AIDS, UNDERDEVELOPMENT AND SEXUAL STEREOTYPES: RETHINKING AIDS IN AFRICA

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Defining AIDS in Africa

History will show that AIDS became one of the leading bio-medical research controversies in the late 20th century. Because Africa plays a major role in the dire predictions about increased AIDS incidence, it is important to distinguish between a virus (HIV) and a syndrome (AIDS) to understand how knowledge about AIDS is created and disseminated. Before international donors pour more money into African AIDS research, or conduct another knowledge-attitude-practice survey, or advocate modifying traditional sexual behavior, they should subject the basic suppositions about AIDS cases in Africa to the same standards of consistency, testability, and parsimony used in the empirical sciences.

"African AIDS" refers to a cluster of clinical symptoms (weight loss, chronic diarrhea, fever, and persistent coughs), none of which are new or uncommon on the African continent. Since 1985, however, these symptoms have been defined as a single "syndrome" and made part of an infectious disease theory that assumed they were caused by a contagious virus (HIV). It was also assumed that this virus could be easily transmitted through sexual contact.

How can a virus cause 29 heterogeneous "AIDS indicator" diseases almost entirely among males in Europe and America but afflict African men and women in equal numbers? The answer is that the World Health Organization uses a definition of AIDS in Africa that differs decisively from the one used in the West. The origins of the former definition are illuminating.

Joseph McCormick and Susan Fisher-Hoch were physicians from the U.S. Centers for Disease Control (CDC) who were instrumental in convening the WHO conference in the Central African Republic in 1985 that produced the "Bangui Definition" of AIDS in Africa. They recently explained the motivation for the conference and the rationale behind the definition that resulted from it:

"We still had an urgent need to begin to estimate the size of the AIDS problem in Africa....But we had a peculiar problem with AIDS. Few AIDS cases in Africa receive any medical care at all. No diagnostic tests, suited to widespread use, yet existed...In the absence of any of these markers [e.g., diagnostic T4/T8 white cell tests], we needed a clinical case definition...a set of guidelines a clinician could follow in order to decide whether a certain person had AIDS or not. [If we] could get everyone at the WHO meeting in Bangui to agree on a single, simple definition of what an AIDS case was in Africa, then, imperfect as the definition might be, we could actually start to count the cases, and we would all be counting roughly the same thing.[emphasis added]

The definition was reached by consensus, based mostly on the delegates' experience in treating AIDS patients. It has proven a useful tool in determining the extent of the AIDS epidemic in Africa, especially in areas where no testing is available. ***Its major components were prolonged fevers (for a month or more), weight loss of 10 percent or greater, and prolonged diarrhea..."***

The doctors wanted to refute the ugly moralism of the 1980s that AIDS was a "gay plague" by convincing the American government that "AIDS was a plague all right, but that no one was immune." McCormick and Fisher-Hoch recalled that:

"experts in STDs continued to regale us with tales of the excessive and often bizarre sexual practices associated with HIV in the West...we were also beginning to see a direct correlation between the number of sexual partners and the rate of infection...Compared to the West,

heterosexual contacts in Africa are frequent, and relatively free of social constraints - at least for the men....There was every reason to believe that, having found heterosexually transmitted AIDS in Kinshasa, we were likely to find it everywhere else in the world."

It was upon these grossly unscientific claims, inaccurate clinical generalization, western notions of social amelioration, and 19th century racist stereotypes about Africans that AIDS became a "disease by definition" and Africa was assigned a central role in the premise that AIDS was everywhere and everyone was at risk. By 1986, "people were falling over one another to get involved in AIDS research," recalled the couple. "They realized that AIDS represented an opportunity for grant money, training, and the possibility of professional advancement...A certain bandwagon mentality took hold. Careers and reputations were riding on the outcome." Since they had no proof that AIDS was sexually transmitted, McCormick and Fisher-Hoch relied on a narrow survey conducted by Kevin DeCock, another CDC epidemiologist. DeCock wanted to determine what had happened to the five Africans out of 600 who had been tested for Ebola virus in 1976 in the small town of Yambuku (northern Zaire) and whose serum, when tested retrospectively ten years later, showed antibodies to HIV. According to McCormick and Fisher-Hoch, "three of the five were dead. To determine if their deaths were attributable to AIDS, Kevin interviewed people who had known them. The friends and relatives of the deceased described an illness marked by severe weight loss and other ailments that left little doubt in Kevin's mind that they had succumbed to AIDS. [emphases added]" It is not known how many of the 595 HIV negative subjects had died or what was their clinical cause of death.

This kind of presumptive diagnosis known as a "verbal autopsy" is widely accepted in Africa, where "no country has a vital registration system that captures a sufficient number of deaths to provide meaningful death rates." While medically certified information is available for less than 30% of the estimated 51 million deaths that occur each year worldwide, **the Global Burden of Disease Study (GBD) found that sub-Saharan Africa had the greatest uncertainty for the causes of mortality and morbidity since its vital registration figures were the lowest of any region in the world - a microscopic 1.1%.**

These 1997 findings prompted The Lancet to acknowledge editorially that "current strategies to improve the world's health may need to be reassessed" and to ponder "how much more money is spent on research into HIV infection [the 30th cause of death] than into the causes of suicide [#12] or the prevention of road-traffic accidents [#9] and why should this be."

