REVIEW ARTICLE

Demographic Characteristics of HIV:
I. How Did HIV Spread?

Henry H. Bauer

Dean Emeritus of Arts & Sciences
Professor Emeritus of Chemistry & Science Studies
Virginia Polytechnic Institute & State University
www.henry-bauer.homestead.com
e-mail: hhbauer@vt.edu

Abstract—"AIDS" was first noted around 1980 in New York, Los Angeles, and San Francisco, among gay men and drug addicts. In 1984 came discovery of human immunodeficiency virus (HIV), presumed to be the sexually transmitted cause of AIDS. Given an apparent latent period of about 10 years, HIV is thought to have arrived in those communities around 1970. Since 1985, tests for HIV have been widely deployed to detect its anticipated spread into other social groups and geographic locales.

HIV-positive people were found in every sector of society and in every part of the United States as soon as testing began. That HIV could have spread so rapidly into the general population across the country from those cities within 15 years is infeasible for several reasons:

(1) the geographic distribution of HIV does not look like a spread from the AIDS epicenters;
(2) that geographic distribution has not changed in two decades;
(3) if anything, the prevalence of HIV has decreased since the mid-1980s;
(4) direct studies have revealed that HIV is not readily transmissible.

The conclusion seems inescapable: HIV tests do not track a virus that spread from the original centers of the AIDS epidemic. HIV is endemic. It is not the cause of the AIDS epidemics of the early 1980s.

Keywords: HIV geography—HIV spread—HIV tests—HIV/AIDS—AIDS

Since 1984, it has been the conventional wisdom that human immunodeficiency virus (HIV) causes AIDS[1]. "HIV tests" have been widely deployed to detect a possible spread of the virus. These tests are for substances taken as characteristic of ***antibodies*** to HIV. It is ***inferred*** that a positive HIV-test reflects actual infection by HIV[2]. The term "F(HIV)" will often be used in this article as an alternative to the longer and commonly used phrase, "prevalence of HIV".

Some groups—principally blood donors and military personnel—have been subject to mandatory testing for two decades, even though presumed to be at low risk for AIDS, because the consequences of possible infection were to be avoided at all costs. Job Corps entrants have also been tested routinely. Less comprehensive surveillance surveys have been made of applicants for marriage licenses, hospital patients, and university students, among others. Testing has been available at a variety of public sites: family planning and obstetric clinics, prisons, drug-treatment centers, and more. Testing has also been common among the two[3] major high-risk groups of **M**en who have **S**ex with **M**en (MSM) and **I**njecting **D**rug **U**sers (IDU).

F(HIV) varies dramatically between high-risk and low-risk groups. It is often 20–40% or more among MSM and often nearly as high—or even as high— among IDU. In hospital patients and clients at sexually transmitted disease (STD) clinics who are not AIDS patients, F(HIV) is typically only a few percent. In the healthiest populations—military personnel and blood donors—it is fractions of a percent.

Data have now accumulated from tens of millions of tests[4], the great majority on groups not thought to be at any great risk. When those results are collated and compared, they turn out to be incompatible with the widely accepted (but not unanimous[5]) view that HIV tests detect the AIDS-causing virus that is presumed to have spread via sex and infected needles from the original centers of AIDS— New York, Los Angeles, and San Francisco. The tests reveal that:

1. HIV was already present in every part of the United States when testing began in 1985.
2. F(HIV) has decreased since 1985.
3. F(HIV) is not uniform across the country: it is higher in the North-East and South, especially the South-East, than in northern, central, or western regions. Whereas F(HIV) is always high in New York, there are no indications that it originated in and spread from Los Angeles or San Francisco.
4. That geographic distribution of F(HIV) is the same for disparate social groups, whose sexual behavior and drug-related behavior are hardly alike: military cohorts, Job Corps, child-bearing women, people tested at publicly funded clinics, IDU, MSM.
5. The pattern of geographic distribution of F(HIV) has not noticeably changed since testing began.
6. Direct observation has revealed that HIV is not readily transmissible via sex or needle-stick.

These points have not hitherto been stressed; presumably they were over-looked because the emphasis in HIV-testing was on monitoring the spread of infection in order to devise strategies for confining that spread. The results of HIV surveys appear not to have been used to test the hypothesis that HIV is infectious, to study the epidemiology of **HIV** by contrast to the epidemiology of AIDS.

