS is always informed and driven by the best available science, not by donations," said Mark Isaac, Elizabeth Glazer's vice president for policy, when asked to comment. "The full body of research, as well as our extensive experience, validates the safety and efficacy of single-dose nevirapine as one of several options to prevent mother-to-child transmission of HIV."*

[4] *Africa, as the news media never tires of telling us, has become ground zero of the AIDS epidemic. The clinical definition of AIDS in Africa, however, is stunningly broad and generic, and was seemingly designed to be little other than a signal for funding. It is in no way comparable to Western definitions. The "Bangui definition" of AIDS was established in the city of Bangui in the Central African Republic, at a conference in 1985. The definition requires neither a positive HIV test nor a low T-cell count, as in the West, but only the presence of chronic diarrhea, fever, significant weight loss, and asthenia, as well as other minor symptoms. These happen to be the symptoms of chronic malnutrition, malaria, parasitic infections, and other common African illnesses. (In 1994 the definition was updated to suggest the use of HIV tests, but in practice they are prohibitively expensive.) Even when HIV tests are performed, many diseases that are endemic to Africa, such as malaria and TB, are known to cause false positives. The statistical picture of AIDS in Africa, consequently, is a communal projection based on very rough estimates of HIV positives, culled from select and small samples, which are extrapolated across the continent using computer models and highly questionable assumptions.*

which has the advantage of short-circuiting the question of whether the drugs actually work, and of utterly obviating the question of whether they are even remotely safe. This situation has had particularly tragic ramifications on the border between the class of Americans with good health insurance, who are essentially consumers of pharmaceutical goods, and those without insurance, some of whom get drugs "free" but with a significant caveat attached: They agree to be experimented on. These people, known in the industry as "recruits," are pulled in via doctors straight from clinics and even recruited on the Internet into the pharmaceutical industry and the government's web of clinical trials, thousands of which have popped up in recent years across the nation and around the world. Such studies help maintain the industry's carefully cultivated image of benign concern, of charity and progress, while at the same time feeding the experimental factories from which new blockbuster drugs emerge. "I call them what they are: human experiments," says Vera Hassner Sharav, of the Alliance for Human Research Protection in New York City. "What's happened over the last ten to fifteen years is that profits in medicine shifted from patient care to clinical trials, which is a *huge* industry now. Everybody involved, except the subject, makes money on it, like a food chain. At the center of it is the NIH, which quietly, while people weren't looking, wound up becoming the partner of industry."

By June 2004, the National Institutes of Health had registered 10,906 clinical trials in ninety countries. The size of these trials, which range from the hundreds to more than 10,000 people for a single study, creates a huge market for trial participants, who are motivated by different factors in different societies but generally by some combination of the promise of better health care, prenatal care, free "access" to drugs, and often—especially in the United States—cash payments. Participating doctors, whose patient-care profits have been dwindling in recent years because of insurance-company restrictions, beef up their incomes by recruiting patients.

Dr. Jonathan Fishbein is hardly a rabble-rouser. But he is a passionate advocate of "good clinical practice," or GCP, a set of international standards that were adopted in 1996, as clinical-trial research boomed. The GCP handbook states: "Compliance with this standard provides public assurance that the rights, safety, and well-being of trial subjects are protected, consistent with the principles that have their origin in the Declaration of Helsinki, and that the clinical trial data are credible." During the decade prior to his arrival at DAIDS, Fishbein had overseen and consulted on hundreds of clinical trials for just about every pharmaceutical company. Fishbein knew, before he took his job as director of the Office for Policy in Clinical Research Operations at DAIDS, that there was a troubled study haunting the whole division. Nobody was supposed to talk about it, but it hung heavily in the air. "Something about Uganda, that's all I knew," he says. There was a trial staged there, a big one, that had been plagued with "problems," and there was also a lot of talk about one particular employee connected to this trial who would need to be disciplined. Soon he discovered just how bad the situation was. "The HIVNET thing," he recalls, "it hit me like a fire hose when I walked in there."

FISHBEIN KNEW THAT A TROUBLED STUDY WAS HAUNTING DAIDS. "SOMETHING ABOUT UGANDA, THAT'S ALL I KNEW"

Fishbein's position was new. "It sounded like a very important position," he says. "I was to oversee the policies governing all the clinical-research operations, both here and abroad." He was told he would have "go–no go" authority over individual trials. It wasn't long before Fishbein realized that he was, in effect, taking a job that was the equivalent of piloting an already airborne plane. "They had all these trials going on, and hundreds of millions of dollars flowing in every year, but there was apparently no one in a senior position there who really had clinical expertise—who knew all the nuances, rules, and regulations in the day-to-day running of clinical trials." DAIDS, when Fishbein came to work there in 2003, was running about 400 experimental trials both in the United States and abroad.

A DAIDS project officer close to the HIVNET study closed the door when she had her first meeting with Fishbein. She had also crossed over from the private sector, and so she and Fishbein shared a disillusionment over how much shoddier and more chaotic the research culture was within the government, compared with industry. "I'm really frightened about the stuff that goes on here," she told him. "We really need somebody." This project officer, who for her own protection cannot be named, told Fishbein that the division's flagship study in Africa—HIVNET 012—had been wracked with problems and completely lacking in regulatory standards. She told Fishbein that the trial investigators were "out of control," and that there was no oversight of them, and nobody with either the inclination or the authority to make them adhere to safety standards. What Fishbein subsequently learned entangled him in a story with eerie echoes of John Le Carré's *Constant Gardener*.

For our purposes, the story of nevirapine begins in 1996, when the German pharmaceutical giant Boehringer Ingelheim applied for approval of the drug in Canada. The drug had been in development since the early 1990s, which was a boom time for new HIV drugs. Canada rejected nevirapine twice, once in 1996 and again in 1998, after the drug showed no effect on so-called surrogate markers (HIV viral load and CD4 counts) and was alarmingly toxic. In 1996, in the United States, the FDA nonetheless gave the drug conditional approval so that it could

be used in combination with other HIV drugs.[5]

By this time, Johns Hopkins AIDS researcher Brooks Jackson had already generated major funding from the NIH to stage a large trial for nevirapine in Kampala, Uganda, where the benevolent dictator Yoweri Museveni had opened his country to the lucrative promise of AIDS drug research, as well as other kinds of pharmaceutically funded medical research. HIVNET 012, according to its original 1997 protocol, was intended to be a four-arm, Phase III, randomized, placebo-controlled trial.[6] Its sole sponsor was listed as the National Institute of Allergy and Infectious Diseases (NIAID), though one of the investigators was a Boehringer employee. The "sample size" was to be 1,500 HIV-1 infected Ugandan women more than thirty-two weeks pregnant. The four arms they would be divided into were 1) A single dose of 200mg nevirapine at onset of labor and a single 2mg dose to the infant forty-eight to seventy-two hours post-delivery, and 2) a corresponding placebo group; 3) 600mg of AZT at onset of labor and 300mg until delivery, with a 4mg AZT dose for the infant lasting seven days after birth, and 4) a corresponding placebo group. There were to be 500 women in each "active agent" arm and 250 in each placebo arm. The study was to last eighteen months, and its "primary endpoints" were to see how these two regimens would affect rates of HIV transmission from mother to child, and to examine the "proportion of infants who are alive and free of HIV at 18 months of age." Another primary objective was to test the "safety/tolerance" of nevirapine and AZT. HIVNET's architects estimated that more than 4,200 HIV-positive pregnant women would deliver at Mulago hospital each year, allowing them to enroll eighty to eighty-five women per month. Consent forms were to be signed by either the mother or a guardian, by signature or "mark." One of the exclusion criteria was "participation during current pregnancy in any other therapeutic or vaccine perinatal trial."

Although HIVNET was designed to be a randomized, placebo-controlled, double-blind, Phase III trial of 1,500 mother/infant pairs, it wound up being a no-placebo, neither double- nor even single-blind Phase II trial of 626 mother/infant pairs. Virtually all of the parameters outlined for HIVNET 012 were eventually shifted, amended, or done away with altogether, beginning with perhaps the most important—the placebo controls. By a "Letter of Amendment" dated March 9, 1998, the placebo-control arms of HIVNET were eliminated. The study as reconstituted thus amounted to a simple comparison of AZT and nevirapine.

On September 4, 1999, *The Lancet* published HIVNET's preliminary results, reporting that "Nevirapine lowered the risk of HIV-I transmission during the first 14–16 weeks of life by nearly 50 percent." The report concluded that "the two regimens were well-tolerated and adverse events were similar in the two groups." The article also reported that thirty-eight babies had died, sixteen in the nevirapine group and twenty-two in the AZT group. The rate of HIV transmission in the AZT arm was 25 percent, while in the nevirapine group it was only 13 percent. As *Hopkins Medical News* later reported, the study was received rapturously. "The data proved stunning. It showed that nevirapine was 47 percent more effective than AZT and had reduced the number of infected infants from 25 to 13 percent. Best of all, nevirapine was inexpensive—just $4 for both doses. If implemented widely, the drug could prevent HIV transmission in more than 300,000 newborns a year."

With the results of the study now published in *The Lancet*, Boehringer, which previously had shown little interest in HIVNET, now pressed for FDA approval to have nevirapine licensed for use in preventing the transmission of HIV in pregnancy.

There were complications, however. On December 6, 2000, a research letter in *The Journal of the American Medical Association* warned against using nevirapine for post-exposure treatment after two cases of life-threatening liver toxicity were reported among health-care workers who'd taken the drug for only a few days. (One of them required a liver transplant.) The January 5, 2001, issue of the CDC's *Morbidity and Mortality Weekly Report* (*MMWR*) contained an FDA review of MedWatch—an informal reporting system of drug reactions—that highlighted an additional twenty cases of "serious adverse events" resulting from fairly brief nevirapine post-exposure prophylaxis. "Serious adverse events" were defined as anything "life-threatening,

[5] *Asked to comment about the Hafford case, HIVNET 012, and the larger nevirapine controversy, Boehringer Ingelheim provided the following statement: "Viramune ® (nevirapine) was an innovation in anti-HIV treatment as the first member of the non-nucleoside reverse transcriptase inhibitor (NNRTI) class of drugs. Now in its tenth year of use, Viramune has been used as a treatment in more than 800,000 patient-years worldwide."*

[6] *The study was originally titled "HIVNET 012: A Phase III Placebo-Controlled Trial to Determine the Efficacy of Oral AZT and the Efficacy of Oral Nevirapine for the Prevention of Vertical Transmission of HIV-1 Infection in Pregnant Ugandan Women and Their Neonates." "Randomization" means that people are randomly chosen for one arm of the study or another, a procedure that is supposed to even out the variables that could affect the outcome. "Placebo controls" are the bedrock of drug testing and are the only way to know whether the treatment is effective. Phase I trials involve a small group of people, twenty to eighty, and are focused on safety and side effects. In Phase II trials the drug is given to an expanded cohort, between 100 and 300, to further evaluate safety and begin to study effectiveness. Phase III drug trials expand further the number of people enrolled, often to more than 1,000, and are meant to confirm a drug's effectiveness, monitor side effects, and compare it with other treatments commonly used. A small Phase I trial preceded HIVNET 012 that studied the safety, primarily, of nevirapine in pregnant women but also looked at efficacy. It was called HIVNET 006, and it enrolled twenty-one pregnant women for initial study. Of twenty-two infants born, four died. There were twelve "serious adverse events" reported. The study also showed that there was no lowering of viral load in the mothers who took the study drug (the industry's agreed-upon standard for interrupting maternal transmission).*

permanently disabling," or requiring "prolonged hospitalization, or [. . .] intervention to prevent permanent impairment or damage." The *MMWR* stressed that there probably were more unreported cases, since the reporting by doctors to MedWatch is "voluntary" and "passive."

But NIAID was on another track altogether, either oblivious of or undeterred by the toxicity controversy. In 2001, Boehringer Ingelheim submitted its supplemental licensing request to the FDA. The request was submitted based entirely on the results of HIVNET, as published in *The Lancet*. Around the same time, the South African Medicines Control Counsel (MCC) conditionally approved nevirapine for experimental use in mother-to-child transmission treatment. To its credit, however, the FDA decided to go to Kampala, inspect the site, and review the data itself.

Since Boehringer had not originally intended to use this study for licensing purposes, it decided to perform its own inspection before the FDA arrived. Boehringer's team arrived in Kampala and did a sample audit. They were the first to discover what a shambles the study was. According to Boehringer's preinspection report, "serious non-compliance with FDA Regulations was found" in the specific requirements of reporting serious adverse events. Problems also were found in the management of the trial drug and in informed-consent procedures. DAIDS then hired a private contractor, a company named Westat, to go to Uganda and do another preinspection. This time the findings were even more alarming. One of the main problems was a "loss of critical records." One of two master logs that included follow-up data on adverse events, including deaths, was said to be missing as the result of a flood. The records failed to make clear which mothers had gotten which drug, when they'd gotten it, or even whether they were still alive at various follow-up points after the study. Drugs were given to the wrong babies, documents were altered, and there was infrequent follow-up, even though one third of the mothers were marked "abnormal" in their charts at discharge. The infants that did receive follow-up care were in many cases small and underweight for their age. "It was thought to be likely that some, perhaps many, of these infants had serious health problems." The Westat auditors looked at a sample of forty-three such infants, and all forty-three had "adverse events" at twelve months. Of these, only eleven were said to be HIV positive. The HIVNET team had essentially downgraded all serious adverse events several notches on a scale it had created to adapt to "local" standards. That downgrade meant, among other things, that even seemingly "life-threatening" events were logged as not serious. Deaths, unless they occurred within a certain time frame at the beginning of the study, were not reported or were listed as "serious adverse events" rather than deaths. In one case, "a still birth was reported as a Grade 3 adverse event for the mother."

As a defense, the HIVNET team often cited ignorance. They told the Westat monitors that they were unaware of safety-reporting regulations, that they'd had no training in Good Clinical Practice, and that they had "never attempted a Phase III trial." The principal investigators and sub-investigators "all acknowledged the findings [of the audit] as generally correct," the Westat report said. "Dr. Guay and Dr. Jackson noted that many ('thousands') of unreported AE's and SAE's occurred. . . . They acknowledged their use of their own interpretation of 'serious' and of severity." "All agreed" that the principal and subinvestigators "had generally not seen the trial patients," and "all agreed" that in evaluating adverse and serious adverse events "they had relied almost entirely on second or third hand summaries . . . without attempting to verify accuracy." Westat also discovered that half the HIV-positive infants were also enrolled in a vitamin A trial, which effectively invalidates any data associated with them.

In light of the Westat report, DAIDS and Boehringer asked the FDA for a postponement of its inspection visit. The FDA responded by demanding to see the report immediately. On March 14, 2002, the FDA called a meeting with DAIDS, Boehringer, and the trial investigators. "They reprimanded the whole gang," says Fishbein. Then they said to Boehringer: Withdraw your application for extended approval, if you want to avoid a public rejection." Boehringer complied with the FDA's demand, though statements put out by NIAID made it sound as if the company had withdrawn the application for FDA approval in a spirit of profound concern for protocol. In South Africa, a few months later, the news focused on

the angry chorus of AIDS experts and activists, speaking as one. The South African MCC was reconsidering its approval of nevirapine for pregnant women because of Boehringer's withdrawal and the growing HIVNET controversy. The Associated Press reported that "activists fear the government, notorious for its sluggish response to the AIDS crisis, is pressuring the council to reject nevirapine, and that it could misrepresent the current discussions as proof the drug is toxic. Studies show nevirapine given to HIV-pregnant women during labor and to their newborn babies can reduce HIV transmission by up to 50 percent." The problem with such statements, of course, is that the study in question was precisely the one that established the claim that nevirapine cut HIV transmission.

Two inspections had now declared HIVNET to be a complete mess: Boehringer's own and Westat's, which had been performed in conjunction with DAIDS. But the ways in which the various players were tethered together made it impossible for DAIDS to condemn the study without condemning itself.[7] But DAIDS was well aware of what had transpired.

According to DAIDS's public version of events, which was dutifully echoed in the AIDS press, the trouble with HIVNET was that it was unfairly assailed by pedantic saboteurs who could not grasp the necessary difference between U.S. safety standards and the more lenient standards that a country like Uganda deserved. Two weeks after the fifty-seven-page Westat report was delivered, the deputy director of NIAID, Dr. John LaMontagne, had set the tone by stating publicly: "There is no question about the validity [of the HIVNET results] . . . the problems are in the rather arcane requirements in record keeping." DAIDS was so dismissive of the Westat report that Westat's lawyers eventually put officials on notice that they were impugning Westat's reputation.

Meanwhile, as the investigations continued, nevirapine had long since been recommended by the World Health Organization and registered in at least fifty-three countries, and Boehringer had begun shipping boxes of the drug to maternity wards across the developing world. In 2002, President Bush announced a $500 million program to prevent maternal transmission of HIV in which nevirapine therapy would play a major role—despite the fact that the drug has never received FDA approval for this purpose.

Two inspections had now declared HIVNET to be a complete mess: Boehringer's own and Westat's

In 2003, when Jonathan Fishbein was drawn into the HIVNET saga, the cover-up (for that, ultimately, is what the NIH response had become) was ongoing. In response to the massive failures documented by Boehringer and Westat, DAIDS embarked on a "remonitoring review" in an attempt to validate the study's results. Ordinarily, an outside contractor would be retained for such a complex project, but Tramont made the decision to keep the remonitoring in-house. Drafting the review was a massive undertaking that took months of research, lengthy interviews with the investigators, and painstaking analysis of poorly organized documentation, as the DAIDS team attempted to learn what had actually taken place in Kampala. Even so, Tramont wanted the HIVNET site reopened in time for President Bush's visit to Uganda. In March 2003, Tramont and his staff gathered together the different sections and substantially rewrote the report, especially the safety section, minimizing the toxicities, deaths, and record-keeping problems. The rewritten report concluded that nevirapine was safe and effective for the treatment of mother-to-child transmission of HIV, thus saving HIVNET 012 from the scrapheap of failed scientific studies.

While preparing the safety review section, however, an NIH medical officer named Betsy Smith noticed a pattern of elevated liver counts among some of the babies in the AZT arm. Following FDA regulations, she drafted a safety report documenting this finding and gave it to Mary Anne Luzar, a DAIDS regulatory affairs branch chief. Luzar forwarded the safety report to the FDA. The HIVNET investigators were furious; Tramont, who had previously signed off on the safety report, ordered a new version to be drafted, essentially retracting the previous one, and sent it to the FDA.[8] The political stakes were very high: nevirapine was now a major element in the Administration's new $15 billion African AIDS program—on July 11, President Bush even toured the HIVNET site in Kampala, which DAIDS had reopened for the occasion over Fishbein's objections.

By late June 2003, Jonathan Kagan, the deputy director of DAIDS, asked Fishbein to sign off on a reprimand of Luzar for insubordination. Fishbein reviewed the HIVNET documentation and concluded that Luzar had done nothing wrong, that she had simply followed protocol. Fishbein's refusal to go along with Luzar's reprimand amounted to a refusal to participate in the HIVNET cover-up. In July, Tramont sent an email to all DAIDS staff instructing them not to speak about HIVNET at all. "HIVNET 012 has

[7] *Brooks Jackson declined to comment for this article. Laura Guay responded with the following statement: "Several in-depth reviews of the conduct and results of the HIVNET 012 trial as well as the data collected from subsequent trials and PMTCT programs, have substantiated the HIVNET 012 conclusions that Nevirapine is safe and effective in preventing mother-to-child HIV transmission. Nevirapine remains one of the most important tools for the prevention of mother-to-child HIV transmission in the developing world, where there are still hundreds of thousands of HIV-infected pregnant women who do not have access to any HIV testing, antiretroviral therapy, or HIV care at all. For many programs struggling to establish PMTCT programs with limited resources, Nevirapine is often the only option available." Family Health International, the NIH contractor originally responsible for monitoring HIVNET 012, contested the Westat report and said that the results of the study had been validated by the NIH and the Institute of Medicine.*

[8] *Smith and Luzar have been forbidden by the NIH to speak to the press about HIVNET. Luzar was deposed by Fishbein's attorney in his wrongful-termination lawsuit, Stephen Kohn, in December 2004, and this account is partially based on her deposition.*

been reviewed, re-monitored, debated and scrutinized. To do any more would be beyond reason. It is time to put it behind us and move on. Henceforth, all questions, issues and inquiries regarding HIVNET 012 is [*sic*] to be referred to the Director, DAIDS."[9]

What followed, as internal emails and memorandums clearly show, was a vicious and personal campaign on the part of Kagan and Tramont to terminate Fishbein's employment. DAIDS officials wrote emails in which they worried about how to fire him without creating repercussions for NIAID director Anthony Fauci, who had given Fishbein a commendation for his work. The communiqués took on conspiratorial tones as Tramont led the operation and mapped out its challenges. On February 23, 2004, Tramont emailed Kagan: "Jon, Let's start working on this—Tony [Fauci] will not want anything to come back on us, so we are going to have to have iron-clad documentation, no sense of harassment or unfairness and, like other personnel actions, this is going to take some work. In Clauswitzian style, we must overwhelm with 'force.' We will prepare our paper work, then . . . go from there." The web now included several more NIH/NIAID employees, who weighed in with suggestions about how best to expel Fishbein without leaving damning legal fingerprints on the proceedings.