Racism and African Sexuality

The conventional thinking asserts that AIDS in Africa is somehow explicable by Africans' sexual predilections. Such insinuations merit close scrutiny since generalizations about African sexual practices are analytically useless for an internally diversified continent of 650 million people. Nonetheless, at the 10th International AIDS Conference in Yokohama (August 1994), Dr. Yuichi Shiokawa claimed that AIDS would be brought under control only if Africans restrained their sexual cravings. Professor Nathan Clumeck of the Universite Libre in Brussels was skeptical that Africans will ever do so. In an interview with Le Monde, Clumeck claimed that "sex, love, and disease do not mean the same thing to Africans as they do to West Europeans [because] the notion of guilt doesn't exist in the same way as it does in the Judeo-Christian culture of the West." Such myths about the sexual excesses of Africans are old indeed. Early European travelers returned from the continent with tales of black men performing carnal feats with unbridled athleticism, with black women who were themselves sexually insatiable. These affronts to Victorian sensibilities were cited, alongside tribal conflicts and other "uncivilized" behavior, as justification for colonial social control.

AIDS researchers added new twists to an old repertoire: stories of Zairians who rub monkeys' blood into cuts as an aphrodisiac, of ulcerated genitals, and of philandering East African truck drivers who get AIDS from prostitutes and then go home to infect their wives. **A facetious letter in The Lancet cited a passage from Lili Palmer's memoirs as evidence for how a large male chimpanzee's "anatomically unmistakable signs of its passion for [Johnny] Weismuller" on the Tarzan set in 1946 "may provide an explanation for the inter-species jump" of HIV infection.** No one has ever shown that people in Rwanda, Uganda, Zaire, and Kenya - the so-called "AIDS belt" - are more active sexually than people in Nigeria which has reported only 1148 AIDS cases out of a population of 100 million or Cameroon which reported 3072 cases in 10 million. **No continent-wide sex surveys have ever been carried out in Africa. Nevertheless, conventional researchers perpetuate racist stereotypes about insatiable sexual appetites and carnal exotica.** They assume that AIDS cases in Africa are driven by a sexual promiscuity similar to what produced - in combination with recreational drugs, sexual stimulants, venereal disease, and over-use of antibiotics - the early epidemic of immunological dysfunction among a small sub-culture of gay men in the West. The research from Africa suggests nothing of the sort. In 1991 researchers from Medicins Sans Frontieres and the Harvard School of Public Health did a survey of sexual behavior in Moyo district of northwest Uganda. **Their findings revealed behavior that was not very different from that of the West.** On average, women had their first sex at age 17, men at 19. Eighteen per cent of women and 50% of men reported premarital sex; 1.6% of the women and 4.1% of the men had casual sex in the month preceding the study, while 2% of women and 15% of men did so in the preceding year.

The media misrepresentations that link sexuality to AIDS have spawned inordinate anxieties and moral panics in regions of Africa already afflicted with extreme poverty, ravaged by war, and deprived of primary health care delivery systems. **The "disaster voyeurism" of tabloid journalism enables them to use AIDS to sell "more newspapers than any other disease in history. It is a sensational disease - with its elements of sex, blood and death it has proved irresistible to editors across the world." Public health seems to require salesmanship, not skepticism.** The media's appetite for scary scenarios and its disdain for alternative perspectives enables it to treat Africa in apocalyptic terms. This marketing of anxiety helps to promote behavior modification programs to "save Africa." Disregarding the morbidity and mortality data from the Global Burden of Disease Study, journalists maintain that "AIDS is by far the most serious threat to life in Africa."

Several serious consequences of claiming that millions of Africans are threatened by AIDS make it politically acceptable to use the continent as a laboratory for vaccine trials and the distribution of toxic drugs of disputed effectiveness like ddI and AZT. Campaigns that advocate monogamy or abstinence and ubiquitous media claims that "safe sex" is the only way to avoid AIDS inadvertently discourage Africans from visiting a public health clinic for fear of receiving some "fatal" AIDS diagnosis. And even Africans "with treatable medical conditions (such as tuberculosis) who perceive themselves as having HIV infection fail to seek medical attention because they think that they have an untreatable disease." Some Western scientists, such as Dr. Luc Montagnier, the French virologist who discovered HIV, claim that the practice of female circumcision facilitates the spread of AIDS. Yet Djibouti, Somalia, Egypt, and Sudan, where female genital mutilation is the most widespread, are among the countries with the lowest incidence of AIDS. AIDS cases in Africa are thought to constitute a "heterosexual paradox" because the sexually equal distribution contrasts sharply with the 12-1 ratio of men to women with AIDS in North America and Western Europe. Most frightening to people of the developed world is what the African example seems to augur for them. Whereas AIDS in the industrialized countries is almost exclusively a disease of a small percentage of homosexuals, intravenous drug users, and recipients of tainted blood transfusions, AIDS in Africa is said to be as general and indiscriminate a killer as such long-time African curses as malaria, schistosomiasis, and sleeping sickness (trypanosomiasis). In Africa, where women contracted "Slim Disease" in numbers roughly equal to males, there is no evidence of any correlation between being immune deficient