Demographic Characteristics of Positive HIV Tests

F(HIV) varies characteristically with age, race, and sex (among other things), with remarkable uniformity in all studied groups. These regularities appeared to me so surprising, and so incompatible with the accepted view of HIV/AIDS, that I consulted the Centers for Disease Control and Prevention; they responded, ''Your data 'regularities' appear to be true, and we agree that they are not 'artifacts'''[6].

F(HIV) increases with age from the teens up to middle age (somewhere between 30 and 50) before decreasing again at greater ages [1–5, 16–23]. The variation is much more pronounced with men than with women. Among teenagers, F(HIV) is often higher among females than among males [2, 3, 17, 18, 24]. Above the teenage years and into middle age, F(HIV) is higher among men than among women [2, 3, 6, 7, 16, 18, 23, 24]. That dependence on age is shown in every social group and in every racial sub-group for which data have been reported. Thus age influences F(HIV) as an independent variable similarly in a qualitative but not a quantitative fashion for males and females.

Race, too, influences F(HIV) characteristically and independently: ''One of the most striking observations from these surveys is the marked race/ethnicity differences in HIV prevalence. In nearly all of the populations, prevalence was substantially higher among blacks than among whites. Although data from Hispanics were less consistent, prevalence among Hispanics was lower than among blacks and slightly higher than among whites in most populations'' (p. 38 in [5]). Those generalizations are illustrated and thereby also confirmed in a great number of individual studies [for example, 2, 3, 5–9, 16–19, 23–25]. In samples large enough to yield meaningful data on other ethnic groups, Asian subjects always tested lower than whites [4, 5, 17, 21, 22, 26]. In the few groups large enough to report F(HIV) separately for Native Americans, it was closer to that among white Americans than among any other racial group [4, 5, 9, 17, 21, 22].

These demographic features of F(HIV) data have intriguing and far-reaching implications as to what HIV tests actually detect, to be discussed in Parts II and III of this series of articles. For the present purpose—to examine the postulated spread of HIV across the country and into the general population—the influence of these demographic variables must be acknowledged when comparisons are made between groups whose compositions differ by age, race, and sex.

Even where testing has been mandatory for all members of a given group—which has been the case since 1985 for military cohorts, the Job Corps, and blood donors—those groups still represent only samples, of volunteers, from the general population. The compositions of these samples will fluctuate as to age, race, and sex, even when a given type of group—military recruits, say—is compared for different periods of time. If a particular cohort of military recruits comprises an unusually high proportion of black males in their twenties, say, and an unusually low proportion of young white females, then F(HIV) ''for military recruits'' will seem abnormally high in that year.

In most cases, the published data do not reveal all the details needed to adjust for such changes in composition. Therefore, when comparisons are made within and between groups of different sizes, studied at different times, some random fluctuations in magnitudes and ratios must be expected. In this article, maps of F(HIV) distribution are used extensively; some random variations in shading of a certain number of States must be expected. There is an extended discussion of this point in the Appendix.

Nevertheless, and almost in spite of this caveat, all tested groups are alike in the major points 1 to 5 above.

HIV Was Already Ubiquitous in the 1980s

HIV was everywhere present in the United States by the time testing began. Already in 1985, F(HIV) was about 0.4 per 1000 among blood donors—the group most carefully screened against individuals from high-risk groups—as far apart as Boston, Washington (DC), Philadelphia, Peoria, Tulsa, Detroit, Los Angeles, and Portland—in other words, in the North Atlantic, Mid-Atlantic, North-East, Mid-West, South Central, Mid-Pacific, and North-West regions [10]. The geographic distribution for donations during 1986–87 is shown in Figure 1. (For details of how maps were re-drawn for this publication to make them as comparable as possible, see the Appendix.)