Fishbein spent months trying to get a fair hearing, petitioning everyone from Elias Zerhouni, the director of the NIH, to Secretary of Health Tommy Thompson. It was around this time that Fishbein became a "ghost." Nobody addressed him in the corridors, in the elevators, in the cafeteria. "There was an active campaign to humiliate me," he says. "It was as if I had AIDS in the early days. I was like Tom Hanks in *Philadelphia*. Nobody would come near me."

In March 2004, Fishbein began seeking whistle-blower protection. He met with congressional staff and attracted enough attention on Capitol Hill to force the NIH to agree to a study by the National Academy's Institute of Medicine (IOM). The terms of that inquiry were skewed from the outset, however, and the nine-member panel decreed that it would not deal with any questions of misconduct. The panel ignored Fishbein's evidence that DAIDS had covered up the study's failures and relied on testimony from the HIVNET investigators and NIH officials. Not surprisingly, it found that HIVNET's conclusions were valid. Six of the nine members on the panel were NIH grant recipients, with yearly grants ranging from $120,000 to almost $2 million.[10]

THE QUESTION SHOULD NOT BE, IS NEVIRAPINE BETTER THAN AZT? BUT, IS NEVIRAPINE BETTER THAN NOTHING?

Fishbein dismissed the IOM report as a whitewash. Indeed, the report's conclusions are hard to credit, given the overwhelming evidence uncovered by the Westat investigation and documentation such as the following email, which was sent by Jonathan Kagan to Ed Tramont on June 19, 2003. Tramont was considering HIVNET researchers Jackson and Guay for an award:

> Ed—I've been meaning to respond on this—the bit about the award. I think that's a bit over the top. I think that before we start heaping praise on them we should wait to see if the lessons stick. We cannot lose sight of the fact that they screwed up big time. And you bailed their asses out. I'm all for forgiveness, etc. I'm not for punishing them. But it would be "over the top" to me, to be proclaiming them as heroes. Something to think about before pushing this award thing . . .

NIAID has issued a total ban against any employee speaking to the press about Fishbein's allegations. Instead, they have posted "Questions and Answers" about the matter on their website. The first question is: "Is single-dose nevirapine a safe and effective drug for the prevention of mother-to-infant transmission of HIV?" Fishbein has said that due to the spectacular failures of the HIVNET trial, the answer to this is not known, and not knowable. Fishbein believes that ultimately the HIVNET affair is not "about" nevirapine or even AIDS,

> but about the conduct of the federal government, which has been entrusted to do research on human beings and to uphold basic standards of clinical safety and accuracy.

NIAID answers its first question mechanically and predictably:

> "Single-dose nevirapine is a safe and effective drug for preventing mother-to-infant transmission of HIV. This has been proven by multiple studies, including the HIVNET 012 study conducted in Uganda."

The phrase "safe and effective" has been baked into both the question and the answer, rendering both blank and devoid of meaning. The "multiple studies" line is a familiar tactic, designed to deflect from the study that is actually being addressed, and that is HIVNET 012.

A short letter published in the March 10, 2005, issue of *Nature* quietly unpegged the core claim of NIAID and its satellite organizations in the AIDS industry regarding nevirapine's "effectiveness." Written by Dr. Valendar Turner, a surgeon at the Department of Health in Perth, Australia, the letter read:

> Sir—While raising concerns about "standards of record keeping" in the HIVNET 012 trial in Uganda, in your News story, "Activists and Researchers rally behind AIDS drug for mothers," you overlook a greater flaw. None of the available evidence for nevirapine comes from a trial in which it was tested against a placebo. Yet, as the study's senior author has said, a placebo is the only way a scientist can assess a drug's effectiveness with scientific certainty.

[9] *At this point the story grows ever more complicated, as Fishbein supported Luzar in a sexual-harassment claim against Kagan.*

[10] *An internal NIH investigation, which was obtained by the Associated Press last summer, vindicated many of Fishbein's charges and concluded that "it is clear that DAIDS is a troubled organization," and that the Fishbein case "is clearly a sketch of a deeper issue." Kagan and Tramont did not return repeated calls for comment. Instead, an NIH spokesman, Dr. Cliff Lane, said that the agency stands by HIVNET 012.*

The HIVNET 012 trial abandoned its placebo group in early 1998 after only 19 of the 645 mothers randomized had been treated, under pressure of complaints that the use of a placebo was unethical.

The HIV transmission rate reported for nevirapine in the HIVNET 012 study was 13.1%. However, without antiviral treatments, mother-to-child transmission rates vary from 12% to 48%. The HIVNET 012 outcome is higher than the 12% transmission rate reported in a prospective study of 561 African women given no antiretroviral treatment.

The letter concluded by asking: "On what basis can it be claimed that 'there's nothing that has in any way invalidated the conclusion that single-dose nevirapine is effective for reducing mother-to-child transmission'? Without supporting evidence from a placebo-controlled randomized trial, such statements seem unwarranted." HIVNET claimed to reduce HIV transmission by "nearly 50 percent" by comparing a nevirapine arm to an AZT arm. Turner's letter points out that 561 African women taking no antivirals transmitted HIV at a rate of 12 percent. Had nevirapine been asked to compete with that placebo group, it would have lost. As it was, there was no placebo group, so HIVNET's results are a statistical trick, a shadow play, in which success is measured against another drug and not against a placebo group—the gold standard of clinical trials. The question should not be, Is nevirapine better than AZT? but, *Is nevirapine better than nothing?*

Independent evidence suggests that it is not.

A 1994 study, for example, that gave vitamin A to pregnant HIV-positive mothers in Malawi reported that those with the highest levels of Vitamin A transmitted HIV at a rate of only 7.2 percent. This is consistent with a vast body of research linking nutritional status to sero-conversion, as well as to general health. Another study on the efficacy of nevirapine in mother-to-child transmission was performed by researchers from Ghent University (Belgium) in Kenya and published in 2004.

Dr. Ann Quaghebeur, who led the Ghent study, was reached at her home near London. I asked her what she thought of the reaction to HIVNET 012. She replied in a very quiet voice, almost a whisper. "Our results showed that nevirapine had little effect. I actually felt it was a waste of resources. HIVNET was just one study, but usually before you apply it in a field setting there should be a few more studies to see if it works in real life. What I think they should have done is wait for more studies before they launched this in all those countries." When I asked her how she explained this, she replied, "Well, I want to be careful, there seems to be an industry now."

The failure of the HIVNET researchers to properly control their study with a placebo group is not as unusual as one might think. In fact, this failure is perhaps the outstanding characteristic of AIDS research in general. The 1986 Phase II trial that preceded the FDA's unprecedented rapid approval of AZT was presented as a double-blind, placebo-controlled study, though it was anything but that. As became clear afterward through the efforts of a few journalists, as well as the testimony of participants, the trial was "unblinded" almost immediately because of the severe toxicity of the drug. Members of the control group began to acquire AZT independently or from other study participants, and eventually the study was aborted and everyone was put on the drug. As in the case of HIVNET, documents obtained by journalist John Lauritsen under the Freedom of Information Act subsequently suggested that data-tampering was widespread. Documents were altered, causes of death were unverified, and the researchers tended to assume what they wished to prove, i.e., that placebo-group diseases were AIDS-related but that those in the AZT group were not. So serious were the deviations from experimental protocol at one Boston hospital that an FDA inspector attempted to exclude data from that center. In the end, however, all the data were included in the results, and

the FDA approved the drug in 1987.[11]

AZT was approved in record time, but that record didn't stand for long. In 1991, the FDA approved another DNA chain terminator, ddI, without even the pretense of a controlled study. Anti-HIV drugs such as Crixivan were approved in as little as six weeks, and cast as a triumph of AIDS activism. This pattern of jettisoning standard experimental controls has continued up to the present, as the HIVNET affair amply demonstrates, and has characterized not only research into new drugs designed to exterminate HIV but the more fundamental questions at the root of AIDS research.

The HIVNET cover-up can only be understood within the larger political context of AIDS. The emergence of this syndrome in the 1980s sparked a medical state of emergency in which scientific controls, the rules that are supposed to bracket the emotions and desires of individual researchers, were frequently compromised or removed entirely. AIDS helped turn disease into politics, and politics, at least in the United States, is all about turning power into money.

No one has been more persistent in calling attention to the failings of AIDS research than Peter Duesberg, a virologist and cancer specialist at the University of California at Berkeley. If Duesberg's name sounds familiar, it's because he has been quite effectively branded in the international media as the virologist who is wrong about HIV. His name entered the popular culture in the late 1980s pre-stamped with wrongness. You knew he was wrong before you knew what he had said in the first place.

In 1987, Duesberg published a paper in the journal *Cancer Research* entitled "Retroviruses as Carcinogens and Pathogens: Expectations and Reality." He was, at the time, at the top of the field of retrovirology, having mapped the genetic structure of retroviruses and defined the first cancer gene in the 1970s. He was the youngest member, at age fifty, ever elected into the National Academy of Sciences. In this paper, which in the words of his scientific biographer, Harvey Bialy, "sealed his scientific fate for a dozen years," Duesberg argued that retroviruses don't cause cancer and concluded by detailing how and why the retrovirus HIV cannot cause AIDS.

As AIDS grew in the 1980s into a global, multibillion-dollar juggernaut of diagnostics, drugs, and activist organizations, whose sole target in the fight against AIDS was HIV, condemning Duesberg became part of the moral crusade. Prior to that 1987 paper, Duesberg was one of a handful of the most highly funded and prized scientists in the country. Subsequently, his NIH funding was terminated and he has received not one single federal research dollar since his pre-1987 Outstanding Investigator Grant ran out. Duesberg lost his lab facilities and had to move twice within a few years to smaller labs on the Berkeley campus, where he spent much of his time writing futile research grant proposals asking to test his hypothesis that AIDS is a chemical syndrome, caused by accumulated toxins from heavy drug use. He lost his graduate students, who were warned that to emerge from his lab would blight their careers. He was denied and had to fight for routine pay increases by his employers at UC Berkeley, where he has tenure and still teaches. He was "dis-invited" from scientific conferences, and colleagues even declared that they would refuse to attend any conference that included him. Duesberg also was banished from publishing in scientific journals that previously had welcomed his contributions, most theatrically by the editor of *Nature*, Sir John Maddox, who wrote a bizarre editorial declaring that Duesberg would be de-

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[11] *AZT, which was developed as a chemotherapeutic agent in 1964 but shelved because of its extreme toxicity, is a DNA chain terminator, which means that it brings DNA synthesis to a halt. It is therefore an extremely efficient cell killer. HIV is a retrovirus, and as such replicates itself by inserting its genes into a cell's genome so that when the cell divides a new copy of the virus is produced. AZT prevents the replication of HIV by killing infected T-cells; unfortunately, it kills all dividing cells indiscriminately, whether they are infected with a retrovirus or not, and will very quickly decimate even a healthy person's immune system. AZT's manufacturer, GlaxoSmith Kline, chose not to comment for this article.*

nied the standard scientific "right of reply" in response to personal attacks that were frequently published in that journal. Prior to 1987, Peter Duesberg never had a single grant proposal rejected by the NIH. Since 1991 he has written a total of twenty-five research proposals, every single one of which has been rejected. "They took him out, just took him right out," says Richard Strohman, an emeritus professor of biology at UC Berkeley.

And what was it, exactly, that Peter Duesberg had done? He simply pointed out that no one had yet proven that HIV is capable of causing a single disease, much less the twenty-five diseases that are now part of the clinical definition of AIDS.[12] He pointed to a number of paradoxes regarding HIV and argued that far from being evidence that HIV is "mysterious" or "enigmatic," these paradoxes were evidence that HIV is a passenger virus.

The classical tests of whether or not a microorganism is the cause of infectious disease are known as Koch's postulates. They state: 1) the microorganism must be found in all cases of the disease; 2) it must be isolated from the host and grown in pure culture; 3) it must reproduce the original disease when introduced into a susceptible host; and 4) it must be found present in the experimental host so infected. Although claims to the contrary have been made, Duesberg maintains that it has never been demonstrated that HIV satisfies all of Koch's postulates. His exhaustive analysis of the peer-reviewed scientific literature has revealed more than 4,000 documented AIDS cases in which there is no trace of HIV or HIV antibodies. This number is significant, because there are strong institutional forces deterring such descriptions and because the vast majority of AIDS cases are never described in formal scientific papers. In fact, most AIDS patients have no active HIV in their systems, because the virus has been neutralized by antibodies. (With all other viral diseases, by the way, the presence of antibodies signals *immunity* from the disease. Why this is not the case with HIV has never been demonstrated.) Generally speaking, HIV can be isolated only by "reactivating" latent copies of the virus, and then only with extraordinary difficulty. Viral load, one of the clinical markers for HIV, is not a measurement of actual, live virus in the body but the amplified fragments of DNA left over from an infection that has been suppressed by antibodies. Another embarrassment for the HIV hypothesis is the extraordinary latency period between infection and the onset of disease, despite the fact that HIV is biochemically most active within weeks of initial infection. This la-

[12] HIV was declared the probable cause of AIDS in a U.S. government press conference in 1984. It was claimed that the virus had been discovered by NIH researcher Robert Gallo. In fact, Gallo had not discovered HTLV-III (Human T-cell Lymphotropic Virus III, as it was known before it was rechristened with the more memorable name HIV). That honor belongs primarily to Luc Montagnier, of the Pasteur Institute, who had sent Gallo a sample of the virus.

tency period, which apparently grows with every passing year, enables proponents of the theory to evade Koch's third and fourth postulates.

The foregoing is merely a sketch of the central mystery presented by the HIV theory of AIDS. There are many more, which Duesberg has laid out very carefully in his scientific papers and in a trade book published ten years ago, but they all boil down to the central point that when it comes to AIDS, basic scientific standards seem no longer to apply.[13] AIDS is a "syndrome" defined by twenty-five diseases, all of which exist independently of HIV. No one has ever demonstrated the cell-killing mechanism by which HIV is supposed to cause all these different diseases, and no one has ever demonstrated how a sexually transmitted virus can manage to restrict itself overwhelmingly to gay men and other AIDS risk groups instead of spreading randomly through the population, as do all other infectious diseases. The "overwhelming" character of the evidence for HIV's causation has always been epidemiological; which is to say, a correlation, a coincidence. Whenever we have AIDS, researchers say, we also have HIV. But this correlation is a result of the official definition of AIDS, which states that a disease counts as AIDS only if it corresponds with HIV antibodies. ("AIDS without HIV" has been given a singularly unmemorable name: idiopathic CD4 lymphocytopenia.)

Given that the evidence for HIV is coincidental, a number of research avenues suggest themselves, yet orthodox AIDS researchers have failed to demonstrate, using large-scale controlled studies, that the incidence of AIDS-defining diseases is higher among individuals infected with HIV than among the general uninfected population. Consequently, it could very well be the case that HIV is a harmless passenger virus that infects a small percentage of the population and is spread primarily from mother to child, though at a relatively low rate. (This hypothesis would tend to explain the fact that the estimated number of HIV-positive Americans has remained constant at about 1 million since 1985.) Nor have large-scale controlled studies been carried out to directly test the AIDS-drug hypothesis, which holds that many cases of AIDS are the consequence of heavy drug use, both recreational (poppers, cocaine, methamphetamines, etc.) and medical (AZT, etc.).[14] Nor have controlled studies been carried out to prove that hemophiliacs infected with HIV die sooner than those who are not infected. Such studies might be expensive and tedious, but expense has never been a serious objection to AIDS researchers, who have spent many billions of dollars in the last twenty years on HIV research and practically nothing on alternative causes or even co-factors. (Even Luc Montagnier, the discoverer of HIV, has stated repeatedly that the virus cannot cause AIDS without contributing causes.)

Attempts to rigorously test the ruling medical hypothesis of the age are met not with reasoned debate but with the rhetoric of moral blackmail: *Peter Duesberg has the blood of African AIDS babies on his hands. Duesberg is evil, a scientific psychopath. He should be imprisoned.* Those who wish to engage the AIDS research establishment in the sort of causality debate that is carried on in most other branches of scientific endeavor are tarred as AIDS "denialists," as if skepticism about the pathogenicity of a retrovirus were the moral equivalent of denying that the Nazis slaughtered 6 million Jews. Moral zeal rather than scientific skepticism defines the field. It has been decided in advance that HIV causes AIDS; consequently all research and all funding must proceed from that assumption. Similarly, it was known in advance that AZT was a "magic bullet" against HIV; the word was out that it was a "life-saving drug" before anyone could possibly verify this, and so scientific controls were compromised. Journalists (myself included) who reported at the time that the drug apparently was killing patients were labeled "AZT refuseniks" and even "murderers."

The nevirapine debate follows the same histrionic, antiscientific pattern. Because of his concerns about the toxicity of this and other antiretroviral drugs, President Thabo Mbeki of South Africa was pilloried in the international press as pharmaceutical companies and their well-funded "activist" ambassadors repeated their mantra about "life-saving drugs." So, too, was Jonathan Fishbein, who never questioned the premise that HIV causes AIDS, tarred and feathered for pointing out that the NIH flagship study on nevirapine was a complete disaster. Fishbein's failure to fall into line, his failure to understand in advance of experimental proof that nevirapine was too important to fail, meant that the AIDS bureaucracy's neutralizing antibodies had to be activated to destroy them.

In the end, the NIH failed to si-

[13] It has been claimed that HIV somehow causes cell death even when it is not present by remote programmed "suicidal" mechanisms. Some researchers claim that HIV exploits special receptors on human T-cells that, due to a hypothetical genetic mutation, many "Caucasian Europeans" lack, but most Africans have. What's interesting is that many gay men also seem to possess these mysterious receptors, as do intravenous drug users and transfusion recipients.

It is claimed that although HIV does not kill the laboratory T-cells used to manufacture AIDS tests, it does kill T-cells in the human body, even though it infects only a very small proportion of them, typically an average of 0.1 percent. HIV does not sicken or kill chimpanzees, though they do produce antibodies. It was recently claimed that HIV appears to be evolving into a form less dangerous to human beings. Such unproven hypotheses about the ingenuity of HIV proliferate in the popular and scientific media like the seasonal flu. Seldom do journalists insist on good hard evidence for these assertions.

[14] There is ample statistical and epidemiological evidence linking the rise of mass drug abuse in the late Sixties and Seventies with the sudden appearance of AIDS. The overwhelming majority of AIDS patients with Karposi's sarcoma, for example, have been heavy users of nitrate inhalers, or "poppers." The case of "super AIDS" that was recently reported in New York turned out upon closer examination to be an individual with an extraordinarily heavy methamphetamine habit.

lence Fishbein. In late December 2005, he won his case and was retroactively reinstated at the agency, though he won't be returning to DAIDS. He is unable to discuss the terms of his settlement, but he has promised to continue his commitment to research integrity and the protection of human research subjects. Peter Duesberg has been less successful, though there are signs of rehabilitation.