and having engaged in promiscuous homosexual intercourse. Intravenous drug use is uncommon among impoverished African villagers - even among city dwellers. Did this mean, deductively, that heterosexual intercourse could put anyone at risk for AIDS? Did the "AIDS epidemic" in Africa portend the future of the developed world? The scientific establishment certainly thought so. **Biomedical funds that had been earmarked to fight African malaria, tuberculosis, and leprosy were diverted into sex counseling and condom distribution, while social scientists shifted their attention to behavior modification programs.**

Good Intentions, Bad Science: HIV Tests and Disease

A reappraisal of AIDS in Africa should also recognize that HIV tests are notoriously unreliable among African populations where other endemic microbes and bacteria cross-react to produce ludicrously high false-positive results. **A 1994 study on central Africa reported that the microbes responsible for tuberculosis, malaria, and leprosy were so prevalent that they registered over 70% false HIV-positive tests.** HIV tests also register positive results in people whose immune systems are compromised for a wide variety of reasons, including chronic parasitic infections and anemia brought on by malaria.

By definition, all viruses that cause a "disease" infect over 30% of the cells they target, are present in the blood at concentrations in excess of 10,000 per milliliter, and are contagious. HIV is such a weak retrovirus that when detected at all, it is present in such low concentrations (about one per milliliter) that only its antibodies can be detected. This is the probable explanation as to why it is barely transmissible, requiring an average 1000 unprotected vaginal sex contacts with an antibody-positive person for someone to "get" HIV. Most HIV tests do not detect a virus but rather viral antibodies that are read with an assortment of proteins that are not even unique to HIV. The notion that antibodies are a prognosis of death defies all classical experience with viruses, microbes, and antibodies. An investigation reported in The Lancet was equally revealing. In Uganda, 9,389 individuals with unequivocal blood test results were enrolled in a study. After two years, 3% had died, 13% had left the area, and 84% remained. **There had been 198 deaths among the seronegative people and 89 deaths in the seropositive ones.** Medical assessments made prior to death were available for 64 of the HIV-positive adults. Of these, five (8%) had AIDS as defined by the WHO clinical case symptoms. **The self-proclaimed "largest prospective study of its kind in sub-Saharan Africa" had tested nearly 9400 people in Uganda, the so-called epicenter of AIDS in Africa. Yet of the 64 deaths recorded among those who tested positive for HIV antibodies, only five were diagnosed as AIDS-induced.** A highly touted 1995 report on the Mwanza region of Tanzania claimed that "improved STD treatment reduced HIV incidence by about 40%...[in] the first randomized trial to demonstrate an impact of a preventive intervention on HIV incidence in a general population." **This occurred despite the fact that "no change in reported sexual behavior was observed in either group."** And on close inspection of the data, one realizes how the 40% reduction was measured. Of the individuals who initially tested HIV-negative, in the intervention group 48 out of 4149 (1.2%) were HIV-positive two years later; 82 of 4400 (1.9%) in the comparison group tested HIV-positive. **The researchers arrived at the "40% reduction" figure merely by comparing the difference between 1.2% and 1.9%.** The Africans in this study had tested positive or negative for antibodies to HIV but the source of their "infection" was unknown. **While the research suggested that a regimen of antibiotics reduced the prevalence of HIV-antibodies, the investigators always assumed, without any evidence whatsoever, that it had somehow reduced the transmission.**

AIDS researchers in Africa assume that there is a correlation between clinical symptoms - weight loss, chronic diarrhea, fever, persistent coughs - and sexual activity. **Correlation - whether one phenomenon is found in tandem with another - is not causation.** Proof of causation requires that we control all variables in order to isolate one variable as a cause, not merely an associated factor. The clinical symptoms that define an AIDS case in Africa are expressed in roughly equal numbers among men and women, not because of alleged heterosexual transmission, but more

likely because the socio-economic conditions that give rise to these symptoms are caused by environmental risk factors to which many Africans are regularly exposed. A literature review in the World Journal of Microbiology and Biotechnology pinpointed the methodological flaw in the belief that AIDS is sexually transmissible:

"Since AIDS is a panoply of diseases or symptoms and signs, the minimum requirement to prove that AIDS is spread by sexual activity is to take an index case, isolate the putative agent, trace the sexual contacts of that case, and then isolate the same agent. To date, no data anywhere of this type has ever been presented either in Africa, or anywhere else.

In the whole history of medicine there has never been an example of a sexually transmitted disease which is spread unidirectionally, and certainly not one that is spread unidirectionally in one country and bidirectionally in another.