For 1985–86, among potential military recruits from 43 states, F(HIV) averaged about 1.5 per 1000 [18]. For 1985–89, F(HIV) among *teenage* applicants for military service, from 200 counties in 41 states, was 0.35 per 1000—about the same among women as among men [24]. In both these instances, the states and counties from which no HIV-positive individuals came were those of low population; the numbers of potential recruits from those locales were not large enough to ensure that something occurring only on the order of 1 per 1000
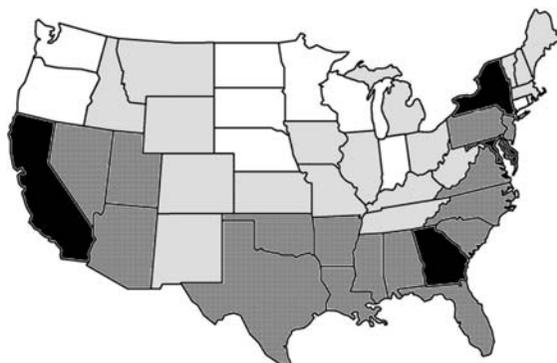

Fig. 1. HIV-1 antibody prevalence in blood donors, combined data from adjacent centers, by state, July 1986 to June 1987. Re-drawn from Figure 6.3(c) in [27]. No shading for 10 states with F(HIV) ≤0.06 per 1000; light shading for 18 states with F(HIV) between 0.07 and 0.14/1000; heavy shading for 15 states between 0.14 and 0.25/1000; blackened for 5 states, 0.26–0.4/1000.

would have been detected. The geographic distributions reported for 1985–86 (310,000 individual tests) and for 1985–87 (1.25 million tests) are shown in Figures 2 and 3, respectively.
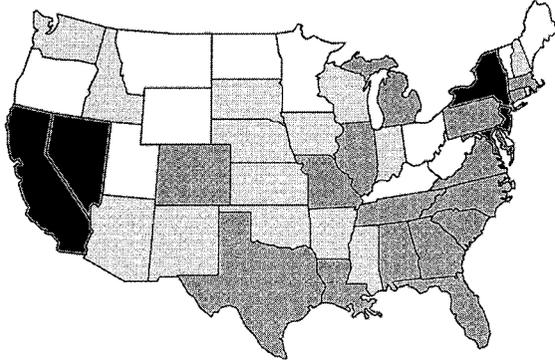

Fig. 2. F(HIV) in the first cohort of military applicants tested (310,000), October 1985–March 1986 [18]. F(HIV) was ≤0.5/1000 in the clear areas, 0.5–≤1.0 in the lightly shaded, 1.0–≤2.0 in the more heavily shaded, and ≥2.0/1000 in the blackened States.

Fig. 3. HIV-1 antibody prevalence in military applicants (1,250,000) by state, October 1985 to September 1987. Re-drawn from Figure 6.3(b) in [27]. (The data were described as "sex-adjusted" without explaining how that adjustment was made.) No shading for 10 states with F(HIV) <0.4 per 1000; light shading for 18 states with F(HIV) between 0.4 and 0.7/1000; heavy shading for 15 states between 0.8 and 1.5/1000; blackened for 1 state, 8–3.4/1000.

In the late 1980s, among applicants for marriage licenses, F(HIV) was a few per 1000 in widely scattered states—Alabama, California, Connecticut, Georgia, Illinois, Louisiana, New Mexico, Oklahoma [20]. During 1988–90, among students at 19 universities and 24 colleges across the USA, F(HIV) was found at a level of 2/1000 [28].

Women who delivered children also tested positive all over the country by the late 1980s, over 4 per 1000 in a few States and as high as 5.8/1000 in New York (Figure 4) [29].

Fig. 4. HIV-positive child-bearing women around the USA, 1988–90 [29]. States with a ? reported
         no data. F(HIV) increased from ≤0.4/1000 in the white areas through 0.5–1.9 in the lightly
         shaded, 2.0–3.9 in the more heavily shaded, to ≥4/1000 in the blackened States.

Members of the Job Corps from all across the country also tested positive, up
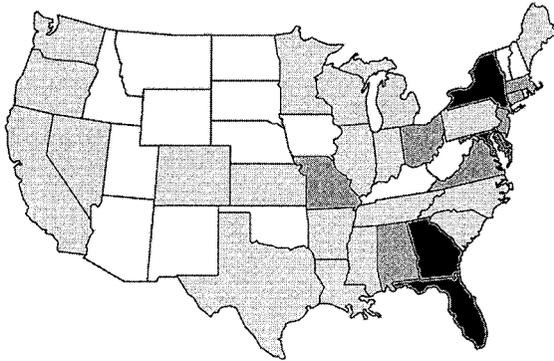to nearly 1% (Figure 5).