Regardless of whether Duesberg is right about HIV, his case, like Fishbein's, lays bare the political machinery of American science, and reveals its reflexive hostility to ideas that challenge the dominant paradigm. Such hostility is not unusual in the history of science,[15] but the contemporary situation is dramatically different from those faced by maverick scientists in the past. Today's scientists are almost wholly dependent upon the goodwill of government researchers and powerful peer-review boards, who control a financial network binding together the National Institutes of Health, academia, and the biotech and pharmaceutical industries. Many scientists live in fear of losing their funding. "Nobody is safe," one NIH-funded researcher told me. "The scientific-medical complex is a $2 trillion industry," says former drug developer Dr. David Rasnick, who now works on nutrition-based AIDS programs in Pretoria, South Africa. "You can buy a tremendous amount of consensus for that kind of money."

"You have to write a grant a year almost. And you have to write four to get one, if you're any good. I got out just in time. Everybody who's still in there says the same thing," says Berkeley's Strohman. "Before the biotech boom, we never had this incessant urging to produce something useful, meaning profitable. Everybody is caught up in it. Grants, millions of dollars flowing into laboratories, careers and stars being made. The only way to be a successful scientist today is to follow consensus. If you're going to produce something and put it on the market you don't want any goddamn surprises. You've got the next quarter to report and you don't want any bad news. It's all about the short term now. Science has totally capitulated to corporate interests. Given their power and money, it's going to be very hard to work our way out of this."

Duesberg has never been afraid to challenge consensus, but contrary to what many in the AIDS establishment would have us believe, he is very far from being a scientific psychopath.[16] In 1997, on the brink

[15] Few today remember the controversies over scurvy and pellagra, which, until the discovery of vitamin C and niacin, were blamed by the medical establishment on mysterious infectious agents. Those who pointed out, even before they knew the cause, that dietary changes cured both conditions were dismissed as flat-earthers.

[16] Nor is Duesberg alone in dissenting from AIDS orthodoxy. More than 2,300 people, mostly scientists and doctors, including Nobelists in chemistry and medicine, have signed the petition of the Group for the Scientific Reappraisal of the HIV-AIDS Hypothesis, which calls for a more independent and skeptical approach to the question of AIDS causality.

of scientific demise in the U.S., Duesberg was quietly invited back to his native Germany to resume his cancer research. During this time, commuting biannually between Mannheim and Berkeley, Duesberg formulated and tested a theory that shifts the focus of cancer causation from the "mutant gene" theory that has reigned for about three decades to a simpler explanation that revives an abandoned thread of research from early in the twentieth century, which posited that cancer is caused by chromosomal malfunction, now known as "aneuploidy."

Harvey Bialy, the founding scientific editor of *Nature Biotechnology*, a sister journal to *Nature*, recently spent four years writing a scientific biography of Duesberg entitled *Oncogenes, Aneuploidy, and AIDS*. The book is a history of the papers, review articles, and letters that Duesberg published between 1983 and 2003, and the responses they generated. I asked him why he wrote the book. "I am persuaded that aneuploidy is the initiating event in carcinogenesis," Bialy said. "Peter has found the genetic basis for cancer. The most immediate application of it will be early diagnosis."

"When aneuploidy, or genetic instability, or whatever linguistic term you want to use, gets reincarnated as the dominant theoretical explanation for the genesis of cancer, Peter Duesberg will be recognized as a major contributor to that," Bialy said. "I wanted to make sure that his contributions were not swept aside or ignored." I asked him about the AIDS controversy. "AIDS is a political thing, and Peter's stuck in it. There's nothing to discuss anymore on that." Bialy made a critical point: Science is amoral and should be. There is no right and wrong, only correct and incorrect. "Duesberg," Bialy said, "is a *classical* molecular biologist. All he is interested in is rigorously testing dueling hypotheses. The twin pillars, AIDS and oncogenes, both are crumbling because of the questions Peter Duesberg put into motion."

"The basis of speciation is changing the content and the number of chromosomes," says Duesberg. "Cancer is essentially a failed speciation. It's not mutation. Cancer is a *species*. A really bad breast, lung, or prostate cancer has seventy, eighty, or more chromosomes. Those are the real bad guys—they're way outside our species. But it's a rare kind of species that as a parasite is more successful in its host than the normal host cell is."

There has been considerable international interest in Duesberg's new research.[17] In January 2004, he hosted a conference on aneuploidy and invited fifty cancer researchers from around the world who also have been working on the connections between aneuploidy and cancer. Seventy showed up, including such luminaries as Thomas Ried, the National Cancer Institute's head of cancer genomics, Gert Auer from the Karolinska Institute in Stockholm, and Walter Giaretti, who heads the equivalent of the NCI in Italy. And on May 31 of last year, amid considerable tension, Duesberg was invited by the National Cancer Institute to give a talk at the NIH. The auditorium crackled with nervous tension as people filed in and took their seats. His talk was succinct and laced with his characteristic irony, but the questions afterward were civilized, with no tangible hostility. All was not forgiven, however. After the talk, while Duesberg remained at the podium talking to a group of people from the audience, I noticed a very angry-looking NIH publicist standing at the back of the room admonishing a colleague, a scientist, who'd posed a question that somehow connected aneuploidy to HIV. "*You opened it up,*" she scolded. "We got through it okay, but you *opened it up*." As the questioner tried to defend himself, a thickset man who'd been standing in the circle said loudly, as though intending to broadcast it across the room: "Well, at least if he's wrong about this he won't be *killing millions of people*."

Nobel laureate Kary Mullis, who discovered the revolutionary DNA technique called the polymerase chain reaction, has long been a supporter of Duesberg, but he has grown weary of the AIDS wars and the political attacks on contrarian scientists. "Look, there's no sociological mystery here," he told me. "It's just people's income and position being threatened by the things Peter Duesberg is saying. That's why they're so nasty. In the AIDS field, there is a widespread neurosis among scientists, but the frenzy with which people approach the HIV debate has slacked off, because there's just so much slowly accumulating evidence against them. It's really hard for them to deal with it. They made a really big mistake and they're not ever going to fix it. They're still poisoning people."

Duesberg thinks that up to 75 percent of AIDS cases in the West can be attributed to drug toxicity. If toxic AIDS therapies were discontinued, he says, thousands of lives could be saved virtually overnight. And when it comes to Africa, he agrees with those who argue that AIDS in Africa is best understood as an umbrella term for a number of old diseases, formerly known by other names, that currently do not command high rates of international aid. The money spent on antiretroviral drugs would be better spent on sanitation and improving access to safe drinking water (the absence of which kills 1.4 million children a year).

It's too late to save people like Joyce Ann Hafford, but it is possible that an open and honest debate about the risks of current AIDS treatments and the scientific questions concerning HIV could save others. n

[17] Even so, the National Cancer Institute still refuses to fund him. Duesberg has submitted five grant proposals to study aneuploidy, and all have been rejected. One of the most influential cancer researchers in the country, Bert Vogelstein, Clayton Professor of Oncology and Pathology at Johns Hopkins University, has written a letter urging the NCI to reconsider. "I agree with him that aneuploidy is an essential part of cancer," Vogelstein wrote. "Dr. Duesberg continues to have a major impact on this burgeoning area of research, through his careful experimental observations as well as through his thoughtful reviews and critiques of the subject. There is no question that he is a world leader in this field of investigation."

Errors in Celia Farber's March 2006 article in Harper's Magazine

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Authors (in alphabetical order by surname):

Robert Gallo MD[1], Nathan Geffen[2], Gregg Gonsalves[3], Richard Jefferys[4], Daniel R. Kuritzkes, MD[5], Bruce Mirken[6], John P. Moore PhD[7], Jeffrey T. Safrit PhD[8]

1. Director Institute of Human Virology, University of Maryland Baltimore
2. Policy, Communications and Research Co-ordinator, Treatment Action Campaign, South Africa
3. Gay Men's Health Crisis
4. Basic Science, Prevention & Vaccines Project, Treatment Action Group
5. Director of AIDS Research, Brigham and Women's Hospital, Associate Professor of Medicine, Harvard Medical School
6. Director of Communications, Marijuana Policy Project (previously a health journalist who covered HIV/AIDS for AIDS Treatment News, Men's Healthand other publications)
7. Professor of Microbiology and Immunology, Weill Medical College of Cornell University, New York
8. Senior Programs Officer, Elizabeth Glaser Pediatric AIDS Foundation; Visiting Assistant Professor, Department of Pediatrics David Geffen School of Medicine, University of California, Los Angeles

Please send correspondence to Nathan Geffen at **nathan@tac.org.za**

This document describes the errors in Celia Farber's March 2006 article in Harper's Magazine, titled *Out of Control: AIDS and the Corruption of Medical Science.*

Our primary concern is with rebutting Farber's misconceptions about HIV/AIDS and antiretrovirals (ARVs). We have not focused our attention on misleading or biased reporting that relate to the NIH; none of us is an NIH employee. We have also ignored the sections on Peter Duesberg's career problems, his rejected funding proposals, and how he is (or is not) regarded by other cancer researchers nowadays; we have no interest in Duesberg, other than to note that he is not an AIDS researcher and has no practical experience in studying HIV.

Using a plethora of false, misleading, biased and unfair statements, Farber attempts to cast scientific institutions and scientists as dishonest. But intellectual dishonesty is the norm for Farber and other AIDS denialists including David Rasnick, Peter Duesberg, Kary Mullis and Harvey Bialy – all people she mentions favourably in her article. David Rasnick works for a vitamin entrepeneur, Matthias Rath. They have conducted unauthorised experiments on people with HIV in South Africa, convincing their subjects to take Rath's vitamin products in dangerously high doses, instead of scientifically recognised treatments for AIDS. It has been alleged that some of their subjects have died due to this experiment.[1] Farber implies financial motives permeate scientific research. Why does Farber not make similar allegations against the AIDS denialists, many of whom are involved in the marketing of unproven alternative medicines?

HIV has been shown to be the cause of AIDS in numerous studies. ARVs have been shown to reduce death and

illness in people with HIV. They have also been shown to reduce mother-to-child transmission (MTCT) of HIV. They often cause side-effects. On rare occasions these can be fatal, but death from HIV/AIDS is a far greater risk. The evidence shows beyond doubt that the benefits of ARVs far outweigh their risks.

We present two tables below. The first is a list of errors in Celia Farber's article in the March 2006 issue of Harper's. The list is possibly incomplete. All of these errors should have been found in the fact-checking process. The second table contains some relevant points about the authorities Farber cites in support of her views.

Guide to the First Table

Page and Column Number

The first column contains the page and column number of the error in Farber's article. If only one number is given, it is the page.

Error Type Key

MISLEADING: Farber implies a false fact without stating it directly. There are 16 such errors.
FALSE: Farber states a false fact. There are 25 such errors.
FAIRNESS: This denotes statements by Farber which are unfair, e.g. implying sinister motives with the flimsiest of evidence. There are ten such errors.
BIAS: Farber neglects key facts which negate her theories. There are five such errors.

There are 56 errors noted in the table.

Topic Key

TESTING: Related to HIV testing ARVs: Related to antiretrovirals
MTCT: Related to mother-to-child transmission prevention
TRIALS: Related to clinical trials
HIV: Related to HIV as the cause of AIDS

Description

The comments of Farber that are referred to are described in italics. Then a description is given why Farber is wrong.

TABLE 1.

Ref (page, col)	Error Type	Topic	Description
37; 1	**MISLEADING**	**TESTING**	***Farber states that pregnancy itself can cause a false positive result. She supplies no supporting reference.***
			A properly conducted HIV-test protocol (which involves at least two HIV tests) has a very small chance of giving a false positive, irrespective of pregnancy status. Farber alleges that Hafford's HIV-test was carried out incorrectly. If this was the case, medical negligence is a different matter to whether HIV tests carried out according to protocol are accurate in pregnant women. HIV tests were highly accurate from the time they were developed in 1984[2] and have become much more accurate over time as the underlying technology has evolved. HIV tests are amongst the most accurate available in medical science. For more on testing see Mirken (2001).[3] For a more technical discussion see Coon (2000).[4] Incidentally, the testing protocol of the PACTG 1022 trial, to which Farber refers, required multiple HIV tests and regular viral load counts. Farber states that Hafford was only tested once. Assuming Farber is right, then Hafford's doctor did not follow the protocol. We, however, are not privy to Hafford's medical records and therefore cannot know if Farber's allegation of Hafford having only one test is correct. Was Harper's privy to this? Consequently, was the allegation fact-checked?
37; 3	**MISLEADING**	**ARVs**	***Farber states that PACTG 1022 probed the "outer limits of bearable toxicity."***
			PACTG 1022 compared ARVs, that had already been found to be safe and effective for treatment in the absence of pregnancy, in pregnant women. All drugs used in the trial had been shown in previous trials to benefit people with HIV. This is why the FDA has registered them. The PACTG 1022 trial happened to find higher than expected toxicity of nevirapine in very specific circumstances. Even here, toxicity was sufficiently rare as to be outweighed by the likely benefits of nevirapine use. The FDA revised its nevirapine recommendations on the basis of this trial. Nevirapine remains an important antiretroviral medicine whose benefits outweigh its risks. Nevirapine (or a drug, efavirenz, used instead of it) has been shown in an analysis of clinical trials to slow disease progression, particularly in patients with low CD4 counts.[5] Safety trials are obviously associated with a calculated risk, but they are permitted when the expected benefits are considered to outweigh this risk. Would Farber suggest that no clinical trials be conducted whatsoever?

Ref (page, col)	Error Type	Topic	Description
38; 3	**MISLEADING**	**ARVs**	***Farber describes the death of one patient and implies this is relevant to the science of HIV.***

To try to get readers to conclude that an ARV related death can be generalised to conclude that the risks of ARVs outweigh their benefits is misleading and unscientific. HIV is a life-threatening condition. The drugs used to treat it are imperfect but have been shown beyond reasonable doubt in numerous clinical trials and analyses of large numbers of patients in real-world settings (operational cohorts) to reduce the risk of illness and death. They are associated with side-effects.

The same scenario applies to chemotherapy for cancer; patients take drugs that cause nausea, vomiting, hair-loss etc, because to do so is preferable to dying from cancer.

Clinical trials, or meta-analyses of clinical trials, have demonstrated direct clinical benefits, i.e. fewer AIDS-related illnesses or deaths, for a number of ARVs, including AZT,[6] lamivudine,[7] didanosine,[8] stavudine,[9] nevirapine,[10] efavirenz,[11] and others.

As ARVs began prolonging the lives and reducing the illnesses of people with HIV, it became the standard of care. In recent clinical trials the control group has to be given this standard of care for ethical reasons. Consequently progression to AIDS or death is unusual in recent clinical trials. Therefore scientists use what are called surrogate markers, CD4 and viral load counts, to determine drug efficacy. These surrogate markers are highly correlated with disease progression.[12]

A meta-analysis of ARV trials has demonstrated that they have a profound effect on reducing progression to AIDS or death.[13]

Furthermore, in practice, ARVs have been shown to reduce illness and deaths in industrialised and developing countries around the world irrespective of race, gender, sexual orientation, age and recreational drug use. We have included a sample of these in the endnotes.[14,15,16,17,18,19,20,21,22,23]

Ref (page, col)	Error Type	Topic	Description
39; 1	**FALSE**	**TESTING**	***Farber states that all "babies born to HIV-positive mother are born positive but most become negative within 18 months."***
			Farber is clearly confused by the passing on of the mother's antibodies to the child, a natural mechanism that protects the child from infectious disease as its own immune system develops. These passively transferred antibodies are eliminated from the child's system within 18 months at most, usually rather sooner. If a child is infected with HIV, it produces its own antibodies, which persist. After 18 months, if the child still tests HIV-antibody positive, it is almost definitely its own antibodies that are producing the result. Furthermore, a PCR test for the presence of the virus itself can accurately determine a child's HIV status by about six weeks after birth.
39; 1	**FALSE**	**TESTING**	***In footnote one, Farber makes various false statements about HIV tests. She comments that HIV tests are not even required for an AIDS diagnosis in Africa. She also claims most HIV tests come back indeterminate or negative when redone. She supplies no references.***
			Most people in the industrialised world, as well as many developing countries, have at least two different HIV antibody tests to confirm they are HIV-positive, as part of the HIV testing protocol. HIV tests are highly accurate. It is false that when most people are retested they test indeterminate or negative. Even the risk of a single HIV ELISA test giving a false positive is less than 1% with today's tests. HIV tests are required for an AIDS diagnosis in South Africa. They are also standard in Botswana, Kenya, Uganda and many other clinics throughout Africa. An AIDS diagnosis cannot be considered definitive without an HIV test. Farber's comment about hopping on a plane from Uganda to Australia to change HIV diagnosis is simply silly hyperbole.
39; 3	**MISLEADING**	**MTCT**	***Farber switches from a discussion of PACTG 1022 to HIVNET 012 and omits to explain a critical distinction.***
			Here Farber misleads in a way that is repeated throughout the remainder of the article. She confuses the short-course nevirapine-only regimen used to reduce MTCT with chronic treatment using nevirapine as one component of a combination of ARVs. Not a single life-threatening event related to short-course nevirapine has been recorded in mother or child in tens of thousands of such uses around the world. The nevirapine toxicity found in PACTG 1022 was in chronic treatment.

Ref (page, col)	Error Type	Topic	Description
40; 1	**FAIRNESS**	**ARVs**	***Farber reports on the publication of PACTG 1022 as if it is something sinister.***
			In fact, its publication was standard scientific procedure. Furthermore, that nevirapine toxicity was reported in the paper is an indication of honestly conducted science that is inconsistent with Farber's implications of some sort of cover-up. There is no logical comparison with the Schiavo case; Farber's analogy is bizarre and hard to understand.
40; 2	**MISLEADING**	**MTCT**	***Farber repeats her mistake on p. 39 col. 3.***
			Farber fails to inform her readers that she is switching back and forth between a discussion of chronic nevirapine use for treatment with short-course nevirapine for MTCT reduction.
40; 2	**FAIRNESS**	**MTCT**	***Farber points out that Elizabeth Glaser Pediatric AIDS Foundation (EGPAF) has taken money from Boehringer Ingelheim and implies this disqualifies them from commenting on the safety of short-course nevirapine.***
			It is the function of the EGPAF, a registered charity, to prevent MTCT. The fact that the EGPAF has taken money from Boehringer Ingelheim does not disqualify it from commenting on the safety of nevirapine. The EGPAF is not selling nevirapine on behalf of Boehringer, but distributing it free of charge to those without access to it. Farber only mentions the EGPAF with respect to affirming the safety and efficacy nevirapine and links this to their Boehringer grant. But many organisations affirmed the safety and efficacy of single-dose nevirapine, including ones without financial connections to the pharmaceutical industry such as the World Health Organisation, the Nobel peace prize-winning organisation, Medecins Sans Frontieres, and the Treatment Action Campaign.
40; 3	**FALSE**	**HIV**	***Footnote 4 states that AIDS is defined differently in Africa.***
			It is true that as more was learned about AIDS, the definition of the disease changed. There is nothing unusual in this; AIDS was only discovered in 1981. It is a testimony to scientific methodology that it only took a few years to discover its cause. An accurate diagnosis of AIDS, throughout the world, does require an HIV-positive test. While there are facilities in Africa which do not even have HIV tests (one of the cheapest components of the medical response to HIV), our knowledge of HIV in Africa is based on studies that have used HIV tests. (Incidentally, facilities that cannot offer HIV testing do not offer ARVs either.)

We show later that numerous studies conducted in Africa have demonstrated that people with HIV have much higher morbidity and mortality than people without HIV. Also see Nicoll and Killewo (2000).[24]

Ref (page, col)	Error Type	Topic	Description
40; 3	**MISLEADING**	**HIV**	***Footnote 4 also states that AIDS happens to have the same symptoms as "chronic malnutrition, malaria, parasitic infections and other common African illnesses."***

HIV, not poverty, predicts progression to AIDS in Africa. Of course, living in poverty increases the risk of acquiring HIV infection, because poor people have less access to information about how HIV is spread and how to avoid contracting this infection. Also, poor people, especially poor women, frequently have less power to negotiate the use of condoms. HIV-infected people living in resource-poor environments can progress more rapidly to AIDS and death because of their reduced access to health care and their diminished state of general health compared to individuals who reside in more affluent settings.