Indeed, given this and the other differences between AIDS in the West and Africa, it is necessary to postulate that HIV must possess unique features...[and] be able to distinguish the gender and country of residence of its host. The only other alternative is to agree with African physicians that positive HIV antibody tests in Africa do not mean infection with HIV and that immnuosuppression and certain symptoms and diseases which constitute
African AIDS have existed in Africa since time immemorial."

Nor is there evidence of widespread secondary or tertiary transmission of HIV/AIDS among heterosexuals in the West either. "This is an important point to consider," warns Michelle Cochrane, "because the foundation of orthodox AIDS science and epidemiology rests upon the premise that HIV/AIDS is relatively frequently transmitted from an index AIDS case (the primary individual) to a secondary AIDS case either through an exchange of semen or blood. In turn, this secondarily 'infected' individual must, per force, be capable of transmitting HIV/AIDS to a third individual (tertiary transmission) by the same means, or an infectious disease epidemic cannot be sustained." In a meticulous review, Cochrane juxtaposed the central tenets of AIDS orthodoxy against the material record of San Francisco AIDS patients' charts. **She found that public health officials persistently over-estimated the risk of contracting HIV/AIDS through sexual activity "while simultaneously under-estimating the proportion of the HIV/AIDS caseload that were attributable to intravenous drug use and/or socio-economic factors which condition access to healthcare and prevention services."** Cochrane showed that health officials conspicuously failed to investigate all risk factors for immunological dysfunction among heterosexual adult females. In their surveillance studies, it was considered sufficient for

"a heterosexual female merely to claim that the source of her infection was sex with an IV drug user or another man at risk for HIV/AIDS...A percentage of the 187 female AIDS cases [out of 24,371 cumulative cases in San Francisco] attributed to sexual transmission would, with proper investigation, be attributable to IV drug use. Epidemiological research in the United States and Europe has never proven that a female has sexually transmitted HIV to a man. [Because] heterosexual transmission of HIV from a male to a female happens with difficulty and very infrequently...all AIDS surveillance statistics on female AIDS cases have been gathered without rigorous scrutiny of the woman's risk for disease and with a bias towards including as many women as possible.[emphasis added]"

The a priori assumptions that directed AIDS surveillance activities in the United States subsequently allowed predictions about an exponential spread of the disease to survive as "common knowledge" despite the lack of empirical data. These are critical points to consider when reviewing epidemiological data on "AIDS" cases in Africa. For the period 1984-95, the WHO compared estimates of HIV seropositivity with the actual numbers of AIDS cases in its

Weekly Epidemiological Reports. **The cumulative result is that 99.95% of all Africans do not "have AIDS" and 97% of those who are presumed to be HIV-positive had not developed AIDS.**

AIDS and the Medicalization of Poverty

Primary health care systems in Africa will remain hampered until public health planners systematically gather statistics on morbidity and mortality to accurately show what causes sickness and death in specific African countries. During the past ten years, as external financing of AIDS programs in Africa dramatically increased, support for other health sectors remained static even though deaths from malaria, tuberculosis, neo-natal tetanus, respiratory diseases, and diarrhea grew at alarming rates. **The political economy of underdevelopment and environmentally caused endemic sickness pose the gravest threats to African health.** Poor harvests, rural poverty, migratory labor systems, urban crowding, ecological degradation, social mayhem, the collapse of state structures, and the sadistic violence of civil wars are the primary threats to African lives. When essential services for water, power, and transport break down, public sanitation deteriorates and the risks of cholera, dysentery, and respiratory infection increase.

WHO Director General Hiroshi Nakajima warns emphatically that "poverty is the world's deadliest disease." Indeed, the leading causes of immunodeficiency and the best predictors for clinical AIDS symptoms in Africa are impoverished living conditions, economic deprivation, and protein malnutrition, not extraordinary sexual behavior or the trace measurements of antibodies for a mysterious virus that has yet to be isolated. The so-called "AIDS epidemic" in Africa has become the medicalization of poverty to justify Western medical intervention in the form of vaccine trials, drug testing, and almost evangelistic demands for behavior modification. AIDS scientists and public health planners must recognize the role of malnutrition, poor sanitation, anemia, and parasitic and endemic infections in producing the clinical AIDS symptoms that are manifestations of non-HIV insults. Socio-economic development, not sexual restraint, is the key to improving health care systems in Africa.

Phillipe and Evelyn Krynen, medically trained charity workers employed by the French group Partage in Kagera Province of Tanzania, report that when "appropriate treatment was given to villagers who became ill with complaints such as pneumonia and fungal infections that might have contributed to an AIDS diagnosis, they usually recovered." In Kenya, a former surgeon, Father Angelo D'Agostino who founded Nyumbani, a hospice for abandoned and orphaned HIV-positive children had experiences that corroborated those of the Krynens:

"People think a positive test means no hope, so the children are relegated to the back wards of hospitals which have no resources and they die. *They are very sick when they come to us. Usually they are depressed, withdrawn, and silent....But as a result of their care here, they put on weight, recover from their infections, and thrive. Hygiene is excellent [and] nutrition is very good; they get vitamin supplements, cod liver oil, greens every day, plenty of protein. They are really flourishing."*

Conclusion

People can be encouraged to behave thoughtfully in their sexual lives if they are provided with reliable information about condom use, contraception, family planning and venereal diseases. Multilateral institutions and African AIDS educators should familiarize themselves with the literature that demonstrates the contradictions, anomalies, and inconsistencies in the HIV/AIDS orthodoxy. They have a major responsibility to consider the non-contagious explanations for "AIDS" cases in Africa and to stop the proliferation of terrifying misinformation that equates sexuality with death.