Fig. 5. F(HIV) in the Job Corps, 1987–90 [2]. F(HIV) was ≤1.0/1000 in the clear areas, 1.0–3.5
         in the lightly shaded, 4.2–6.9 in the more heavily shaded, and ≥7.2/1000 in the black-
         ened States.

These maps illustrate the unsymmetrical geographic distribution found in all
tested groups in the first years of HIV tests. That distribution has not changed over
the years, as illustrated under "Unchanged Geographic Distribution", below.

How Did HIV Enter Low-Risk Groups?

HIV is said to have entered the United States around 1970 among drug users
and gay men in a few large cities. That belief is based on the appearance of
AIDS around 1980 and an estimated latent period of about 10 years between
HIV infection and symptoms denoting AIDS. HIV is presumed to have spread

from these communities of IDU and MSM into other groups via sexual partners of IDU and MSM.

The data show that the geographic distribution of F(HIV) has a distinctive East-and-South weighting for:

- blood donors, drawn from the healthiest people in the population, middle and upper-class sectors being heavily represented;
- child-bearing women, who cover the social and economic gamut;
- the military, comprising graduates from high school or college who are screened to exclude MSM and IDU;
- the Job Corps, comprising largely unemployed youth who did not graduate from high school and who are not screened to exclude drug users or MSM.

To spread into these disparate sectors of the population, all across the country, in only 15 years, HIV would have needed to spread with astounding rapidity to attain so uniform a distribution among these disparate social groups.

But such a spread is incompatible with the evidence. For instance, why would F(HIV) be higher near the AIDS epicenter of the East (New York) than in the epicenters of the West (Los Angeles and San Francisco), as shown in all the distribution maps? And how could it happen that in California [30]—and even in San Francisco, one of the first locales of AIDS [31]—F(HIV) among child-bearing women is quite low (≤2 per 1000) [30, 31] and remained unchanged during six successive years (1990–95) [31]? With so many high-risk males in the vicinity, why were so few women infected?

Those rhetorical questions become moot in view of actual observations of infection rates, reported below, which show that HIV does not spread rapidly via sex or needles: it is transmitted with an efficiency of less than 1%.

Why Is F(HIV) on the Order of Parts per Thousand in so Many Low-Risk Groups?

It is remarkable that every **low-risk** group turns out to contain some HIV-positives, and moreover, that there always seem to be between a few per 10,000 and a few per 1000, even in other countries: in Canada, 0.3/1000 (British Columbia) or 0.6 (Quebec) [32]; in Germany, 0.14 to 0.57/1000 among child-bearing women and about 0.02 among blood donors [33]; in the United Kingdom, 1.4/1000 among women giving birth [34]; in South Africa, 0.4/1000 cumulatively among blood donors in Natal up to 1989 [35] and 0.34/1000 up to 1988 in South Africa as a whole [36]; between 0.36 and 2.2/1000 among pregnant black women in the Transvaal, 1987–88 [37]; 3/1000 among child-bearing women in Johannesburg in 1988–89 [38].

The Data Are Not Misleading

A natural first response to these data is to suggest that such rare HIV-positives as a few per 10,000 or per 1000 represent some sort of artifact; for example, that

groups of supposedly low-risk individuals actually contained a few from high-risk groups, and that therefore the results do not show a penetration of HIV into the general population. Or that these were "false positive" tests.

The data themselves directly disprove such suggestions.

If F(HIV) in low-risk groups resulted from random, occasional inclusion of infected high-risk individuals, then the data could not show the marked regularities over age, sex, and race that in fact they do: some samples would have a positive or two, others none; and there is no reason why random occasional inclusions of this sort would show regularities by age, race, and sex.

Furthermore, among HIV-positives, the ratio between sexes is rarely more than 2 men for each woman and sometimes (among teenagers) less than 1, whereas the ratio is much higher among the high-risk groups (AIDS cases), which, throughout the 1980s, comprised 95% males. Random inclusions from high-risk groups could not account for the numbers of HIV-positive females in the various samples and groups.