As NIAID (2003) explains, the *"diseases that have come to be associated with AIDS in Africa - such as wasting syndrome, diarrheal diseases and TB - have long been severe burdens there. However, high rates of mortality from these diseases, formerly confined to the elderly and malnourished, are now common among HIV-infected young and middle-aged people, including well-educated members of the middle class."* [25]

Sewankambo, N*et al*. **AIDS. 2000 Oct 20;14(15):2391-400** is a study of nearly 20,000 people, both HIV-positive and HIV-negative in a Ugandan district. People with HIV were much more likely to get sick or die. Furthermore death rates in civil servants and the better-educated (i.e. not the poor) were higher than the general population. This was associated with HIV infection.[26]

Statistics South Africa (2005) counted South African death certificates between 1997 and 2002 and found a 57% increase in mortality (only a small portion can be accounted for by improved death registration and population growth). Critically, most of this increase is accounted for in young adults, with the highest proportion of adult deaths in 2002 being 30-39 year olds. Child mortality has also risen dramatically. This is incompatible with poverty as the cause of AIDS, especially in a country where living standards improved to some degree (or at worst stayed the same) during the period studied.[27]

Furthemore, some AIDS-related diseases, e.g. cryptococcal meningitis, are very rare in people without HIV, but very common in Africa in people with HIV.

			We provide further detail in the endnotes.[28]
40; 3	**MISLEADING**	**TESTING**	***Footnote 4 further states that HIV tests are prohibitively expensive in Africa.***
			HIV tests are widely available across Africa. They are not prohibitively expensive for large numbers of people.

Ref (page, col)	Error Type	Topic	Description
40; 3	**MISLEADING**	**TESTING**	***Footnote 4 further states "many diseases that are endemic to Africa, such as malaria and TB, are known to give false positives." Farber fails to supply a reference.***
			The risk of a false positive HIV test in Africa, as elsewhere, is very small if the correct protocol is followed. Some HIV antibody tests have been tested in Africa and found to be very accurate. These are the ones generally used. For example, the *Abbott Determine* rapid test used widely in South Africa has a specificity of at least 98% (and in some studies has achieved close to 100%). When this test is combined with a second rapid test or an ELISA test to determine HIV status, the risk of a false positive is negligible. The contribution of TB and malaria to false positives on today's tests is also negligible. For examples of trials of HIV tests used in Africa and Brazil, see Sauer *et al.* (2000),[29] Phili *et al.* (2002),[30] Ferreira *et al.* (2005),[31] Koblavi-Dème *et al.* (2001),[32] and Foglia *et al.* (2004)[33].
40; 3	**FALS**E	**TESTING**	***Footnote 4 states "The statistical picture of AIDS in Africa, consequently, is a communal projection based on very rough estimates of HIV positives culled from select and small samples, which are extrapolated across the continent using computer models and highly questionable assumptions."***
			(1) Statistical estimates are not extrapolated across the continent, but on a per country basis. (2) Large samples of people with HIV have been taken in a number of countries including Kenya, Botswana, Uganda and South Africa. (3) South Africa's HIV/AIDS surveillance is arguably better than most industrialised countries, let alone developing countries. It comes from annual antenatal surveys, two countrywide household surveys, numerous small community surveys and death certificates. The most widely used computer model used to determine the size of South Africa's epidemic closely matches the prevalence calculated in the latest countrywide household survey. See ASSA (2005)[34] and *Shisana et al.* (2005).[35]

			(4) It is true that estimates of AIDS in most African countries are imprecise, but there is evidence showing beyond reasonable doubt that the African HIV epidemic is massive. For a detailed rebuttal of the claim that HIV is not a serious epidemic in Africa see Geffen (2004).[36]
Ref (page, col)	**Error Type**	**Topic**	**Description**
41; 1	**BIAS**	**TRIALS**	***Farber complains about the growth of clinical trials and claims that everyone profits except the subjects. She also implies that only the poor and disadvantaged are used as subjects.***
			No reference is supplied to support the view that subjects on the whole are not benefiting from clinical trials. Many well-off people participate in clinical trials. The claim that most subjects of clinical trials are put at greater risk than benefit is astonishing, and it certainly contradicts common sense. Not every clinical trial is conducted perfectly, particularly from the perspective of record-keeping. Some are poorly conducted, but the vast majority conform to strict, internationally accepted ethical guidelines and benefit the study subjects.
41; 2	**FAIRNESS**	**TRIALS**	***Farber uses innuendo and rumours, from the perspective of Jonathan Fishbein, to cast aspersions on HIVNET 012, particularly on the honesty of its investigators. No concrete evidence is supplied. Surely this is not acceptable journalism in a magazine of Harpers' quality.***
			In actual fact, problems with HIVNET 012 were identified and made public by NIAID long before Fishbein made an issue of them[37]. The NIAID took steps to address these problems. The problems turned out to have no bearing on the scientific findings of HIVNET 012. Bookkeeping errors should not be automatically equated with a lack of ethics or any problems of a more serious and significant nature. To maintain clinical records in some developing countries (especially very poor ones such as Uganda) is not as simple as doing so in a leading industrialised country clinical research centre for a variety of reasons including financial ones and the shortage of fully trained clinical staff. This does not mean that clinical trials should not be conducted in developing countries or that trials in such countries are necessarily flawed, but there does need to be some understanding of the circumstances that can apply.
41; 3	**MISLEADING**	**ARVs**	***Farber states that Canada rejected nevirapine twice on the grounds that it did not show efficacy with respect to surrogate markers. She says that the FDA nevertheless registered it.***
			Nevirapine has been shown to be effective using surrogate markers of CD4 and viral load count. (See the FDA package insert for details.) Also see the meta-analysis of nevirapine and efavirenz referred to above.[38]

Ref (page, col)	Error Type	Topic	Description
42; 1	FALSE	MTCT	***Footnote 6 states there was no lowering of maternal viral load in the HIVNET 006 safety study.***
			The study states that *"The antiviral activity of nevirapine appeared to be quite strong, resulting in a relatively consistent median 1.3 log reduction in maternal plasma HIV RNA at 1 week after a single 200mg dose in all mothers."* [39]
42; 1	BIAS	MTCT	***Footnote 6 states in relation to HIVNET 006 "Of twenty-two infants born, four died. There were twelve serious adverse events' reported." Farber's implication is that the adverse events and deaths were due to nevirapine.***
			The investigators of this Ugandan study studied drug toxicity in detail. They report *"There were no serious adverse events or grade 3 or 4 clinical or laboratory toxicities thought by investigators to be related to nevirapine among the mothers of either cohort. There were five serious adverse events including two deaths in the infants in cohort 1. Only one of the five serious adverse events was thought by the investigators to be possibly, but not likely, study drug related. This infant developed respiratory distress at birth and seizures after a difficult and prolonged labor requiring the use of forceps. In cohort 2 there were seven serious adverse events, including two infant deaths, although none were related to the study drug."* [40]
42; 2	BIAS	MTCT	***Farber describes how the HIVNET 012 protocol was changed implying this rendered its quality sub-optimal.***
			There is nothing unusual or inappropriate about changing a study protocol if logistics or new scientific developments require it. If the study protocol became unacceptable, it would be rejected for publication. The results of HIVNET 012 were published in *The Lancet*, a leading medical journal.[41]
42; 2	FALSE	MTCT	***Farber claims that HIVNET 012 was supposed to be a phase III trial but wound up being a phase II trial.***
			Farber appears not to know the difference between a phase II and phase III trial, because HIVNET 012 was a randomized phase III trial. It was not double-blind, because the drug administration procedures were so different in each of the two arms. While phase III trials are ideally double-blind, this is not an indispensable requirement. Frequently drugs are tested using an "open-label" procedure.

Ref (page, col)	Error Type	Topic	Description
42; 2	**BIAS**	**MTCT**	***Farber claims HIVNET 012 was not placebo-controlled.***
			This statement is true, but Farber fails to explain critical facts that would allow readers to understand that the trial design was appropriate and that the results are meaningful. A short-course AZT regimen had been found in the PACTG 076 trial to be effective at reducing MTCT. The AZT regimen used in HIVNET 012 was a subset of the PACTG 076 regimen and therefore at least as good as placebo (but probably not as good as the regimen used in PACTG 076). In the nevirapine arm the rate of MTCT was reduced by 47% over that in the AZT arm by the end of the of the study. It would have been unethical to compare nevirapine directly to placebo when it was known that AZT could reduce MTCT. Therefore simple logic shows this: (1) HIVNET 012 AZT regimen is better than or equal to placebo. (2) HIVNET 012 nevirapine regimen is better than HIVNET 012 AZT regimen. Therefore HIVNET 012 nevirapine regimen is better than placebo. For more details, see the Cochrane review of antiretroviral regimens for reducing MTCT.[42] Note that four AZT regimens have been shown to be effective at reducing MTCT.
42; 3	**MISLEADING**	**MTCT**	***Farber quotes Hopkins Medical News stating that nevirapine is more effective than AZT at reducing MTCT.***
			This is not the full story. Short-course nevirapine is better than at least one short course AZT regimen that has been tested (i.e. the one tested in HIVNET 012). There are AZT regimens that are more effective than short-course nevirapine. It should be noted that single-dose nevirapine is a sub-optimal regimen for reducing MTCT, in respect of its efficacy. Its advantage lies in its relative affordability and the simplicity of its use, compared to more complex and expensive regimens. It is considered no more than a starting point for resource-poor health facilities or as one measure that can be used for HIV-positive women whose status is determined too late for other antiretroviral regimens (poor or well-off setting).[43]
43	**BIAS**	**MTCT**	***This page contains a highly biased account of the analysis of HIVNET 012. So as not to labour each of***
			Farber's misrepresentations and omissions, the following should be noted: In all the innuendo and accusations made by Farber and other AIDS denialists, as well as by Fishbein, no evidence has been put forward about the conduct of HIVNET 012 that calls into question its scientific findings.

HIVNET 012 was imperfect. The NIH has been honest about this. They state:

"NIAID and NIH initiated several reviews and re-reviews of HIVNET 012. These reviews identified procedural flaws in the study that led NIAID to implement improvements in the conduct of clinical research it supports both in the United States and abroad. We understand that certain previously recognized criticisms of the conduct of HIVNET 012 have re-emerged, **but stress strongly that throughout multiple reviews, the overall conclusions regarding the safety and efficacy of single-dose nevirapine in this setting have remained intact."** (our emphasis) They further state:

"The statement in the Associated Press article of December 13, 2004, that there may have been thousands of underreported serious adverse events in the HIVNET 012 study implies that those were due to the drug nevirapine. This implication is absolutely false. Remonitoring reports of HIVNET 012 found no additional serious adverse reactions related to nevira pine. The original published study and the multiple subsequent reviews of the HIVNET 012 trial that have carefully scrutinized its data have found only a very small number of serious adverse reactions that potentially might be due to nevirapine." **44**

See also NIAID (2004).***45***

The Institute of Medicine is part of the National Academy of Sciences. One of the purposes of the academy is to act as an independent reviewer of scientific issues. One could view it as the arbiter of scientific disputes of this nature, analogous to the way in which the US Supreme Court rules on matters of jurisprudence. In contrast to Farber or any of the AIDS denialists as well as the Associated Press journalist Farber refers to, the IOM extensively examined the documentation of HIVNET 012, including patient records. It concluded:

"Based on its review, the committee finds no reason to retract the publications or alter the conclusions of the HIVNET 012 study. The committee concludes that data and findings reported in *Guay et al*. (1999) and *Jackson et al*. (2003) are sound, presented in a balanced manner, and can be relied upon for scientific and policy-making purposes." **46**

Short-course nevirapine has been tested in the South African Intrapartum Nevirapine Trial, a much bigger trial than HIVNET 012. Not a single life-threatening event due to nevirapine was found. The trial used double the dose of HIVNET 012 on mothers. It confirmed short-course nevirapine's efficacy too.**47**

Short-course nevirapine has been added to an AZT regimen in a Thai trial and found to further reduce MTCT. The authors of this study state "No serious adverse effects were associated with nevirapine therapy." **48**

Short-course nevirapine has been used extensively in operational settings, e.g. Ayouba *et al.* (2003).[49] From a safety perspective, not a single life-threatening event has been recorded due to short-course nevirapine. From an efficacy perspective, results have been mixed; some cohorts have done well, others less well than expected. There is no cohort however that has reported worse results than would be expected with placebo including the Ghent study referred to by Farber. In the absence of any intervention, The rate of MTCT varies but is seldom less than 25% after a few months in a breast-feeding population,[50] or even predominantly non breast-feeding populations.[51]

In many cases in the developing world the benefit of ARVs for reducing MTCT at the time of delivery is undone by the later transmission of HIV through breast-milk. Resolving this additional mode of transmission is a complex scientific, operational and social undertaking. However, in wealthy countries, paediatric epidemics have been virtually eliminated through a combination of long-course ARV treatments, caesarian sections and formula-feeding. There are also success stories in the developing world, including the Cameroon study cited above, an MSF site in Cape Town, South Africa, a hospital in Johannesburg, South Africa (which found a 9% transmission rate in an operational setting, much lower than would be achieved with placebo) and the Ugandan site where HIVNET 012 was conducted.

Ref (page, col)	Error Type	Topic	Description
44; 3	**FALSE**	**VIT A**	***Farber states that the fact that some of the HIVNET 012 participants were on a vitamin A trial negates data associated with them.***
			If vitamin A supplements were actually effective at reducing MTCT, Farber's statement would be true. However several studies of whether vitamin A supplements reduces MTCT have been conducted. They all found that vitamin A supplementation does not differ from placebo. See the Cochrane review (2006)[52] on this. It is possible that vitamin A supplementation confers other benefits, but even this is unclear as a recent Zimbabwean study demonstrates.[53]
46; 2	**FAIRNESS**	**MTCT**	***Farber says that the terms of the IOM study were skewed from the start because the IOM would not look at issues of misconduct.***

			Investigating misconduct is not the role of the IOM and it was not asked to do so in this case either. The IOM was asked to examine scientific issues. It concluded that the science underlying the HIVNET 012 was sound. It also found that the trial largely conformed to internationally accepted ethical standards. Issues of misconduct are investigated by the NIH's Office of Research Integrity and/or by the Department of Health and Human Services' Office of the Inspector General, if they are justified. We are not aware that any such investigations have been initiated.
46; 2	**FAIRNESS**	**MTCT**	***Farber points out that six of the nine IOM members were NIH grantees. The innuendo is therefore they covered up for the NIH.***
			This is an astounding implied accusation that the magazine should not have permitted without evidence. In effect, Farber impugns the reputation of the six IOM members without offering any evidence that their findings were incorrect or that the implied bias was in any way real. It would be hard to find nine distinguished US scientists in the field of HIV research who do not receive NIH grants, given the role of the NIH as a funding agency. Furthermore, does Farber wish to suggest that the three non-NIH funded IOM members colluded in this suggested cover-up?
46; 3	**FALSE**	**MTCT**	***Farber states that the " 'multiple studies' line is a familiar tactic designed to deflect from the study that is actually being addressed, and that is HIVNET 012."***
			On the contrary, the fact that short-course nevirapine has been demonstrated to be effective in other clinical studies as well as in operational settings is relevant.
46; 3	**MISLEADING**	**MTCT**	***Farber quotes Valendar Turner's letter which makes the same misrepresentation about nevirapine not being tested against placebo discussed above.***
			As explained above nevirapine clearly performed better than placebo, despite Turner's allegations. Of note is that Turner is a prominent AIDS denialist in his own right, so is scarcely an objective reviewer of the trial data.
47; 1	**FAIRNESS**	**MTCT**	***Farber quotes Turner referring to a study of 561 people.***
			We are not sure what the 561 person study is that Turner refers to. No reference is supplied by Farber. We have given references above demonstrating that transmission is generally in the 25% region after a few months.
47; 1	**MISLEADING**	**VIT A**	***Farber's reference to women with higher levels of vitamin A having lower HIV transmission rates implies that HIV MTCT transmission can be resolved with vitamin A supplementation.***

			See above for evidence that vitamin A supplementation is not effective at reducing MTCT. Farber fails to consider that the general ill-health caused by advanced HIV disease is likely to reduce vitamin levels in the body.
47; 2	**FALSE**	**ARVs**	***Farber describes the AZT study that resulted in FDA approval as a phase II study but that was actually presented as a double-blind, placebo controlled study.***
			The AZT study referred to was a double-blind placebo controlled phase III study, known as BW 002.[54] Incidentally, it was for the treatment of HIV, not for the prevention of MTCT. Farber does not make this clear. Other studies demonstrated AZT's efficacy at reducing MTCT transmission.

Ref (page, col)	Error Type	Topic	Description
47; 3	**MISLEADING**	**ARVs**	***Farber states that the "AZT study was unblinded almost immediately because of the severe toxicity of the drug. Members of the control group began to acquire AZT independently or from other study participants."***
			Farber cannot have it both ways. If the BW 002 study became unblinded because of AZT's toxicity, then control group members would surely not have wished to acquire AZT. If the study became unblinded because AZT tasted differently to placebo, then perhaps control group members might have tried to acquire it. But here again, Farber makes a series of old AIDS denialist allegations that the results of BW 002 are invalid because of irregularities in the trial. In effect, Farber asks readers to take the side of a journalist who does not believe that HIV causes AIDS, John Lauritsen, against the considerably more expert opinion of the FDA panel that approved AZT. A number of points need to be made about this: (1) All the trial participants were symptomatic of AIDS or what was called AIDS Related Complex at the time. One out of 145 AZT recipients died on the trial. Nineteen out of 137 placebo recipients died. Furthermore the AZT recipients had fewer opportunistic infections and scored higher on quality of life measurements. This cannot be explained by chance and demonstrated the efficacy of AZT. Hence the FDA registered it.

(2) If as is alleged by the AIDS denialists some subjects became unblinded with the consequence that placebo subjects took AZT, then the results of the trial actually underestimate the efficacy of AZT and the AIDS denialist case is hoisted by its own petard. This is because if AZT was more dangerous than placebo, then there should have been more than just one death on the AZT arm. If the allegation of unblinding is true, then the only logical conclusion is that the number of placebo deaths was fewer than should have been the case, because some of the placebo subjects were given extra life-expectancy by taking AZT. There is simply no logical way for AIDS denialists to explain the massive difference in life-expectancy between the two arms.

(3) BW 002 was not the only placebo-controlled study that demonstrated AZT's efficacy. A placebo controlled trial known as ACTG 016 showed that symptomatic patients with CD4 counts between 200 and 500 were less likely to progress to AIDS. No difference in disease progression was seen in patients with CD4 counts greater than 500.[55]

(4) Fifteen AZT versus placebo studies have been conducted. Not one shows any evidence to support AIDS denialist arguments that AZT causes AIDS or that its risks outweigh its benefits.[56]

(5) Several uncontrolled studies have shown that AZT increases life-expectancy in symptomatic HIV patients.[57]

(6) The BW 002 trial that Farber refers to in the main text involved AZT use as monotherapy. As is now well understood. HIV mutates rapidly resulting in selection for strains of the virus that are resistant to a single drug. Indeed, if AZT was not effective, HIV would not need to mutate to escape it. The short-term benefits demonstrated in the first, placebo-controlled AZT study led to the demand that subsequent trials of potential antiretroviral drugs in patients who had progressed to AIDS did not use a placebo control, but rather employed AZT. Consequently, subsequent studies demonstrated improved survival in individuals receiving dual drug therapy compared to AZT.

(7) Farber makes no mention of the fact that numerous ARV trials have demonstrated that they reduce morbidity and mortality. A meta-analysis of ARV clinical trials found the following:

One ARV reduces progression to AIDS or death by 30% against placebo.

Two ARVs reduce progression to AIDS or death by 40% against one ARV.

Three ARVs reduce progression to AIDS or death by 40% against two ARVs. (Jordan et al. **BMJ. 2002 Mar 30;324(7340):757**. [**Full-Text**])

If the risks of ARVs outweigh their benefits, why does using more of them result in less mortality and morbidity?