References:

1) Charles F. Gilks, "What Use is a Clinical Case Definition for AIDS in Africa?" British Medical Journal, Vol. 303 (November 9, 1991), pp. 1189-90.

2) Recent research among African populations suggests that a person with an over-active immune system that is constantly assaulted by various pathogens or burdened with chronic infections is more susceptible to a positive HIV antibody test result. Zvi Bentwich, et. al., "Immune Activation is a Dominant Factor in the Pathogenesis of African AIDS," Immunology Today, Vol. 16, #4 (1995), pp. 187-91.

3) Joseph B. McCormick and Susan Fisher-Hoch, Level 4: Virus Hunters of the CDC (Atlanta: Turner Publishing, 1996), pp. 188-90.

4) Ibid., p. 176.

5) Ibid., pp. 173-74.

6) Ibid., pp. 179-80.

7) Ibid., p. 193.

8) Henry M. Kitange, et. al., "Outlook for Survivors of Childhood in Sub-Saharan Africa: Adult Mortality in Tanzania," British Medical Journal, Vol. 312 (January 27, 1997), pp. 216-17. The authors report that "a network of people was established in each of the [Tanzanian] study areas whose responsibility it was to inform a field supervisor of all deaths occurring in their areas. Locally known and respected people were selected...when a death was reported, the field supervisor in that area visited the home of the deceased and carried out a 'verbal autopsy.' This entailed interviewing the family by using a standard proforma with the aim of determining the cause of death."

9) Christopher Murray and Alan Lopez, "Mortality by Cause for Eight Regions of the World: Global Burden of Disease Study," The Lancet, Vol. 349 (May 3, 1997), pp. 1269-1276. In a prudent understatement, the authors advise that "the system of collecting cause of death data via 'verbal autopsies' needs to be assessed and improved to provide reliable data on broad categories of causes of death at low cost."

10) "From What Will We Die in 2020?" The Lancet, Vol. 349 (May 3, 1997), p. 1263.

11) Jean-Yves Nau, "AIDS Epidemic Far Worse Than Expected," Le Monde section in Manchester Guardian Weekly (December 14, 1993)

12) For an example of anecdotes and impressionistic tales disguised as "facts" about East African truck drivers and AIDS, see Ted Conover, "Trucking Through the AIDS Belt," The New Yorker (August 16, 1993).

13) Raul Sebastian, "Did AIDS Start in the Jungle?" The Lancet, Vol. 348 (November 16, 1996), p. 1392.

14) World Health Organization, Weekly Epidemiological Record, Vol. 69, #26 (July 1, 1994), p. 189.

15) In a review of Sexual Ecology: AIDS and the Destiny of Gay Men by Gabriel Rotello (New York: Dutton, 1997) and Life Outside: The Signorile Report on Gay Men by Michelangelo Signorile (New York: HarperCollins, 1997), Professor Daniel Kevles notes that with the advent of gay liberation, "bathhouses, while offering a communitarian haven from homophobia, also institutionalized part of the liberation movement, providing sexual opportunities in private cubicles, showers, hallways, and dimly lit 'orgy rooms' devoted to anonymous encounters...Tens of thousands were habitues of the 'circuit' - a series of large gay dance parties held in different places where they used one kind of drug to heighten their sexual energies and another to relax their sphincter muscles." Daniel J. Kevles, "A Culture of Risk," New York Times Book Review (May 25, 1997), p. 8. Signorile claims that the "circuit" was "stoked by substance abuse that would make the ghosts of Studio 54 blush." John Lauritsen and Dr. Joseph Sonnabend have described the unhealthy lifestyle of this very specific cohort of urban gay men in the United States who had unprecedented opportunities for sexual contacts with hundreds, even thousands of partners. It was a ghettoized sub-culture of "fast track" gay men who habitually abused alcohol and drugs that produced the epidemic levels of chronic infection from repeated exposure to a wide range of microbes such asgonorrhea, cytomegalovirus, hepatitis, syphilis, non-specific viral infections, bacterial pathogens, and parasitic infections. Without addressing the underlying socio-economic and environmental causes, the commitment of researchers to lump together the diverse cases of immune-deficiency that began appearing in this sub-culture led them uncritically to accept the unifying hypothesis of a single viral cause based on the similarities of the disease manifestations. See Joseph Sonnabend, "Fact and Speculation About the Cause of AIDS," AIDS Forum, Vol. 2, #1 (May 1989), pp. 2-12; and John Lauritsen, The AIDS War (New York: Asklepios Press, 1993).

16) Doris Schopper, Serge Doussantousse, and John Orav, "Sexual Behaviors Relevant to HIV Transmission in a Rural African Population," Social Science and Medicine, Vol. 37, #3 (August 1993), pp. 401-12.

17) James Deane, "The Role of the Media in the Fight Against AIDS," SIDAfrique, #8/9 (1996), p. 29.

18) "No End of Plagues," The Economist (September 7, 1996), p. 38.

19) For instance, a 31-year old man in Kagera Province (Tanzania) was said to be dying of AIDS. Emaciated and despondent, he worked as fisherman until he became sick in 1992 with diarrhea, chest pains, muscle weakness, and a severe cough. The man stayed with an aunt because his brother and sister refused to see him. "Since I became sick," he told a reporter, "I have not made an effort to go to the hospital because I have no money and my aunt is not able to pay." Susan Okie, "Tanzania Village Devastated by AIDS Deaths," Washington Post (March 15, 1992)

20) "False-Positive Self-Reports of HIV Infection," letter from Chifumbe Chintu, et. al., The Lancet, Vol. 349 (March 1, 1997), p. 649.

21) Thomas Bass, Reinventing the Future: Conversations with the World's Leading Scientists (Reading, Massachusetts: Addison-Wesley, 1994), p. 40.

22) Oscar Kashala, et. al. "Infection with HIV-1 and Human T Cell Lymphotropic Viruses Among Leprosy Patients and Contacts...," Journal of Infectious Diseases, Vol. 169, (February 1994), pp. 296-304.

23) Eleni Papadopulos-Eleopulos, et. al., "Is A Positive Western Blot Proof of HIV Infection?" Bio/Technology, Vol 11 (June 1993), pp. 696-707 explains why there is no correlation between a positive HIV antibody test result and the isolation of HIV itself. The authors conclude that "the use

of HIV antibody tests as predictive, diagnostic and epidemiological tools for HIV infection needs to be carefully reappraised." See also, Eleni Papadopulos-Eleopulos et. al., "The Isolation of HIV: Has It Really Been Achieved?" Continuum, Vol. 4, #3 (September/October 1996). Another recent study reports that even if HIV-1 is detected in the blood or cervical secretions of an HIV-seropositive woman, "the amount of HIV-1 excreted in the cervicovaginal fluid is independent of the quantity of virus present in the blood cells or plasma." Suraiya Rasheed, et. al., "Presence of Cell-Free Human Immunodeficiency Virus in Cervicovaginal Secretions is Independent of Viral Load in the Blood of Human Immunodeficiency Virus-Infected Woman," American Journal of Obstetrics and Gynecology, Vol. 175, #1 (July 1996), p. 123. Richard Strohman, Professor Emeritus of Molecular Biology at University of California (Berkeley), points out that "HIV science has always been based not on detection of real infectious units (real virus) growing under some reasonable standard condition in living cells in the lab. Rather it is based upon a high tech series of assays constructed so that disappearingly small quantities of the virus, or some part of the virus, or some trace (aura) of viral presence may be measured. We have substituted the measurement for the real thing, like substituting the menu for the meal." (E-mail message, July 7,1997)

24) Daan W. Mulder, et. al., "Two-Year HIV-1-associated Mortality in a Ugandan Rural Population," The Lancet, Vol. 343 (April 23, 1994), pp. 1021-23.

25) Heiner Gosskurth, et. al. "Impact of Improved Treatment of Sexually Transmitted Diseases on HIV Infection in Rural Tanzania: Randomized Controlled Trial," The Lancet, Vol. 346, (August 26, 1995), pp. 530-36. The a priori assumptions of the research team are evident in an exchange with Richard Hayes (London School of Hygiene and Tropical Medicine), the corresponding author for the research group. On October 14, 1996, I sent a series of nine questions to Hayes to clarify the group's findings. Hayes' responses on March 14, 1997 are indented after each question below:

a) Among the twelve village health centers on or near Lake Victoria where "annual HIV incidence" was 1%, what techniques did researchers use to distinguish between the incidence or prevalence of HIV and the transmission of HIV? What method was used to determine that HIV was actually "spreading" or that the incidence of new cases had decreased?

1. "We measured the incidence of HIV infection by following up a random sample of adult residents over two years. The annual incidence is the proportion of seronegative subjects who seroconvert, divided by two (because of the two-year follow-up period). In the 'comparison communities' (which did not receive the improved STD services), 1.9% seroconverted over two years, giving an annual incidence of about 1% as stated. In the 'intervention communities' (which did receive the improved services) only 1.2% sero-converted, so the incidence of new infections was about 40% lower, presumably as a result of the intervention."

b) The survey suggests that improving the STD case-management brought about a 42% reduction of HIV incidence. What percentage of the patients who were initially diagnosed with a sexually transmitted disease also initially registered positive for HIV-antibodies?