(8) A recent ARV study by the NIH, the largest ever conducted, found that the continuous use of ARVs resulted in half the rate of disease progression and death than occurred when treatment was interrupted.[58]

If the risks of ARVs outweigh their benefits, why does taking them continuously result in less mortality and morbidity than taking them occasionally?

(9) As cited above (see note regarding page 38 column 3), numerous cohort analyses from around the world, both in developing and wealthy countries, demonstrate that ARVs are prolonging and improving life substantially. More examples of the efficacy of ARVs from different cohorts are being published regularly.

There is much more but the above should be sufficient to demonstrate that Farber's arguments are without merit. None of this should imply that ARVs are not associated with side-effects, which in rare circumstances are fatal. But the evidence is beyond doubt that their benefits outweigh their risks.

Ref (page, col)	Error Type	Topic	Description
47; 3	**FAIRNESS**	**ARVs**	***Farber states that the BW 002 trial was aborted. Her tone implies this was sinister. She fails to explain the legitimate reasons for terminating the trial.***
			The trial was terminated because an interim analysis revealed that AZT was much better than placebo. Continuing to keep patients on placebo would have therefore been unethical. This is standard practice in clinical trials. Anything else would endanger the lives of patients.
48; 2	**FALSE**	**ARVs**	***Farber states that the FDA approved ddI without even the pretense of a clinical trial in 1991. She creates the impression that ddI is an untested medicine.***
			First, ddI was, of course, tested in a clinical trial prior to its approval by the FDA. The results of the trial were published after the drug was approved.[59]This is neither unusual nor sinister, and the results of the trial were available to the FDA during the approval process. She also fails to point out that ddI has been tested in a number of clinical trials. A Cochrane meta-analysis found that ddI added to AZT regimens reduced death and morbidity.[60]

Ref (page, col)	Error Type	Topic	Description
48; 2	FAIRNESS	ARVs	***Farber states in relation to clinical trials "This pattern of jettisoning standard experimental controls has continued up to the present ... ."***
			On the contrary, clinical trials are more closely scrutinised than before and, as a result, their scientific and ethical qualities are ever-improving. More needs to be done to improve the separation of clinical trials from those with a financial interest in their outcome, but this does not mean that clinical trials are a cesspit of corrupted science.
48; 2	FALSE	ARVs	***Footnote 11 states that AZT is a DNA chain terminator and kills all dividing cells indiscriminately. Farber further states "AZT prevents the replication of HIV by killing infected T-cells." Apparently GlaxoSmithKline was asked to comment on this. If Harper's had an appropriate fact-checking process for scientific issues, it would have been realised that this should have been fact-checked with expert researchers, not the manufacturer of the drug.***
			AZT does not kill cells indiscriminately. At concentrations below those that are toxic to human cells, AZT interferes directly with HIV replication within the living, infected cell, by inhibiting the conversion of the viral RNA into DNA.[62] A more detailed description of how AZT works is given in an endnote.[63] AIDS researchers and clinicians do not claim that AZT is a perfect drug; undoubtedly it can and does cause side effects. As with most drugs used to treat, say, cancer, the therapeutic index for AZT is less than ideal, but the dangers of not treating HIV infection strongly outweigh the risks of doing so. AZT therefore remains a highly useful drug for HIV therapy. This has been shown in clinical trials and cohort analyses as demonstrated by several references in our endnotes.
49; 1	FALSE	HIV	***Farber appears to agree with Duesberg's view that HIV is incapable of causing a single disease.***
			HIV causes a progressive decline of the immune system by depleting CD4+ T-cells. Eventually the immune system becomes dysfunctional and incapable of fighting off diseases that it normally would. People with advanced HIV-disease are more susceptible than the general HIV-negative population to about 30 different diseases, many of them with high mortality rates.[64]
49; 2	FALSE	HIV	***Farber cites Duesberg that HIV has not fulfilled Koch's postulates. No argument or references are provided to back this up.***
			HIV as the cause of AIDS meets all four of Koch's postulates.[65]

			(Postulate one) Studies have found HIV in almost every case where a person has been diagnosed with AIDS. Obviously there will be occasional misdiagnoses, as with any disease. (See our explanation of Farber's next error as well.) (Postulate two) HIV can be isolated from AIDS patients and grown in laboratories. PCR tests can count the amount of HIV in blood. The virus is easily, and has been on numerous occasions, photographed using electron microscopes. (Postulate three) Most people with HIV experience immune system decline, eventually leading to AIDS.[66] Postulate three does not require every, or even most, hosts to reproduce the disease. But in the case of HIV, the vast majority of people progress to AIDS. Furthermore, there are well-documented cases of workers developing AIDS after being being infected with HIV in their laboratories. Likewise a case of a US dentist who infected six of his patients with HIV has been documented. Three died of AIDS. One developed AIDS. Five of the patients had no other proposed risk factors for AIDS. In both these examples, tests were done which confirmed the origins of their infections. These two examples not only meet postulate three but all four postulates. (Postulate four) PCR tests show the presence of HIV in infected people. That HIV is the cause of AIDS has arguably been demonstrated more thoroughly than is the norm for any disease with a viral causation.
49; 2	**FALSE**	**HIV**	***Farber cites Duesberg that there are 4,000 AIDS cases in which HIV was absent.***
			This is false. The actual situation in the US is described accurately as follows *"A survey of 230,179 AIDS patients in the United States revealed only 299 HIV-seronegative individuals. An evaluation of 172 of these 299 patients found 131 actually to be seropositive; an additional 34 died before their serostatus could be confirmed (Smith et al. **N Engl J Med. 1993 Feb 11;328(6):373-9**).*[67] Of note is that, in extremely rare cases, HIV-infected people die of AIDS so quickly that they do not develop antibodies to the virus, but nevertheless their virus can be isolated.[68] If Duesberg has made this astonishing finding, he should be able to publish it in a credible peer-reviewed scientific journal. Of course this has not been done. As explained previously and in more detail later, numerous studies from around the world, including Africa, the epicentre of the epidemic, demonstrate that mortality and morbidity is much higher in people with HIV.

Ref (page, col)	Error Type	Topic	Description
49; 3	**FALSE**	**HIV**	***Farber writes: "In fact, most AIDS patients have no active HIV in their systems, because the virus has been neutralized by antibodies."***
			PCR tests demonstrate that HIV is active in people with HIV antibodies. Most of the HIV in the body is located within solid lymphoid tissues, where it is transmitted by cell-to-cell spread. Antibodies are unable to interfere efficiently with this process. Furthermore, whenever effective neutralizing antibodies are generated within the body, HIV responds by mutating to generate resistant variants that are unaffected by these antibodies.[69]
49; 3	**FALSE**	**HIV**	***Farber states "HIV can be isolated only by 'reactivating' latent copies of the virus, and then only with extraordinary difficulty." She supplies no reference.***
			This is false. Virus isolates are routinely made in clinical and basic research laboratories. It is true that more virus is produced by reactivating latent cells, but this is not what Farber is saying.
49; 3	**FALSE**	**HIV**	***Farber states "With all other viral diseases, by the way, the presence of antibodies signals immunity from the disease. Why this is not the case with HIV has never been demonstrated."***
			The presence of antibodies all too often does not signify immunity from disease (e.g. herpes zoster, herpes simplex, hepatitis C, hepatitis B, dengue - all of these viruses can cause disease in the presence of virus-specific antibodies). HIV is a retrovirus and as such it integrates upon infection. Antibodies specific to a retrovirus almost always means the patient is infected and the levels of antibody usually correlate to some extent with the level of virus replication. We present more detail in an endnote.[70]
49; 3	**FALSE**	**HIV**	***Farber writes "Viral load, one of the clinical markers for HIV, is not a measurement of actual, live virus in the body, but the amplified fragments of DNA left over from an infection that has been suppressed by antibodies."***
			This is nonsense. First, viral load assays do not measure DNA, they measure HIV's content of RNA genomes (HIV is an RNA-containing, not a DNA-containing virus). Second, there is ample evidence that the signals from plasma viral load assays are proportional to the infectious virus content of plasma. In numerous studies monitoring cohorts of HIV patients, viral load increases with time.[71] How is this possible if all that is left over are the "fragments ... from an infection that has been suppressed"?

Ref (page, col)	Error Type	Topic	Description
50; 1	**FALSE**	**HIV**	***Farber claims that the latency period of HIV allows evasion of Koch's third and fourth postulates. She gives no reference.***
			HIV as the cause of AIDS does meets all four of Koch's postulates as has been shown above.
50; 1	**FALSE**	**HIV**	***Farber states that all infectious diseases spread randomly through the population, but HIV does not.***
			HIV is primarily sexually transmitted and sexually transmitted infections do not spread randomly through the population. Sexually transmitted infections consistently target people who have more partners, use condoms less frequently, and visit sex workers.
50; 1	**MISLEADING**	**HIV**	***Footnote 13 contains multiple scientific errors and perpetuates several misconceptions about HIV and AIDS that are commonly listed on AIDS denialist web sites.***
			Farber writes "It has been claimed that HIV somehow causes cell death even when it is not present by remote programmed 'suicidal' mechanisms." It is difficult to discern what Farber is trying to say here, because as written, the sentence makes no scientific sense whatsoever. Perhaps the most plausible interpretation of Farber's train of thought is that she is alluding to the death of CD4+ T-cells by a mechanism known as apoptosis (sometimes called "programmed cell death") during HIV infection. The underlying science here is complex, and specialist reviews on viral pathogenesis should be consulted for a fuller picture.[72,73,74] We provide a detailed explanation as an endnote.[75]
50; 1	**FALSE**	**HIV**	***Farber states in footnote 13 "Some researchers claim that HIV exploits special receptors on human T-cells that, due to a hypothetical genetic mutation, many 'Caucasian Europeans' lack, but many Africans have. What's interesting is that many gay men also seem to possess these mysterious receptors, as do intravenous drug users and transfusion recipients."***
			Again, these sentences betray Farber's ignorance of a substantial body of scientific information. As we explain in a detailed endnote[76], a fraction of Caucasians have genetic predispositions which render them less likely to contract HIV (not immune) or more likely to progress slowly. However, the vast majority of Caucasians have no known genetic predisposition that makes them less likely to contract HIV or progress to AIDS. Many Caucasians do contract HIV and do progress to AIDS.

Ref (page, col)	Error Type	Topic	Description
50; 1	**FALSE**	**HIV**	***Farber further states in footnote 13 "It is claimed that although HIV does not kill the laboratory T-cells used to manufacture AIDS tests, it does kill T-cells in the human body, even though it infects only a very small proportion of them, typically an average of 0.1 percent."***
			There are three inaccuracies in this sentence. First, HIV does kill T-cells in the laboratory, as was recorded in the very earliest papers on the isolation of HIV dating from 1983-1984. Second, "laboratory T-cells" have not been used to "manufacture AIDS tests" for many years now (the technology has evolved well beyond the early methods of the mid-1980's which were based on the production of inactivated HIV particles in permanent T-cell lines that had been carefully selected for relative resistance to the cell-killing effects of HIV). Third, HIV does directly kill, or otherwise cause the death of a substantial fraction of the total CD4+ T-cell complement of the body. Farber is presumably alluding to measurements of the HIV infection status of CD4+ T-cells present in the bloodstream, which constitute only a small proportion of the total amount of these cells present in the body as a whole. Most CD4+ T-cells cells are, in fact, located in solid lymphoid tissues, particularly in the gut-associated lymphoid tissue. The loss of CD4+ T-cells from such tissues upon HIV infection is rapid in rate and substantial in extent.[77]
50; 1	**FALSE**	**HIV**	***Farber further states in footnote 13 that "HIV does not sicken or kill chimpanzees."***
			It is true that HIV replicates inefficiently in chimpanzees, to a much lower level than it does in humans so it usually does not cause disease. However, there are recorded examples of HIV causing immunodeficiency in these animals.[78,79] Many agents which cause disease in man are unable to cause disease in a host of other species because they fail to infect, or infect poorly, or produce a different response. HIV has probably been in the chimpanzee population for a very long time. Therefore it is plausible that natural selection has rendered it less harmful. We note the presumably unintended irony in Farber's closing sentence in this footnote: *"Seldom do journalists insist on good hard evidence for these assertions."* In fact, most professional science writers do exactly that. Perhaps Farber will take the trouble to do so in the future.

Ref (page, col)	Error Type	Topic	Description
50; 2	**FALSE**	**HIV**	***Farber proceeds from column 2 to the end of column 3 to postulate other causes of AIDS and makes various statements about the demographics of AIDS. No sources are cited for her ponderings. This is a particularly poorly researched and fact-checked part of the article.***

Not a single credible peer-reviewed article published in a credible scientific journal since 1990 offers any support for what she says here. Instead of a complete point-by-point explanation, some critical comments are offered:

(1) HIV does affect the heterosexual population in the US, not just gay men.[80] The US population in which HIV infection is now spreading most rapidly is African-American women. Poverty (where untreated sexually transmitted infections, lack of prevention knowledge, lack of power of women to negotiate condom use, increased frequency of transactional sex are more likely than in wealthier populations), unprotected anal sex (due to greater risk of abrasions), blood transfusions, intravenous needle reuse and exposure to multiple partners. All increase risk of HIV transmission and explain the demographic aspects of the disease with which Farber fumbles.

(2) In contrast to Farber's implication that proposed causes of AIDS other than HIV have not been tested, they have – in great depth. These studies have found that in the absence of HIV none of recreational drug use, poverty, malnutrition and homosexuality can predict the onset of AIDS. Footnote 14 is consequently false too. There is no evidence that recreational drug use is the cause of AIDS. We quote an NIAID rebuttal to this myth:

"[I]n a prospectively studied cohort in Vancouver, 715 homosexual men were followed for a median of 8.6 years. Among 365 HIV-positive individuals, 136 developed AIDS. No AIDS-defining illnesses occurred among 350 seronegative men despite the fact that these men reported appreciable use of inhalable nitrites ("poppers") and other recreational drugs, and frequent receptive anal intercourse (Schechter *et al*. **Lancet. 1993 Mar 13;341(8846):658-9**).

Other studies show that among homosexual men and injection-drug users, the specific immune deficit that leads to AIDS - a progressive and sustained loss of CD4+ T cells - is extremely rare in the absence of other immunosuppressive conditions. For example, in the Multicenter AIDS Cohort Study, more than 22,000 T-cell determinations in 2,713 HIV-seronegative homosexual men revealed only one individual with a CD4+ T-cell count persistently lower than 300 cells/mm^3 of blood, and this individual was receiving immunosuppressive therapy (Vermund *et al*. **N Engl J Med. 1993 Feb 11;328(6):442**).

In a survey of 229 HIV-seronegative injection-drug users in New York City, mean CD4+ T-cell counts of the group were consistently more than 1,000 cells/mm^3 of blood. Only two individuals had two CD4+ T-cell measurements of less than 300/mm^3 of blood, one of whom died with cardiac disease and non-Hodgkin's lymphoma listed as the cause of death (Des Jarlais *et al*. **J Acquir Immune Defic Syndr. 1993 Jul;6 (7):820-2**).[81]

The use of some recreational drugs, such as metamphetamines, can place individuals at greater risk of acquiring HIV infection by lowering inhibitions and increasing the probability of engaging in, e.g., unsafe sexual practices. This does not mean that "drugs cause AIDS."

(3) Farber's claim that researchers have failed to demonstrate a higher incidence of AIDS in people with HIV is false. See the above studies. There are many more. Here is a further tiny sample of such studies, including some from Africa:

(i) The Multicenter AIDS Cohort Study (MACS) and the Women's Interagency HIV Study (WIHS) consisted of 8,000 participants in the US. It demonstrated that participants with HIV were approximately 1,100 times more likely than people without HIV to get a disease associated with AIDS.[82]

(ii) A one-year South African study of 1,792 HIV-positive and 2,970 HIV-negative gold miners found that miners with HIV were nearly three times more likely to be hospitalised and nine times more likely to die than HIV-negative ones.[83]

(iii) Researchers at Chris Hani Baragwanath Hospital in Johannesburg looked at deaths of HIV-positive and HIV-negative children between 1992 and 1996. They found that deaths increased among HIV-positive children but decreased among HIV-negative ones.[84]

(iv) A study in Uganda of nearly 20,000 people found that HIV-positive people had a death rate more than twenty times higher than HIV-negative people.[85]Incidentally, in this study, educated people and civil servants were more likely to die, which is inconsistent with poverty being the cause of AIDS (though it certainly is an exacerbating factor).

(v) In Cote d'Ivoire, HIV-positive people with TB were 15 times more likely to die within six months than HIV-negative people with TB.[86]

(vi) A study in Rwanda found that death was 21 times higher for HIV-positive children than for HIV-negative children.[87]

(vii) A study of pregnant women at King Edward Hospital in Durban, South Africa found that those with HIV had a ten times higher rate of turberculosis than those without.[88]

(viii) A study of over 6,000 people with haemophilia in the United Kingdom found that those with HIV had a much higher death rate. The death rate amongst HIV-negative haemophiliacs stayed stable during the analysis period (1977 to 1991). The death rate amongst haemophiliacs who contracted HIV rose dramatically from 1984 to the end of the study period.[89] This disproves Farber's assertion that no studies have been carried out to determine if haemophiliacs infected with HIV die sooner than those not infected.

(ix) As explained by the NIH *"Similar data have emerged from the Multicenter Hemophilia Cohort Study. Among 1,028 hemophiliacs followed for a median of 10.3 years, HIV-infected individuals (n=321) were 11 times more likely to die than HIV-negative subjects (n=707), with the dose of Factor VIII having no effect on survival in either group* (Goedert. **Lancet. 1995 Nov 25;346(8987):1425-6**).[90] Factor VIII is Duesberg's proposal for higher mortality in haemophiliacs with HIV. This study debunked this notion. See Cohen (1994)[91] for a more detailed discussion.

For further examples showing more illness and death among people with HIV, see NIAID (2003).[25] A diligent search on Medline will elicit even more examples.

(4) Farber provides no reference for her claim that HIV is a harmless passenger virus. The claim is false and disproven by the evidence presented in this document.

(5) Farber provides no reference for her claim that HIV is primarily spread from mother-to-child. The claim is false. Most HIV transmission is through heterosexual sex.

(6) In footnote 14 Farber claims that the majority of Kaposi's sarcoma patients are heavy users of nitrate inhalers. She gives no reference. Assuming she's right, if a sizeable minority are not, then nitrate inhalers cannot be the cause of Kaposi's sarcoma.

Once infected with HIV, recreational drug use and poverty are factors in the progression of HIV to AIDS, but HIV progresses to AIDS in sufficiently large numbers of well-off people who do not use recreational drugs to disprove that drugs or poverty are the cause of AIDS.

50; 3	**FAIRNESS**	**HIV**	***Farber criticises those who compare AIDS denialism to Holocaust denialism.***

Analogous to holocaust denialism, AIDS denialism is an insult to the memory of those who have died of AIDS, as well as to the dignity of their families, friends and survivors. As with Holocaust denialism, AIDS denialism is pseudo-scientific and contradicts an immense body of research.

			But in contrast to Holocaust denialism, AIDS denialism directly threatens lives *today* by trying to fool laypeople at risk of HIV not to get tested for the virus or not to practice safer sex. It also tries to fool those who need ARVs not to take them.
Ref (page, col)	**Error Type**	**Topic**	**Description**
51; 1	**MISLEADING**	**HIV**	***Footnote 15 refers to the scientific consensus on scurvy being overturned when it was finally realised that it was due to a vitamin C deficiency. The implication is that this is similar to the case of HIV.***
			Our present-day understanding of HIV and AIDS results from the efforts of thousands of scientists publishing tens of thousands of studies over 25 years. No other disease in history has been studied in this depth. It would require the exposure of an unprecedented conspiracy or duping for the scientific consensus on HIV as the cause of AIDS and the benefits of ARVs to be overturned. This is absurdly implausible. ARVs are probably more studied than any other class of drugs. Comparing HIV science to the history of scurvy is misleading and silly. Furthermore, the link between vitamin C and scurvy was definitively discovered in the 1930s, at the onset of the modern era of medical research. Scientific method in medicine has developed dramatically since then. Furthermore, Farber provides no reference for those proposing citrus fruit as a remedy for scurvy being dismissed as flat-earthers. Although scurvy was not properly understood until the 1930s, as early as the 17thcentury the surgeon general of the British East India Company suggested using fresh food including oranges, limes etc. as a preventative measure. This soon became standard practice in the British Royal Navy.[92] But Farber is also highly selective. She fails to mention the successes of pharmaceutical products in medical science. These include the numerous infections treated by penicillin, treatments for diabetes, cardiovascular disease, cancer, etc. These treatments have had a substantial effect on improved life-expectancy since the 20thcentury.
51; 3	**FAIRNESS**	**HIV**	***Footnote 16 refers to the reappraising petition with its approximate 2,300 signatories.***
			It appears that Farber is referring to one of two petitions on the virusmyth website (we assume this because Farber, as usual, does not supply references). Farber is a signatory to one of these petitions a fact that indicates she had prejudged views before writing the Harper's piece. The petition texts are vague. They have been open for signing for years. It is not clear how to remove one's name from them if one wishes to do so. It is difficult to verify which of them are scientists or have conducted AIDS research.