2. "I do not understand this question. What do you mean by 'the patients initially diagnosed with an STD"? If you mean those presenting for STD treatment at the health units, HIV testing was not carried out on these patients. The point of the study was to make available the improved STD services for everyone living in the intervention communities (regardless of HIV status). We then measured the HIV impact by testing a random cohort of individuals at baseline and follow-up. These HIV tests were carried out in a population-based survey, not through the health units where the STD treatment was provided."

c) The intervention group received a proper supply of antibiotics to treat bacterial STDs and were actively encouraged towards health care-seeking behavior. Wouldn't that suggest that people who receive better health care are less likely to register HIV+?

3. "Of course the whole point of our trial was that improved health care (specifically improved STD treatment) reduced HIV incidence in the general population. However, I am not sure what you mean by 'less likely to register HIV+'? Do you mean that the antibiotics given to STD patients would interfere in some way with the HIV serological tests? There is no evidence of any such effect, as far as I am aware."

d) There was no discernible difference in the reported sexual behavior or frequency of condom use in the intervention and control communities. While the intervention of drug therapies may have played a role in reducing HIV seroprevalence, what would that necessarily suggest about HIV transmission?

4. "Transmission implies the occurrence of new cases as the virus is spread from one individual to the next. This is measured as the 'incidence' of new infections, as explained above, and our results showed a clear effect of the intervention on incidence. We assume the explanation for this is that it is much easier for the HIV virus to be transmitted from one sexual partner to the other if one of them has another STD (this is the so-called STD Cofactor Effect). By treating STDs promptly and effectively, you should be able to reduce their duration and hence prevalence, so that it becomes much more difficult for the HIV virus to be transmitted." [see footnotes #13 and #14 above]

e) What did the research team identify as the "measured risk factors for HIV infection"? Which of those risk factors were significantly reduced?

5. "Question not understood. What part of the paper does this refer to?"

f) The baseline HIV prevalences were 3.8% for the intervention group and 4.4% for the comparison group. The prevalence of active syphilis was 8.7% and 8.3% respectively and The Lancet article states that the "treatment regimens would be expected to achieve cure in over 90% of cases of sexually transmitted diseases...Over the two years of follow-up there were 48 seroconversions (1.2%) in the intervention group and 82 (1.9%) in the comparison group... HIV incidence...was consistently lower in the intervention community..." Did fewer people register HIV-positive antibody results because they received antibiotics to treat their STDs, quite apart from any changes in sexual behavior?

6. "See 3. above. You are correct that the effect on HIV is attributed to the improved treatment of STDs, and not to any change in sexual behavior. In fact no change in sexual behavior was seen."

g) The Lancet report states that the difference between 1.2% and 1.9% is the "overall reduction in HIV incidence of about 42% over two years of follow-up...[and]...the most plausible explanation for our results is that the STD treatment programme reduced HIV incidence by shortening the average duration of the STDs, thus effectively reducing the probability of HIV transmission" even though actual sexual behavior involving condom usage did not change at all. Did the researchers report the incidence of new STDs in either group two years later?

7. "We only surveyed our cohort at baseline and at follow-up two years later. You would need much more intensive follow-up to accurately measure STD incidence during this period. However, we were able to measure the prevalence of STDs at follow-up, and could demonstrate a significant effect of the intervention on active syphilis and on symptomatic male urethritis. These

findings are consistent with the explanation given in 4. above. A paper setting out the STD data in more detail is about to be submitted for publication."

h) Although the report states that the STD intervention program "evidently had a substantial effect on HIV incidence in this rural population," since there was no noticeable change in "risk behavior" among the intervention communities, why wouldn't researchers conclude that improving the health care makes people healthier even in the absence of demonstrable changes in the sexual behavior practices thought to facilitate HIV transmission?

8. "That is exactly what we did conclude. Improved health care (specifically improved STD treatment services) reduced HIV transmission without any change in the sexual behavior of the population." [Actually the report showed that it putatively reduced the incidence or prevalence, not transmission.]

i) Please elaborate on the "cofactor effects" mentioned in the conclusion: "the impact of improved STD treatment depends on the proportion of HIV infections in the general population that are attributable to the cofactor effects of STDs..."

9. "This relates to the 'STD Cofactor Effect' mentioned above (see 4.). Obviously, in a population where STDs are present at a very low level, few HIV infections would be attributable to the enhancing effect of STDs, and so removing STDs from the population would have little effect on the HIV epidemic. Conversely, in parts of the world where STD prevalences are very high, it is possible that a large proportion of HIV infections could be avoided by removing or reducing the prevalence of STDs through improved treatment, and this is what our results suggest in Mwanza."

26) For a small sample of articles that uncritically apply the contagious HIV/AIDS theory to Africa, see: John C. Caldwell and Pat Caldwell, "The African AIDS Epidemic," Scientific American (March 1996), pp. 62-68; Simon Gregson, "Will HIV become a Major Determinant of Fertility in Sub-Saharan Africa?" Journal of Development Studies, Vol. 30, #3 (April 1994), pp. 650-79; and Kelly Lee and Anthony B. Zwi, "A Global Political Economy Approach to AIDS: Ideology, Interests and Implications," New Political Economy, Vol. 1, #3 (1996), pp. 355-73.