			By contrast, the Durban Declaration was signed by approximately 5,000 scientists, most of them engaged in HIV/AIDS research, in a short space of time. It declared what was already obvious and accepted: that HIV is the cause of AIDS. In any case, as the arguments here clearly show, there is no need to appeal to authority to demonstrate that HIV is the cause of AIDS, and that the benefits of ARVs outweigh their risks. The public domain research demonstrate these facts beyond reasonable doubt.
52	**FALSE**	**ARVs**	***Farber states "Duesberg thinks that up to 75 percent of AIDS cases in the West can be attributed to drug toxicity. If toxic AIDS therapies were discontinued, he says, thousands of lives could be saved virtually overnight."***
			This is merely Duesberg's opinion, for which there is not a shred of evidence. The evidence presented or referenced in this document demonstrates that Duesberg is wrong. Does Farber, in fact, disagree with Duesberg? In a recent widely circulated email, she states that her Harper's article "does not, for example, say that all AIDS drugs are ghastly, or worthless. In each article (in the past) where I have addressed HAART I have included, clearly, the fact that the regimens have absolutely helped people who are very sick." Or does she simply disagree with herself?

TABLE 2

Farber appeals to a number of authorities to confirm her views. It is worth noting the following:

Name	Description
David Rasnick	Rasnick is (or was until recently) on the payroll of Matthias Rath, a vitamin salesman who claims micronutrients treat HIV, heart disease, cancer, diabetes, asthma and many other serious diseases. Rath also claims that all pharmaceutical products are toxic and of no benefit in treating any of the aforementioned diseases.

	Farber refers to a nutritional study being conducted by Rasnick in South Africa. Here are the facts on this disgraceful unethical debacle: Rath and Rasnick have recently conducted a clinical trial in South Africa. The trial received no regulatory approval, involves convincing ill HIV patients not to take ARVs and promises them that the mega doses of vitamins (far in excess of RDA) prescribed treat AIDS. Investigations into this trial have resulted in allegations that a number of deaths have occurred and consequently litigation is underway in South Africa to stop it (launched by the Treatment Action Campaign and South African Medical Association). Rath and Rasnick have the support of the South Africa Minister of Health. Herein lies a scandal that Harper's should really be running a story on. For more information on the deeds of Rasnick's employer, see TAC (2006).[93]
Karry Mullis	Farber points out that Mullis discovered the PCR and is a Nobel laureate. What she fails to mention is that he has a wide range of odd beliefs. He does not believe in global warming, but does believe he might have been abducted by aliens and is partial to astrology. Entertaining perhaps, but if you're going to argue from authority this is not someone to quote.
Peter Duesberg	Farber fails to point out that Duesberg has almost no track record of published AIDS-related research in credible peer-reviewed journals. Farber also fails to point out that his new cancer hypothesis is also considered pseudo-science by most cancer scientists. Farber is also wrong about Duesberg being the youngest member ever elected to the National Academy of Sciences. There were many younger than 50, even at the time Duesberg was elected. Farber further claims that the National Cancer Institute (NCI) "refuses" to fund Duesberg. The NCI relies on peer review groups that score grants. If his grants fared poorly in study sections, that's far from NCI's "refusal" to fund him. So the implication that the institution has blackballed him from funding is both false and misleading. He, like anyone else, can continue submitting grant applications, and if peers deem it worthy, he'll receive funding.
Harvey Bialy	Farber points out that Bialy is the founding scientific editor of Nature Biotechnology. He no longer holds any position with Nature.
Jonathan Fishbein	NIAID publicly revealed the problems with the HIVNET 012 study long before Fishbein was hired. His original complaint was about recertifying the study site, and it somehow mutated into concerns about efficacy and safety of the intervention itself. He nor anyone else has demonstrated any evidence that undermines HIVNET 012's scientific results (e.g. an unrecorded side effect or death likely due to nevirapine).

	Fishbein, Farber writes, "supported Luzar in a sexual harassment claim against Kagan." Fishbein in fact filed it as a third party complaint.

Celia Farber	Celia Farber has been publishing articles for many years denying that HIV causes AIDS or that ARVs are effective.[94] Her views have been rebutted by the scientific community. She is not a scientist, yet clearly brings highly prejudiced views to this issue. She is obviously out of her depth trying to overturn the scientific consensus. Yet Harper's proceeded to publish her grossly inaccurate article without conducting proper fact-checking.

Correction History

3 March 2006: First public version. Sent to Harper's.
4 March 2006:
Modified (37;3) "Nevirapine remains an important antiretroviral medicine whose risks outweigh its benefits." to "Nevirapine remains an important antiretroviral medicine whose benefits outweigh its risks."
(b) Modified Bruce Mirken affiliation.
5-22 March: references standardised and typing errors corrected. Thank you to Sister Mary Elizabeth of **AEGiS** for assisting with these changes. This version is final.

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76 Like all viruses, HIV uses specific receptor molecules to bind to and then enter the cells it infects. The receptors used by HIV are CD4 and either CCR5 or CXCR4, proteins found on, inter alia, human CD4+ T-cells. The human CCR5 gene exists in several mutant forms (alleles), the most important of which is known as CCR5-delta32. This mutant allele encodes a defective protein that is not expressed on the cell surface. Individuals with one copy of the CCR5-delta32 gene (and one copy of the normal CCR5 gene) therefore have a reduced complement of CCR5 receptors for HIV; these individuals are heterozygous for the defective CCR5 allele. Individuals with two copies of the CCR5-delta32 gene have no functional CCR5 proteins on their cells; they are homozygous for the defective CCR5 allele. CCR5-delta32 homozygotes are strongly protected from acquiring HIV infection, because the most commonly transmitted variants of HIV require CCR5 to enter cells (and hence the human body). These individuals can, however, still be infected by the less commonly transmitted HIV variants that use, instead of CCR5, the CXCR4 receptor, so the protection conferredby the mutant CCR5-delta32 allele is not absolute. CCR5-delta32 heterozygotes are not protected from acquiring HIV infection, but they progress less rapidly to AIDS and death than individuals with two normal CCR5 alleles because the lower levels of CCR5 on their cells support the spread of HIV within the body less efficiently. The CCR5-delta32 mutant allele arose spontaneously in the human genome several thousand years ago, most probably in Northern Europe (hence the allusion to "European Caucasians"). Its frequency nowadays in European populations varies from countryto-country (higher in the north of the continent, less so in the south) but, on average, about 1% of contemporary Europeans are CCR5-delta32 homozygotes and about 15-20% are CCR5-delta32 heterozygotes. The mutant allele is also found in populations that have migrated from Europe during the past few thousand years (e.g., in North Americans of European origin) and in populations that have bred with Europeans. It is not found in populations of African or Asian origin. The distribution of the CCR5-delta32 allele is not affected by behavior, so "gay men, intravenous drug usersand transfusion receptors" are as likely (or unlikely) to possess the mutant as any other members of the general population in which they reside. There is nothing "mysterious" about any of this; the facts are clearly established in the scientific literature, and even in popular science magazines, as shown by a small selection of the available reviews on the subject (Berger EA, Murphy PM, Farber JM. *Chemokine receptors as HIV-1 coreceptors: roles in viral entry, tropism, and disease*. **Annu Rev Immunol. 1999;17:657-700**; Moore JP, Kitchen SG, Pugach P, Zack JA. *The CCR5 and CXCR4coreceptors--central to understanding the transmission and pathogenesis of human immunodeficiency virus type 1 infection*. **AIDS Res Hum Retroviruses. 2004 Jan;20(1):111-26**; O'Brien SJ, Moore JP. *The effect of genetic variation in chemokines and their receptors on HIV transmission and progression to AIDS*. **Immunol Rev. 2000 Oct;177:99-111**; O'Brien SJ, Dean M. *In search of AIDS-resistance genes*. **Sci Am. 1997 Sep;277 (3):44-51**).

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HIV TESTS CANNOT DIAGNOSE HIV INFECTION

A reply to several of the numerous fallacies contained in the document entitled "Errors in Celia Farber's March 2006 article in Harper's Magazine" (Gallo et al 2006).

Roberto A. Giraldo, MD[1] Etienne de Harven, MD[2]

CONTENTS

1. Several false statements regarding HIV testing by Gallo, Geffen, Gonsalves, et al. (Gallo et al 2006).

On March 4, 2006, Robert Gallo, together with pro-antiretroviral HIV activists from the Treatment Action Campaign in South Africa, the Gay Men's Health Crisis in the USA, the Elizabeth Glaser Pediatric AIDS Foundation, also in the USA, and others (Gallo et al 2006), released an alleged rebuttal of an article by Celia Farber in the March 2006 issue of Harper's Magazine: "Out of control: AIDS and the corruption of medical science" (Farber 2006).

Regarding HIV testing, Gallo and his co-authors assert that (Gallo et al 2006):

"HIV tests were highly accurate from the time they were developed in 1984 and have become much more accurate over time as the underlying technology has evolved. HIV tests are amongst the most accurate available in medical science."

"A PCR test for the presence of the virus itself can accurately determine a child's HIV status."

"An AIDS diagnosis cannot be considered definitive without an HIV test."

"Farber's comment about hopping on a plane from Uganda to Australia to change HIV diagnosis is simply silly hyperbole."

"The risk of a false positive HIV test in Africa, as elsewhere, is very small if the correct protocol is followed. Some HIV antibody tests have been tested in Africa and found to be very accurate. These are the ones generally used. For example, the Abbott Determine rapid test

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[1] **Physician specialist in internal medicine, infectious and tropical diseases. Independent AIDS researcher. Member of the South African Presidential AIDS Advisory Panel. Former President of Rethinking AIDS. Queens, New York.**

[2] **Physician, specialist in pathology, virology and electron microscopy. Emeritus Professor of Pathology, University of Toronto, Toronto, Canada. Member of the South African Presidential AIDS Advisory Panel. President of Rethinking AIDS, France.**

used widely in South Africa has a specificity of at least 98% (and in some studies has achieved close to 100%). When this test is combined with a second rapid test or an ELISA test to determine HIV status, the risk of a false positive is negligible. The contribution of TB and malaria to false positives on today's tests is also negligible."

"A properly conducted HIV-test protocol (which involves at least two HIV tests) has very small chance of giving a false positive, irrespective of pregnancy status."

However, available scientific data do not validate these statements. Several established scientific facts supporting the contention that HIV tests cannot diagnose HIV infection are as follows:

2. Pharmaceutical companies acknowledge that HIV tests are not specific for HIV.

The primary tests for the diagnosis of HIV infection are two antibody tests, the ELISA and Western blot, and a genetic test, the PCR or "Viral Load" test. However, the ELISA and Western blot tests only detect antibodies against what are erroneously accepted to be HIV proteins or antigens. Similarly, the PCR or Viral Load test for HIV only detects copies of fragments of RNA that have arbitrarily been regarded as the nucleic acid of HIV. None of these tests detect the HIV virus itself, nor do they detect HIV particles.

The pharmaceutical corporations that manufacture and commercialize these test kits acknowledge the inaccuracy of the tests. This explains the seemingly surprising statement included in the kit inserts: "Elisa testing alone cannot be used to diagnose AIDS, even if the recommended investigation of reactive specimens suggests a high probability that the antibody to HIV-1 is present" (Abbott 1997).

The insert for one of the kits for administering the Western blot warns: "Do not use this kit as the sole basis of diagnosis of HIV-1 infection" (Epitope Organon Teknika).

In like manner, the insert that accompanies a very frequently used test for PCR Viral Load warns: "the Amplicor HIV-1 Monitor test is not intended to be used as a screening test for HIV or as a diagnostic test to confirm the presence of HIV infection" (Roche 2003).

Therefore, the pharmaceutical drug manufacturers acknowledge the fact that neither the ELISA, nor the Western blot, nor the Viral Load tests for HIV are specific to diagnosis HIV infection.

Interestingly, the only valid method of establishing the sensitivity and the specificity of a diagnostic test in clinical medicine is to compare the test in question with its gold standard. The only possible gold standard for the HIV tests is the human immunodeficiency virus itself, HIV. Since HIV has never been isolated as an independent, free and purified viral particle, it is not possible to properly define either the sensitivity or the specificity of any of these tests. Currently, the sensitivity and the specificity of the tests for HIV are arbitrarily defined, not by comparison to purified HIV itself, but by comparison of the tests in question with the clinical manifestations of AIDS, or with T4 cell counts. This explains why Abbott clearly states: "At present there is no recognized standard for establishing the presence and absence of HIV-1 antibody in human blood. Therefore sensitivity was computed based on the clinical diagnosis of AIDS and specificity based on random donors" (Abbott 1997). Since there is no gold standard for defining the specificity of the tests used for the diagnosis of HIV infection, all HIV-positive results for HIV infection must be considered false positives. Therefore no individual can validly be identified as either HIV-positive or HIV-negative.

The large majority of AIDS researchers, journalists, lay people, and health care workers themselves do not understand the limitations of these tests because they do not have access to

the relevant data. Additionally, little or no concern is expressed by medical faculties and research institutions with regard to communicating these facts to physicians, let alone to the general public.

3. HIV has never been either isolated or purified as a real virus.

Proper procedures for isolating and purifying retroviruses (formerly known as RNA tumor viruses) were established as early as 1964 (O'Connor et al 1964; De Harven 1965a,b, 1974).

The most common sources of material from which retroviruses can be isolated and purified are blood (viremia), other tissue homogenates, and supernatant fluids from infected cell cultures (de Harven 1965a,b).

The most frequently used technique for isolation and purification of retroviruses includes the following primary steps: (1) Concentration of the viral particles by centrifugation; (2) Electron microscopy monitoring of the concentrated viral particles; (3) Biochemical and genetic analysis of the purified viral particles; (4) Controlling the experiments to avoid misinterpreting endogenous retroviruses as exogenous infectious retroviruses; and (5) Biological tests to ascertain if the isolated retrovirus is indeed potentially pathogenic and virulent (O'Connor et al 1964; De Harven 1965a,b, 1974).

However, neither Montagnier, nor Gallo, nor Levy et al. had adhered to these techniques when they claimed to have isolated "the AIDS virus" in 1983 and 1984 (Barré-Sinousi et al 1983; Papovic et al 1984; Gallo et al 1984; Levy et al 1984). The first two steps were omitted; they did not provide the electron microscope evidence that particles from the "infected" culture supernatant, sedimenting at 1.16 gm/ml of sucrose, were composed primarily of viral particles (concentrated viral particles). Instead, they provided electron microscope photographs of stimulated/activated cultured lymphocytes releasing particles similar to retroviruses. These same particles, however, can be released by "non infected" stimulated/activated lymphocyte cultures (Dourmashkin et al 1993). Unfortunately, the experiments were not properly controlled; where were the electron microscopy photographs of "infected" as well as "not infected" culture supernatants sedimenting at 1.16 gm/ml of sucrose, EM micrographs required to determine whether or not viral particles were concentrated at that gradient? Additionally, where were the electron microscopy pictures of "non infected" lymphocytes grown under identical culture conditions?

The alleged existence of HIV was asserted from the study of proteins, reverse transcriptase activity (RT), and RNA fragments that were found in culture supernatants, not from the direct analysis of purified viral particles.

Surprisingly, the existence of HIV was then claimed indirectly, on the basis of the presence in complex cell cultures and/or "HIV-positive" individuals of (1) proteins/glycoproteins such as gp160/150, gp120, gp41/45/40, p34/32, p24, and p18/17, each claimed to belong to HIV; (2) enzymes such as reverse transcriptase that supposedly belongs to HIV; and (3) RNA or DNA fragments that supposedly belong to HIV (Papadopulos-Eleopulos et al 1993, 1996, 1997a, 1997b, 1997/8; Turner 1996, 1997/1998, 1998; Philpott 1997; Giraldo et al 1999; de Harven 1997/8, 1998, 2002a,b). However, none of these substances have been proven to belong to HIV. How could it be proven that the molecules found in those cultures actually belong to viral particles that have never been properly purified? How could it possibly be demonstrated that these substances are not simply cellular microvesicles or cell debris contained in the cultures and that happen to sediment at the same density as retroviruses? In order to prove that those molecules, allegedly regarded as "markers", are part of a retrovirus named HIV, it would have been absolutely necessary to purify the retroviral particles, separating the particles from everything else. This has never been done with HIV (Papadopulos-Eleopulos et al 1996; de Harven 1998; Giraldo et al 1999).

However, long before the appearance of the first cases of AIDS, researchers working on "RNA tumor viruses", currently known as retroviruses, clearly knew that the first prerequisite for the study of virus subcomponents or molecules is to obtain highly purified virus preparations (de-The & O'Connor 1966). After purifying the "murine leukemia virus", these authors were able to employ selected chemicals (i.e. tween-ether, ribonuclease, detergents) to disrupt the purified particles and release the internal components (de-Thé & O'Connor 1966). This was never done with HIV.

One of us has insisted that: "The specificity of viral markers depends on the success of virus isolation and purification. Without fully demonstrated success in virus isolation and purification, identification of *viral markers* is extremely hazardous and can lead to severe misinterpretation of clinical data. A dramatic illustration of this is to be found in current HIV research. In this case, the virus (HIV) has never been properly isolated, since sedimentation in sucrose gradient at the density of 1.16 g/mL was erroneously considered to yield *pure virus*, systematically ignoring that material sedimenting at that density contains large amounts of cell debris and cellular microvesicles (Gluschankof et al 1997; Bess et al 1997). Therefore, proteins and nucleic acids found in such *1.16 bands* are very likely to be of cellular origin and cannot be used as viral markers. Such a faulty methodology has had extremely serious consequences, i.e. the world-wide use of HIV-antibody tests, ELISA and Western blot, which dangerously lack specificity, as demonstrated in 1993 by Papadopulos et al. (1993), in Australia" (de Harven 1999).

"More disturbing is the fact that some 'markers' are searched for in the 1.16 gradient sedimenting material which is the density where intact virions are expected to be found, but not their molecular fragments. If lysed retrovirus particles released molecular markers, the 1.16 samples should at least initially allow researchers to demonstrate virus particles by electron microscopy. However, after 15 years of most intensive HIV research, two independent groups finally decided to explore by electron microscopy the ultrastructural features of the material sedimenting at the 1.16 density. Working on 'HIV-1 infected T-cell' cultures supernatants, both groups found that it contains primarily cellular debris and cell membrane vesicles which could definitely not be identified with HIV particles and rare 'virus-like' particles (Gluschankof et al 1997; Bess et al 1997). Still this is the type of sample in which 'viral markers' are currently identified and used to measure the effects of anti-viral drugs in current clinical trials" (de Harven 1998).

The reverse transcriptase activity (RT) found in culture supernatants by researchers who claim to have isolated "the AIDS virus" (Barré-Sinousi et al 1983; Papovic et al 1984; Gallo et al 1984; Levy et al 1984) could just as well have a cellular origin, since this enzyme is ubiquitous (Ross et al 1971; Beljanski 1972; Varmus 1987; Coffin et al 1997). RT is not a unique feature of retroviruses, as it was mistakenly thought to be by Montagnier, Gallo and Levy's group.

HIV has never been either isolated or purified as intact viral particles. Therefore, there is no scientific data validating the contention that what is currently referred to as HIV is in fact a virus!

There does not exist a single test tube in any laboratory anywhere containing purified particles of HIV. Researchers working with what they believe to be HIV in laboratories all around the world are most likely not working with HIV particles at all. They are working with proteins, enzymes, or fragments of RNA that have been arbitrarily regarded as belonging to HIV.

The fact that after 25 years of intense research HIV has been neither isolated nor purified in terms of classical virology indicates to us that the infectious view of AIDS as a contagious viral disease is based on an apparently non-existent microbe!

4. So-called "HIV proteins" are not specific markers of HIV.

In the early 1980s, frustrated retroviral cancer researchers trying to prove that AIDS was a retroviral disease, arbitrarily defined what they erroneously called "the AIDS virus proteins," "the AIDS virus enzymes," and "the AIDS virus RNA," which were found in the supernatant of cultures, without having previously either isolated or purified the retroviral particles, i.e., separated them from cellular microvesicles and cell debris, as has been was explained in the previous section.

Montagnier's group from the Pasteur institute in France, for example, determined what they call "viral antigens" through a series of immunoprecipitation experiments (Western blot) using cord blood lymphocytes mixed within very complex cell culture systems, with virus from patient 1 as a source of "viral antigens" and antiserum to HTLV-I P24 and serum from patient 1 and 2, and arbitrarily decided that: "three major proteins could be seen: the p25 protein and protein with molecular weights of 80,000 and 45,000. The 45K protein may be due to contamination of the virus by cellular actin which was present in immune precipitates of all the cell extracts" (Barré-Sinoussi et al 1983). Without having previously purified viral particles, they erroneously concluded that, "these results, together with the immuno precipitates, indicate that the retrovirus from patient 1 contains a major p25 protein, similar in size to that of HTLV-1 and different immunologically" (Barré-Sinoussi et al 1983).

Gallo's group from the National Cancer Institute performed Western blot using "lysates of HTLV-III producer cell clones" and serum diluted 1:500, and, also without having previously purified viral particles, arbitrarily decided that, "antigens newly expressed after viral infection and recognized by the human serum used included p65, p55, p41, p39 and p24. A large protein with a molecular weight of approximately 130,000 and a protein of 48,000 were also detected" (Schüpbach et al 1984). However, they also concluded that, "these results show clearly that the antigens detected after viral infection are either virus-coded proteins or cellular antigens specifically induced by the infection" (Schüpbach et al 1984). Additionally, they concluded that, "extensive accumulation of p24 and p41 occurred in the virus preparation which showed that these molecules are the major components of the virus preparation. Allegedly, P24 and p41 were, therefore, considered as viral structural proteins" (Schüpbach et al 1984).

Levy's group of researchers, from the University of California in San Francisco, performed standard indirect immune fluorescence procedures using HTLV-1, LAV and ARV "infected cells" and serum diluted 1:10. They found that antibodies against what was supposed to be ARV (AIDS Related virus) in 88% of AIDS with Kaposi's sarcoma, 100% in AIDS with opportunistic infections, in 93% of male sexual partners of AIDS patients, and in 57% of clinically healthy homosexual men (Levy et al 1984).

These three groups of researchers decided, arbitrarily, that the proteins they found in cell cultures apparently infected with "the AIDS virus" were "HIV proteins." These proteins had not been and have never been extracted directly from isolated, purified viral particles. They could, therefore, just as well have a human cellular origin.

On the other hand, in 1997, the Gluschankof group in France and Germany, as well as the Bess et al group in the United States demonstrated that when one follows the routine procedure to isolate retroviruses from cultures that are supposedly infected with HIV, it is not possible to either isolate or purify virus particles, separated from cellular microvesicles and cell debris, even in fractions sedimenting at the density, in sucrose gradients, where retrovirues are classically known to sediment (Gluschankof et al 1997; Bess et al 1997). They rightly warned that, "caution must therefore be exercised in terms of the presence of cellular vesicles when viral immunogens (*proteins*) are density gradient enriched" (Gluschankof et al 1997), because "human cellular antigens have been found associated with HIV-1 preparations" (Gluschankof et al 1997). Therefore, these 1997 papers from the Gluschankof and Bess groups provide an

objective demonstration that what are commonly called "HIV proteins" or "HIV antigens" or "HIV immunogens" are not specific markers of HIV and could very well originate from the cultured cells.

In this regard, our colleagues from Perth, Australia, have explained several times that the Western blot antigens, proteins, glycoproteins or bands - p120, p41, p32, p24/25, p17/18 - allegedly considered to be specific HIV proteins may not be encoded by the HIV genome but may in fact represent cellular proteins originating from the cultured human cells (Papadopulos-Eleopulos et al 1993, 1997a; Turner 1996, 1997/1998). The normal cell component actine probably corresponds to what is known as gp41, while gp120/160 probably represent gp41 oligomers (Papadopulos-Eleopulos et al 1993).

Therefore no one has, to date, presented evidence that the so-called HIV proteins or antigens [gp160/150, gp120, gp41/45/40, p34/32, p24, p18/17], are really constituents of HIV (Papadopulos-Eleopulos et al 1993, 1996; de Harven 1998, 2002a, 2003; Giraldo 2002a; Giraldo et al 1999).

The proteins and glycoproteins listed above ("HIV antigens") are claimed to appear exclusively when one co-cultures supposedly infected blood with abnormal cells from leukemic patients, or from umbilical cord lymphocytes (Papadopulos-Eleopulos et al 1996; de Harven 1998). Quite probably, the same molecules could be obtained from similar cultures in the absence of "HIV" infection. However, very crucial control experiments were never performed (de Harven 1998, 2003, 2004) specially when researchers used cord blood lymphocytes. These cells of placenta provenance are very likely to be a source of endogenous, probably defective retroviruses (Panem 1979; de Harven 2002b).

Moreover, the cultures where the above substances have been found have been heavily stimulated with phytohemagglutinin, IL-2, antiserum to human interferon, and other agents (Papadopulos-Eleopulos et al 1996; de Harven 1998, 2003). These culture stimulants are oxidizing agents and could be expected to stimulate the expression of endogenous retroviruses (Papadopulos-Eleopulos et al 1996). Control experiments on these important points cannot be found in the literature. Interestingly, neither HIV itself nor any HIV markers can be found when the cultures are treated with antioxidants (Papadopulos-Eleopulos 1988, 1998/9; Papadopulos-Eleopulos et al 1992, 1993).

Unfortunately, these alleged "HIV proteins" or "HIV antigens" are used as antigens in the serologic tests for HIV, and this explains the complete lack of specificity of these tests.

5. So-called HIV-RNA is not a specific marker of HIV.

The HIV viral load test is an amplification genetic test that makes copies of fragments of RNA that arbitrarily have been regarded as parts of the HIV genome. These fragments of RNA are found in culture supernatants or in patient's blood. They are never, however, extracted directly from purified viral particles. What is known as "HIV RNA" might just as well originate from cultured cells or be present in the blood of persons undergoing stress. It could also originate from endogenous, non-infectious retroviruses.

Moreover, it has been established that the human genome contains a sizable proportion of endogenous retrovirus-related sequences (Mager & Freeman 1987; Lieb-Mösch et al 1990).

In the decade prior to the appearance of AIDS, during President Richard Nixon's "War Against Cancer", in order to identify "viral proteins" and to extract "viral RNA" samples, researchers successfully used highly purified retrovirus specimens from "viremic" animals. The method applied to achieve this purification of a typical retrovirus was rapid, inexpensive and reproducible (de Harven 1965a,b). However, "most surprisingly, nobody has ever succeeded in

demonstrating HIV particles in the blood of any AIDS patient by this simple method, even though patients could have been selected for presenting a so-called high 'viral load' as determined by PCR methods" (de Harven 2003). PCR is a genetic technique that does not count viral particles at all (Mullis & Faloone 1987), as physicians and lay people may think. It merely makes copies of what is supposed to be HIV RNA (Roche 2003).

"It appears very likely that PCR methods amplify small RNA fragments, more frequently observed under conditions of stress and other chronic illnesses (Urnovitz et al 1999), and which include retroviral segments originating from human endogenous retroviruses. This is not surprising since about 2% of the human genome have marked homology with retroviral genome (Löwer et al 1996). Consequently, 'measuring' the 'viral load' by PCR methods is likely to have no relationship whatsoever with real quantification of a hypothetical exogenous HIV viremia. Kary Mulis himself, Nobel Prize laureate for his discovery of the PCR method, categorically rejects the use of 'his' method for quantitative measurements of a hypothetical HIV viremia (Mullis 1998)" (de Harven 2003).

"HIV cloning" is, likewise, very misleading. Without first isolating and purifying retroviral particles, the cloning of a "specific HIV-RNA" is not possible (Papadopulos-Eleopulos et al 1996; de Harven 1998; Giraldo et al 1999). Neither does the cloning of fragments of nucleic acid found in supernatants of supposedly "HIV-infected" cultures indicate HIV. The only way to properly achieve HIV cloning would be first to isolate and purify HIV particles and then to extract RNA from the core of the purified particles. This has never been done with HIV!

However, in 1985, researchers from the National Cancer Institute and from the Dana-Farber Cancer Institute of Harvard University claimed to have found the "complete nucleotide sequence of the AIDS virus, HTLV-III" (Ratner et al 1985). They arbitrarily stated that: "The complete nucleotide sequence of two human T-cell leukemia type III (HTLV-III) proviral-DNA each have four long open readings frames, the first two correspond to the *gag* and *pol* genes. The fourth open reading frame encodes two fractional polypeptides, a large precursor of the major envelope glycoprotein and a smaller protein derived from the 3'-terminus long open reading frame analogous to the long open reading frame (*lor*) product of HTLV-I and –II;" "the HTLV-III is 9,749 pairs (bp) long. The overall structure of the provirus resembles that of other retroviruses" (Ratner et al 1985). And, they continue, "sequences from different clones of HTLV-III allow an analysis of the level of sequence diversity of the virus. A comparison of clones BH8 and BH5 with BH10 demonstrates a 0.9% base pair polymorphism in the coding regions of the genome and a 1.8% base pair polymorphism in the non-coding regions. The heterogeneity among HTLV-III clones shown here could represent sequence divergence developing in culture in a given individual over a period of time, or polymorphic differences in viruses from different individuals. Diversity among different HTLV-III isolates seems to be greater than that between different HTLV-I isolates. Thus, it is likely that most of the divergence among the HTLV-III clones analyzed here represents differences in strains in different individuals" (Ratner et al 1985). However, this statement can only be valid for a fragment of DNA (HTLV-III clone) that the American researchers arbitrarily considered to be "T-cell leukemia type III (HTLV-III) proviral-DNA." Individuals reading this without a critical perspective might therefore be mislead by the researchers from the NIH and Harvard University.

One of us described this chaotic situation during a debate on AIDS in Africa, held at the European Parliament in Brussels, as follows: "the 'Viral Load' of newspapers and magazines, all over the world is extremely high, meaning the number of pictures of HIV published almost daily in the world's press. These pictures are extremely attractive, and are frequently rich in artificial colors. They clearly exemplify the danger of misinforming the public with computer graphics. To publish such images brings to the attention of the general public, and of the medical profession as well, an apparently crystal-clear message: yes, HIV has been isolated since one can portray it under the electron microscope. All these images represent computerized rationalizations" (de Harven 2003), always derived from particles observed in

complex and probably contaminated cell cultures, but never derived directly from one single AIDS patient.

"HIV viral load" cannot, therefore, diagnose HIV infection.

6. False positive reactions on the HIV tests.

There are abundant scientific publications explaining that there are more than 70 different documented conditions that can cause the antibody tests to react positive without an HIV infection (Johnson 1993, 1995, 1996a,b; Hodgkinson 1996; Turner 1996, 1997/8; Shenton 1998; Papadopulos-Eleopulos et al 1993; Giraldo 1997d, 2000a; Giraldo et al 1999).

Some of the conditions that cause false positives on the so-called "AIDS test" are: past or present infection with a variety of bacteria, parasites, viruses, and fungi including tuberculosis, malaria, leishmaniasis, influenza, the common cold, leprosy and a history of sexually transmitted diseases; the presence of polyspecific antibodies, hypergammaglobulinemias, the presence of auto-antibodies against a variety of cells and tissues, vaccinations, and the administration of gamma globulins or immunoglobulins; the presence of auto-immune diseases like erythematous systemic lupus, sclerodermia, dermatomyositis and rheumatoid arthritis; the existence of pregnancy and multiparity; a history of rectal insemination; addiction to recreational drugs; several kidney diseases, renal failure and hemodialysis; a history of organ transplantation; presence of a variety of tumors and cancer chemotherapy; many liver diseases including alcoholic liver disease; hemophilia, blood transfusions and the administration of coagulation factor; and even the simple condition of aging and some vaccinations, to mention the most important (Johnson 1993, 1995, 1996a,b; Hodgkinson 1996; Turner 1996, 1997/8; Shenton 1998; Papadopulos-Eleopulos et al 1993; Giraldo 1997d, 2000a).

Christine Johnson, from California, has listed, from the scientific literature, the following conditions that cause false-positive reactions in the antibody tests for HIV (Johnson 1996a,b):

- Natural occurring polyspecific antibodies (Barbacid et al 1980; Healey & Bolton 1993).
- Anti-carbohydrate antibodies (Snyder & Fleissner 1980; Healey & Bolton 1993; Cordes & Ryan 1995).
- Antibodies with a high affinity for polystyrene used in the test kits (Arnold et al 1994; Pearlman & Ballas 1994; Yoshida et al 1987).
- HLA antibodies to class I and II leukocyte antigens (Blanton et al 1987; Bylund 1992; Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993; Sayers et al 1986; Schleupner 1990; Schochetman & George 1992; Steckelberg & Cockerill 1988; Yu et al 1989).
- Passive immunization (receipt of gammaglobulin or immune globulin as prophylaxis against infection) (Ascher & Roberts 1993; Cordes & Ryan 1995; Gill et al 1991; Jackson et al 1988; Lai-Goldmnan et al 1987; Isaacman 1989; Profitt & Yen-Lieberman 1993; Piszkiewicz 1987; Yale et al 1994).
- Administration of human immunoglobulin preparations (Bylund et al 1992).
- Hypergammaglobulinemia (high levels of antibodies) (Moore et al 1986; Peterman et al 1986).
- Globulins produced during polyclonal gammopathies, very common in groups at risk for AIDS (Bylund et al 1992; Cordes & Ryan 1995; Schleupner 1990).
- Anti-lymphocyte antibodies (Mathe 1992; Ujhelyi et al 1989).
- Anti-collagen antibodies (found in gay men, hemophiliacs, Africans of both sexes and people with leprosy) (Mathe 1992).
- Multiple blood transfusions (Cordes & Ryan 1995; Ng 1991; Peterman et al 1986; Proffit & Yen-Lieberman 1993; Schochetman & George 1992; Yu et al 1989; Sayre 1996).
- Individuals with coagulation defects (Bylund et al 1992; Schochetman & George 1992).
- Hepatitis B vaccination (Jackson et al 1988; Lee et al 1992; Pearlman & Ballas 1994; Profitt & Yen-Lieberman 1993).

- Tetanus vaccination (Pearlman & Ballas 1994).
- False positive in other serologic tests, including RPR for syphilis (Bylund et al 1992; Fleming et al 1987; Moore et al 1986; Schleupner 1990; Schocheman & George 1992).
- Healthy individuals as a result of poorly-understood cross-reactions (Bylund et al 1992).
- Anti-hepatitis A IgM antibody (Schleupner 1990).
- High levels of circulating immune complexes (Biggar et al 1985; Moore et al 1986).
- Presence of normal human ribonucleoproteins (Cordes & Ryan 1995; Schleupner 1990).
- Malaria (Biggar et al 1985; Charmot & Simon 1990).
- Visceral Leishmaniasis (Ribiero et al 1993).
- Leprosy (Andrade et al 1991; Kashala et al 1994).
- Tuberculosis (Kashala et al 1994).
- Mycobacterium avium (Kashala et al 1994).
- Autoimmune diseases: systemic lupus erythematous, scleroderma, connective tissue disease, dermatomyositis (Bylund et al 1992; Leo-Amador et al 1990; Pearlman & Ballas1994; Proffit & Yen-Lieberman 1993; Ranki et al 1992; Schochetman & George 1992).
- Systemic Lupus erythematosus (Esteva et al 1992; Jindal et al 1993).
- Rheumatoid arthritis (Ng 1991).
- Serum-positive for rheumatoid factor, antinuclear antibodies, and other autoantibodies (Dock et al 1988; Steckelberg & Cockerill 1988; Yoshida et al 1987).
- Anti-smooth muscle antibody (Schleupner 1990).
- Anti-mitochondrial antibodies (Cordes & Ryan 1995; Schleupner 1990).
- Anti-microsomal antibodies (Mortimer et al 1985).
- Other antinuclear antibodies (Cordes & Ryan 1995; Schleupner 1990; Steckelberg & Cockerill 1988).
- Anti-T-cell antigen antibodies (Cordes & Ryan 1995; Schleupner 1990).
- Renal failure (Cordes & Ryan 1995;Jindal et al 1993; Schleupner 1990).
- Hemodialysis (Bylund et al 1992; Fassbinder et al 1986; Peterman et al 1986; Schochetman & George 1992; Ujhelyi et al 1989).
- Alpha interferon therapy in hemodialysis patients (Sungar et al 1994).
- Renal transplantation (Burkhardt et al 1987; Cordes & Ryan 1995; Neale et al 1985; Schleupner 1990; Ujhelyi et al 1989).
- Organ transplantation (Agbalika et al 1992; Ng 1991).
- Upper respiratory tract infection (cold or flu) (Challakere & Rapaport 1993).
- Acute viral infections, DNA viral infections (Cordes & Ryan 1995; Pearlman & Ballas 1994; Profitt & Yen-Lieberman 1993; Schleupner 1990; Steckelberg & Cockerill 1988; Voevodin 1992).
- Flu (Ng 1991).
- Flu vaccination (Arnold et al 1994; Challakere & rapaport 1993; Cordes Y Ryan 1995; Hsia 1993; MacKenzie et al 1992; Profit & Yen-Lieberman 1993; Simonsen et al 1995).
- Herpes simplex I (Langedijk et al 1992).
- Herpes simplex II (Challakere & rapaport 1993).
- Epstein-Barr virus (Ozanne & Fauvel 1988).
- Recent viral infection or exposure to viral vaccines (Challakere & Rapaport 1993).
- Pregnancy in multiparous women (Cordes & Ryan 1995; Ng 1991; Profitt & Yen-Lieberman 1993; Steckelberg & Cockerill 1988; Ujhelyi et al 1989; Abbott 1997).
- Cancers (Pearlman & Ballas 1994).
- Multiple myeloma (Bylund et al 1992; Profitt & Yen-Lieberman 1993; Steckelberg & Cockerill 1988).
- Hematologic malignant disorders and lymphomas (Burkhardt et al 1987; Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993; Schleupner 1990; Steckelberg & Cockerill 1988).
- Q fever with associated hepatitis (Yale et al 1994).
- Hepatitis (Sungar 1994).

- Alcoholic liver disease (Bylund et al 1992; Cordes & Ryan 1995; Mendenhall et al 1986; Pearlman & Ballas 1994; Schleupner 1990; Schochetman & George 1992; Steckelberg & Cockerill 1988).
- Primary sclerosing cholangitis (Schochetman & George 1992; Steckelberg & Cockerill 1988).
- Primary biliary cirrhosis (Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993; Schleupner 1990; Steckelberg & Cockerill 1988).
- Stevens-Johnson syndrome (Burkhardt et al 1987; Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993).
- Sticky blood in Africans (Mortimer et al 1985; Papadopulos-Eleopulos 1988; Pearlman & Ballas 1994).
- Heat-treated specimens (Jungkind et al 1986; Schleupner 1990; Schochetman & George 1992; Smith et al 1987; Van Beers et al 1985).
- Lipemic serum (Schochetman & George 1992).
- Hemolyzed serum (Schochetman & George 1992).
- Hyperbilirubinemia (Bylund et al 1992; Cordes & Ryan 1995).
- Proteins in the equipment used for these tests (Cordes & Ryan 1995).
- Other retroviruses (Blomberg et al 1990; Cordes & Ryan 1995; Dock et al 1988; Schleupner 1990; Tribe et al 1988).