27) Eleni Papadopulos-Eleopulos, Valendar Turner, John Papadimitrou and Harvey Bialy, "AIDS in Africa: Distinguishing Fact from Fiction," World Journal of Microbiology and Biotechnology, Vol. 11 (March 1995), pp. 141-42.

28) Michelle Cochrane, "The Social Construction of Knowledge on HIV and AIDS: With a Case Study on the History and Practices of AIDS Surveillance Activities in San Francisco," Ph.D. dissertation, Department of Geography, University of California, Berkeley, April 1997, p. 253. Cochrane's dissertation is a case study of the emergence of AIDS and the creation of abureaucracy for AIDS surveillance in San Francisco. Orthodox epidemiological and surveillance knowledge on AIDS in San Francisco played a key role in the construction of a global consensus on AIDS historiography and science. According to Cochrane, this knowledge displays a remarkable coherence and internal consistency that is marshaled to refute any critique of ist assumptions about the etiology, epidemiology, and history of AIDS. The AIDS Sero-epidemiology and Surveillance Branch in San Francisco constitutes the greatest repository in the world for primary documentation on AIDS. It includes the medical charts and case files for every one of the 24,371 AIDS patients cumulatively reported since 1981 in the city. Cochrane demonstrates how the vested interests of institutions, organizations, and individuals perpetuated the orthodox consensus that HIV causes AIDS, "a conclusion which persists despite the presence of multiple lacunae or anomalies that the theory has not resolved." (pp. 322-24)

29) Cochrane, op. cit., p. 7.

30) Ibid., pp. 259-60.

31) For instance, even though South Africa reported only 1,120 AIDS cases in 1995 but 90,292 cases of tuberculosis in 1994, AIDS was accorded a much higher national profile and larger budget so that it now dominates clinical practice across all medical fields ranging from pediatrics to neurology. World Health Report 1996, p. 130; "South Africa: Country Profile," The Lancet, Vol. 349 (May 24, 1997), p. 1542.

32) World Health Organization, Bridging the Gaps: The World Health Report 1995 (Geneva: WHO, 1995), Table 5 (p. 18) and Table A3 (p. 110); and World Health Organization, Fighting Disease, Fostering Development: The World Health Report 1996 (Geneva: WHO, 1996), Table 4 (p. 24) and Table A3 (p. 127).

33) WHO, The World Health Report 1995, v.

34) This is further elaborated in Charles Geshekter, "Outbreak? AIDS, Africa, and the Medicalization of Poverty," Transition, #67 (Fall 1995), pp. 4-14; and Cindy Patton, Inventing AIDS (New York: Routledge, 1990), especially Chapter 4, "Inventing African AIDS."

35) Cited in Neville Hodgkinson, "Cry, Beloved Country: How Africa Became the Victim of a Non-existent Epidemic of HIV/AIDS," in P. H. Duesberg (ed.), AIDS: Virus- or Drug-Induced? (Amsterdam: Kluwer Publishers, 1996), p. 353.

36) Hodgkinson, op. cit.. pp. 350-51.

37) Recent critical studies include: Richard and Rosalind Chirimuuta, AIDS, Africa and Racism (London: Free Association Books, 1989); Neville Hodgkinson, AIDS: The Failure of Contemporary Science (London: 4th Press, 1996); Elinor Burkett, The Gravest Show on Earth (Boston: Houghton Mifflin, 1995); Hiram Caton, The AIDS Mirage (Sydney: University of New South Wales Press, 1994); Robert Root-Bernstein, Rethinking AIDS: The Tragic Cost of Premature Consensus (New York: Free Press, 1993); and Peter Duesberg, Infectious AIDS:Have We Been Misled? (Berkeley: North Atlantic Books, 1996).

For an expose of the CDC's deceitful scare campaign in the United States, see Amanda Bennett and Anita Sharpe, "AIDS Fight is Skewed by Federal Campaign Exaggerating Risks," Wall Street Journal (May 1, 1996) and David R. Boldt "Aiding AIDS: The Story of a Media Virus," Forbes Media Critic (Fall 1996). The CDC believed that exaggerating the risks to the entire American population was the only way to drum up widespread support for measures and funding to combat AIDS. Thus, the theme of its public service ad campaign launched in 1987 was, "If I can get AIDS, anyone can." But from 1990 to 1992, the proportion of heterosexuals (aged 18-49) in high risk American cities who reported multiple sexual partners increased from 15% to 19%, while condom sales decreased by 1%, and 65% of respondents admitted they used condoms either sporadically or not all. Americans are not practicing safe sex and for this reason teen pregnancies and real venereal diseases are on the rise. Yet "AIDS" cases continue to decrease sharply and even the fraction of Americans that is assumed to be HIV-antibody positive has declined from an estimated 1 million in 1985 to 700,000 in 1996. See, Joseph A. Catania, et. al., "Risk Factors for HIV and Other Sexually Transmitted Diseases and Prevention Practices Among U.S. Heterosexual Adults: Changes from 1990 to 1992," American Journal of Public Health, Vol. 85, #11 (November 1995), pp. 1492-99.

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