Therefore, there is a growing number of conditions known to cause the tests for HIV to react positively in the absence of HIV, i.e. false positives.

Interestingly, all of the conditions causing "HIV tests" to react positive in the absence of HIV are conditions which are present, with varied distribution and concentration, in many recognized “AIDS risk groups” in the developed countries, as well as in a large percentage of Africans and people from other parts of the developing world. This means that in all probability many drug users [including some mothers], certain gay males, and some hemophiliacs in the developed countries, as well as the vast majority of inhabitants in most countries of Africa, Asia, South America and the Caribbean, who have positive reactions to the tests for HIV, may very well do so due to conditions other than being infected with HIV (Johnson 1993, 1995, 1996a,b; Hodgkinson 1996; Turner 1996, 1997/8; Shenton 1998; Papadopulos-Eleopulos et al 1993, 1997; Giraldo 1997c, 2000a).

It is shocking to realize that a diagnosis of HIV infection is so frequently based on tests that are not specific for HIV, and even more so when one realizes that these non specific tests lead to the prescription of highly toxic anti-retroviral drugs.

7. The real meaning of being “HIV-positive” or “seropositive”

The definition of AIDS, as developed by the United States Federal Government’s Centers for Disease Control and Prevention, requires a positive result on the antibody test for HIV (CDC 1992). The importance of HIV in this definition is so strong that, currently, many AIDS researchers, health care professionals and lay people, in following the lead of the United States Institute of Medicine, the National Academy of Sciences and most AIDS researchers, now refer to "AIDS" as "HIV Disease" (Institute of Medicine 1986; Volberding & Cohen 1994; Fauci 1993; Staprans & Feimberg 1997; Lewis & Ho 2003; Wormser 2004).

However, AIDS in many countries of Africa can be diagnosed without an HIV test or any other laboratory test. This was decided by American public health officials and the World Health Organization at a conference in Bangui, in the Central African Republic, in October 1985 (Quinn et al 1986). This allows health professionals to diagnosis AIDS in Africa based only on routine clinical symptoms and signs presented by the patient. However, the most prevalent diseases in Africa are a direct consequence of chronic poverty and are usually manifested by symptoms and sings that are included in the Bangui definition of AIDS, such as weight loss, chronic

diarrhea, prolonged fever, persistent cough, generalized pruritus. Even worse: "the presence of generalized Kaposi's sarcoma, cryptococcal meningitis are sufficient, by themselves, for the diagnosis of AIDS" in Africa (Quinn et al 1986).

In the United States, a positive result on "the AIDS test" - ELISA and Western blot antibody tests - is indicative of HIV infection and predictive of AIDS (Feimberg & Volberding & Cohen 1994; Pins et al 1997; Metcalf et al 1997; Weiss 1998; Holodny & Busch 2003). Also in the United States a diagnosis of HIV-positivity can be done only after the same blood of a person has reacted positive four times in the ELISA test on two consecutive days and one time in the Western blot test. If AIDS is an infectious disease, it would be the very first infectious disease that requires the repetition of the same antibody test four times in order to know if those antibodies are present or not. If the ELISA test was as specific for HIV as claimed, why is it that this test needs to be repeated four times on the same blood specimen before declaring a positive HIV result? This does not happen with any other well-known infectious disease!

The antibody tests are neither standardized nor reproducible, with respect to HIV. They are, by themselves, meaningless because they mean different things in different individuals, in different laboratories and in different countries (Papadopulos-Eleopulos et al 1993). They are interpreted differently in the United States, Russia, Canada, Australia, Africa, Europe and South America (CDC 1989; Zolla-Pazner et al 1989; De Cock et al 1991; Voevodin 1992; Maskill & Gutz 1992), which means that a person who is positive in Africa can be negative when tested in Australia; or a person who is negative in Canada can become positive when tested in Africa (Continuum 1995). More embarrassingly, when the same sample of blood was tested on the Western blot test in 19 different laboratories, 19 different results were obtained (Lundberg 1988).

Nor are results from the "HIV Viral Load test" reproducible. This can be seen in the wide range of variability that is accepted in the quality controls set by the companies making and commercializing the test. For example, Roche accepts low control having a range between 630 and 10,000 copies per ml [Lot # G05467], and high control having a range between 80,000 and 720,000 copies per ml [Lot # G05466] [Roche, Amplicor HIV-1 Monitor test Lot # G13330, expiration October 2006]. Most important of all, the problems with the lack of a gold standard for "HIV infection" also apply to the evaluation of the specificity of the PCR or Viral Load test (Papadopulos-Eleopulos et al 1993; Johnson 1996c; Philpott & Johnson 1996; Giraldo 2000a). As a consequence, the specificity of the Viral Load test for HIV has never been defined properly and, therefore, "Viral Load" positive results are likely to be false-positives for HIV.

The fact that the defenders of the "HIV is the cause of AIDS" hypothesis had to use genetic amplification – the PCR test – is a strong argument against HIV as the cause of AIDS. To have to amplify tiny amounts of genetic material in the blood of AIDS patients in order to identify HIV, instead of culturing the entire virus and isolating it, violates one of the central rules of infectious diseases, because in the severity climax of any real infectious disease the patient has the highest amount of microbes in his/her tissues. It is at that time, therefore, that it should have been easy to isolate the microbes without having to use PCR genetic amplification.

Interestingly, there are many HIV researchers who are now questioning the identical issues that we (AIDS dissidents) have been critiquing for more than two decades: Where is the scientific proof that AIDS can be sexually transmitted and that it can also be transmitted from mothers to babies during pregnancy, delivery and breastfeeding? (Gisselquist et al 2002; Brewer et al 2003; Gisselquist & Potter 2004).

On the other hand, all of the medical conditions listed in the previous section and that cause false-positive results on "tests for HIV" are characterized by states of inflammation with the subsequent chronic stimulation/activation of the immune system. They are also characterized

by having high levels of immunoglobulins (antibodies) in the blood, as well as high levels of oxidative stress.

Similarly, individuals "at risk for AIDS" and who react positively on "HIV tests" are also characterized as having high levels of antibodies, chronic stimulation/activation of their immune systems (Papadopulos-Eleopulos et al 1993, 1997a,b; Shallengerger 1998; Giraldo et al 1999; Giraldo 1997b, 2000a), as well as high levels of free radicals, specially oxidizing species (Dworkin et al 1986; Fabris et al 1988; Papadopulos-Eleopulos 1988; Turner 1990; Giraldo 1997a,b,c, 2000a; Shallenberger 1998; Giraldo et al 1999).

Moreover, a prerequisite for a person to turn his/her "HIV-negative" status into "HIV-positive" is to have low levels of antioxidants in the blood, such as vitamins A, C and E, zinc and selenium (Moore et al 1993; Mehendale et al 2001; McDonald et al 2001; Giraldo 2003b). Also, antioxidant vitamins have been found to avoid the progression of "HIV-positive" individuals into the clinical manifestations of AIDS (Fawzi & Hunter 1998; Fawzi et al 2004; McNeil 2004). Moreover, "HIV-positive" mothers who have a normal blood level of vitamin A and zinc seem to deliver "HIV-negative" babies (Fawzi & Hunter 1998; Fawzi et al 2004).

High levels of antibodies, present in "HIV-positive" individuals, are regarded as resulting from exposure to significant quantities of recreational drugs, semen, sexual lubricants, factor VIII, blood and blood components, sexually transmitted infections, other infections, mental distress, as well as to parasites, malnutrition, lack of clean water, and other unsanitary conditions (Papadopulos-Eleopulos et al 1993, 1997a,b; Shallengerger 1998; Giraldo et al 1997b,c). All these cause oxidative stress (Papadopulos-Eleopulos 1988; Turner 1990; Papadopulos-Eleopulos et al 1993, 1997a,b; Giraldo 1997b,c; Shallengerger 1998; Giraldo 2000b; Giraldo et al 1999). Some defenders of the HIV dogma call these oxidizing agents "cofactors". However, multiple and chronic exposures to a variety of these factors represent, by themselves, potential causes of AIDS (Giraldo 1997b, 2000a,b). As a result of chronic exposures to these factors, immune systems are chronically stimulated, with the subsequent production of polyspecific antibodies readily detected, non-specifically, on the ELISA and Western blots tests.

Biochemically speaking, the body responds, non-specifically, to exposures to cocaine, sex lubricants, malnutrition, electromagnetic fields or to unclean water and parasites. The non specificity of these "stresses" was discovered by Hans Selye in the middle of the last century (Selye 1936, 1946; 1982).

The serologic tests for HIV (ELISA and Western blot) may react positively in the presence of poly-specific antibodies. Positive result on antibody tests for HIV could, therefore, result from chronic antigenic stimulation, rather than from a hypothetical infection with an exogenous retrovirus such as HIV (Giraldo 1997a-e, 2000b; Giraldo et al 1999). Chronic antigenic stimulation of the immune system may be the consequence of multiple, repeated, and chronic exposures to immunological stressor agents (Snyder & Fleissner 1980; Barbacid et al 1980; Wing 1995). Similarly, positive results on PCR tests for HIV can result from the presence of fragments of genetic material in the blood of individuals exposed to a variety of stressor agents (Urnovitz et al 1999; Giraldo et al 1999). Therefore, the reactivity on the three main tests for HIV (ELISA, Western blot, and PCR or Viral Load) could simply result from multiple responses to a variety of chemical, physical, biological, mental and nutritional stress (Giraldo 1997a-e; Giraldo 2000b, 2002). Additionally, the degree of reactivity on "HIV tests" may be proportional to the level of exposures to immunological stressor or oxidizing agents.

In this regard, the HIV phenomenon has been plausibly explained as a response of cells to different types of stress: "Human Immunodeficiency Virus type-1 (HIV-1) replication and proviral gene expression are exquisitely responsive to factors that induce cellular stress" (Bate et al 2000).

Interestingly, Giraldo had the opportunity to demonstrate that all human blood samples react positively on the ELISA test when the tests are run with non-diluted serum. This indicates that all individuals have antibodies against what is supposed to be HIV. The individuals that react positively with straight serum would have a smaller quantity of antibodies than those reacting positively when the serum is diluted 400 times (Giraldo 1998/9). This observation has been confirmed by Yugoslavian and Italian researchers (Metlas et al 1999).

Along the same lines, no one is HIV viral load negative. All samples of human blood, tested by PCR Viral Load, always demonstrate the presence of copies of "HIV RNA." The standard protocol for HIV Viral Load declares a blood sample negative if less than 400 copies of HIV RNA are found. Similarly, the ultrasensitive protocol for HIV viral load declares a blood sample negative if less than 50 copies of HIV RNA are found (Roche 2003). No single human being is, therefore, entirely free of copies of "HIV RNA" in his/her blood. We all are "HIV Viral Load" positive to some degree. Whether this is due to minimal expression of endogenous retroviruses or to universal exposures to stressor agents remains to be analyzed.

In addition, exposure to immunological stressors or oxidizing agents is the cause of mild to moderate levels of immune suppression present in many non-symptomatic individuals who react positively on the "tests for HIV." If exposure to immunological stressors is not stopped, or if the individual is not detoxified, the health status of these individuals will frequently worsen, their immune systems eventually collapsing with the subsequent development of the clinical manifestations of AIDS. The immune system has three main functions: (a) defense against intruders, (b) surveillance of the growth of some tumors, and (c) homeostasis or balance of all body organs and systems. With the collapse of these three functions, opportunistic infections, opportunistic tumors, and opportunistic metabolic diseases may develop. As a matter of fact, this is AIDS. AIDS, rather than being an infectious/viral disease, appears to be a toxic and nutritional syndrome (Giraldo 1997a-e; 2002).

The successful nutritional and antioxidant therapies in the prevention and treatment of AIDS (Giraldo 2003a,b) can now be better understood.

On the other hand, if "the AIDS test" (ELISA and Western blot) were in fact detecting antibodies to HIV, it would not be logical to conclude that these antibodies indicate an active infectious process. The presence of antibodies to any virus simply means humoral immune response to that virus, and not necessarily that the virus is still active and pathogenic (Evans 1989; Zinkernagel 1993; Mims et al 1995). In most instances, antibodies against viruses indicate immunity. This is the very basis of vaccination against viral diseases. Even if ELISA and Western blot tests were specific for antibodies against HIV, the question would remain to find out why, in the case of AIDS, the presence of antibodies indicates disease, rather than protection against the incriminated microorganism?

There is no justification for the fact that patients as well as the general public have never been made aware of all of the preceding facts, a scientific dereliction resulting from widespread censorship. Without a clear awareness of the considerable uncertainties concerning the so-called tests for HIV, people cannot make informed decisions. Individuals should have the capacity to make informed choices (Ken et al 1996; O'Mara 1998; Silverman 1998). However, the possibility to express informed choice implies easy access to verifiable information. There is no justification for the fact that most people have not been informed about the serious inaccuracy of the tests for HIV. Withholding or obscuring these facts is a serious breach of public trust, violating as it does people's capacity to express valid informed consents that are essential in all decision making concerning their health care.

Fortunately, Celia Farber's article in the March 2006 issue of Harper's magazine (Farber 2006) is an example of a high level of professional journalism that gives us the hope that the era of inexcusable censorship on all matters related to HIV/AIDS is finally over.

8. Experiments proposed during the South African Presidential AIDS Advisory Panel.

In year 2000, during the South African Presidential AIDS Advisory Panel meetings (first in Pretoria, then in Johannesburg), the so-called "AIDS dissidents" proposed nine experiments. The goal of some of them was to determine, once and for all, whether HIV could be isolated and purified according to classical virological methods, and what is the real significance of testing positively on "tests for HIV." However, due to the strong censorship and pressure from the HIV establishment, these experiments have not been carried out yet.

De Harven proposed to attempt isolation of HIV, following classical techniques for isolating and purifying retroviruses (O'Connor et al 1964; de Harven 1965a,b,1974). For that purpose he proposed to take blood from AIDS patients with very high results in the "HIV Viral Load test" and who accordingly should have large numbers of circulating HIV particles (viremia). (www.polity.org.za/govdocs/reports/aids/aidspanel).

Giraldo proposed studying the uncertain significance of "positive" HIV tests, comparing 6 different groups of people and performing ELISA, Western blot, Viral Load, together with complete hematological and chemical profiles as a means to evaluate their general health, as well as evaluating their immunological, nutritional, and oxidative status. Groups to be studied were: (a) A group of healthy individuals from different ages; (b) A group of patients with chronic clinical conditions unrelated to AIDS; (c) A group of non-symptomatic individuals from the conventional AIDS risk groups who react negatively on "HIV tests;" (d) A group of non-symptomatic individuals from the conventional AIDS risk groups who react positively on "HIV tests;" (e) A group of patients with clinical manifestations of AIDS who react positively on "HIV tests;" (f) A group of patients with clinical manifestations of AIDS who react negatively on "HIV tests"
(www.polity.org.za/govdocs/reports/aids/aidspanel).

The result of such an experiment could determine whether the so-called HIV tests bear any relationship to an individual's level of exposure to stressor or oxidizing agents. If so, the tests could possibly be used as a measure of an individual's level of oxidative stress.

9. Conclusions and Recommendations

9.1. Particles closely resembling retroviruses demonstrated by electron microscopy in the classical paper concerning "HIV isolation" (Barre-Sinoussi et al 1983; Papovic et al 1984; Levy et al 1984) were not demonstrated as originating from "pre-AIDS" nor from AIDS patients. They could, most likely, originate from lymphocytes that were mixed in these complex cell cultures, i.e. cord blood lymphocytes.

9.2. "HIV reverse transcriptase" described in classical papers on "HIV isolation" (Barre-Sinoussi et al 1983; Papovic et al 1984; Levy et al 1984) is not a specific marker of HIV, since that enzyme is present in all living cells and could, therefore originate from the cell debris contaminating the alleged viral samples.

9.3. The specificity of the retroviral origin of so-called "HIV-proteins" described in the classical paper (Barre-Sinoussi et al 1983; Papovic et al 1984; Levy et al 1984) could have been demonstrated only after successful purification of HIV. As acknowledged by Luc Montagnier, HIV has not been purified (Papadopulos-Eleopulos et al 1997/98) and the "HIV-proteins" cannot, therefore, be used as reliable markers of HIV.

9.4. "Sequencing of HIV-nucleic acid" is not a specific marker of HIV either, for the same reason, i.e., the lack of any successful purification of the virus.

9.5. In 1997, Glushankoff's group in Europe, and Bess's group in the United States (Glushankoff et al 1997; Bess et al 1997), were not able to isolate nor to purify HIV from cell cultures regarded as active producers.

The word "isolation" as used by the most noted researchers (Barre-Sinoussi et al 1983; Gallo et al 1984; Levy et al 1984) can be very misleading, as has been pointed out many times (Papadopulos-Eleopulos 1988; Papadopulos-Eleopulos et al 1993, 1996, 1997a,b; Turner 1996, 1997/1998, 1998; de Harven 1998, 2003; Giraldo 2000a; Giraldo et al 1999).

9.6. Retroviral particles have never been either isolated or purified directly from any individual AIDS patient. Claims of successful isolation have always been made from the analysis of highly complex (and frequently contaminated) cell cultures.

9.7. Therefore, since no retrovirus has ever been clearly demonstrated to be associated with AIDS patients, the HIV/AIDS hypothesis has to be fundamentally reappraised.

9.8. If AIDS were indeed caused by a retrovirus, how can we explain that more than 25 years of considerable research efforts, based exclusively on that single hypothesis, have failed to isolate the responsible exogenous retrovirus? How can we explain that after more than twenty-five years we still have no curative treatment, no vaccine, and no verifiable epidemiological predictions? Obviously, time is pressing us to courageously ask the essential question, i.e., is the HIV=AIDS hypothesis correct? We must realize that it is possible to view AIDS differently, entirely outside the fields of infectious diseases and retrovirology.

Rather than being viral and infectious, AIDS could more likely be a toxic and nutritional disease caused by multiple, chronic and repeated exposures to immunological stressor agents, which can have a chemical, physical, biological, mental, or nutritional origin (Giraldo 1997a-d, 2000b, 2002).

Note: For further scientific facts demonstrating that "HIV tests cannot diagnose HIV infection" we recommend the careful study of the publications at the following websites:

www.rethinkaids.com
www.robertogiraldo.com
www.theperthgroup.com
www.virusmyth.net

Most unfortunately, the type of information provided in this article cannot be found in the "peer-reviewed" medical journals, due to the strong censorship exercised by the HIV orthodoxy. However, this should surprise no one, since it only reflects the profound crisis currently affecting the peer-review system (Horrobin 1990, 1996, 2001). "Peer-review is one of the sacred pillars of the scientific edifice" (Goodstein 2000). However, all indications are that: "Far from filtering out junk science, peer-review may be blocking the flow of innovation and corrupting public support of science...Those who disagree are almost always dismissed in pejorative terms such as 'maverick,' 'failure,' and 'driven by bitterness'...The peer-review processes in both academia and industry have destroyed rather than promoted innovation" (Horrobin 2001).

Furthermore: "Peer-review is also the process that controls access to funding, and here the situation becomes much more serious: Failure to pass the peer-review process might well mean that a project is never funded" (Horrobin 2001). Two decades of AIDS dissident efforts provide many examples of the systematic rejection of funding for non-HIV related AIDS research.

Interestingly, the scientific establishment, its journals, and its grant-giving bodies "consistently refuses open scrutiny" (Horrobin 2001). Rothwell and his group "have provided solid evidence

of something truly rotten at the core of science" (Rothwell et al 2000). They report: "it is not surprising that the public is increasingly skeptical about the agenda and the conclusions of science...Public support can only erode further if science does not put its house in order and begin a real attempt to develop validated processes for the distribution of publication rights, credit for completed work, and funds for new work...If science is to have any credibility — and also if it is to be successful — the peer-review process must be put on a much sounder and properly validated basis or scrapped altogether" (Rothwell et al 2000).

Let us unite with love and compassion to defend humankind from "AIDS and the corruption of medical science."

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