Similarly, the regularities in the data exclude explanation in terms of "false positive" tests. The same trends by age, sex, race, and geographic distribution are shown over the range of F(HIV), from parts per 10,000 (among blood donors) to parts per 1000 among the military, significantly higher in the Job Corps, and higher still in many public testing clinics. (Data to be cited at length in Parts II and III show that these regularities are seen even when F(HIV) reaches levels of 10% or more in the high-risk groups.) Thus, if there were a significant proportion of "false positives", they would have to be showing the same demographic characteristics as the genuine positives. The tests would then be non-specific and worthless.

All the reported tests were carried out under the auspices of the Centers for Disease Control and Prevention or the Army HIV Research Office or the American Red Cross, using the recommended methodology of duplicate ELISA tests followed by Western blot. If these results are to be dismissed as artifacts or false positives, then no published data at all could be taken as reliable.

In any case, as already noted[6], the Centers for Disease Control and Prevention have agreed that the regularities described here are real and not artifactual.

The Prevalence of HIV Has Decreased

Not only was HIV everywhere in the country by 1985; F(HIV) actually decreased after 1985 in military cohorts, the Job Corps, people tested at publicly funded sites, and blood donors (Figure 6).

That decrease is seen not only for those disparate social groups overall, but also within them in both sexes[7] [6, 8] and for all racial sub-categories [8, 39]. It is seen in individual States [4][8] as well as for the nationwide samples. In the Army, the decrease is seen in each of the separately observed categories of applicants, active-duty soldiers, National Guard, and Army Reserve [6, 24].

Strangely, the same declining trend was seen in Germany. F(HIV) in donated
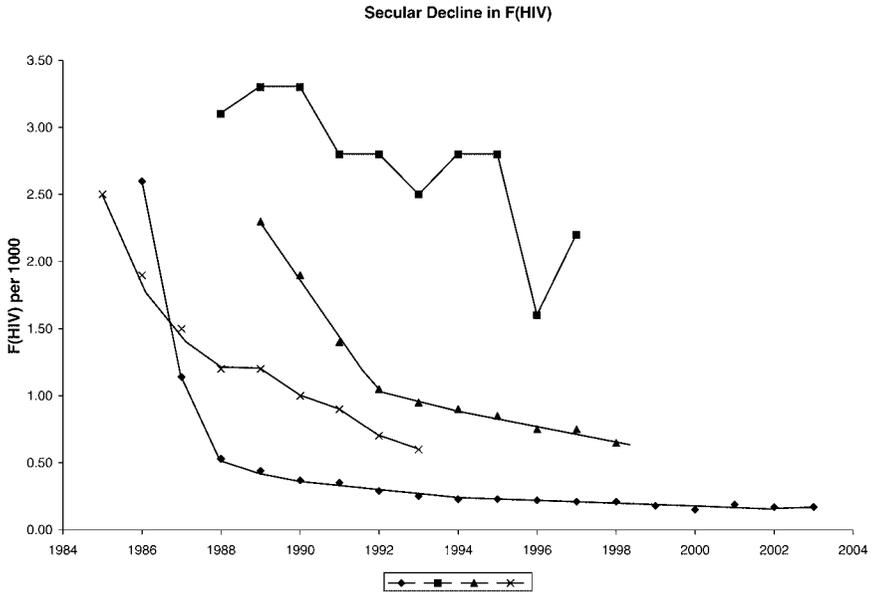
Secular Decline in F(HIV)

Fig. 6. Decline in F(HIV) observed in Job Corps (■—data from [3, 5, 8]), at public testing sites (▲—data from [4, 21, 22]), among applicants for military service (◆—data from [6]), and among blood donors (X—data from [8]). The actual magnitudes differed between these groups; for readier comparison in this figure, the numbers for blood donors were multiplied by 10 and those at public sites were halved. The point to be made is simply that of a decrease over time in all groups.

blood was 0.098/1000 in 1985, declined for several years, and has been in the range of 0.024–0.009/1000 since then [33].

Even more curious, however, is simply that there was a decrease at all, anywhere, among any group. Under the currently accepted view of HIV/AIDS, signs of infection by HIV—the antibodies detected by ''HIV tests'' —are irreversible: ''antibody to HIV is a consistent and persistent marker of prior infection. Thus, the seroprevalence in a given population at a given time documents the *cumulative* infection among members of the population up to that time'' (pp. 1169–1170 in [40]; emphasis added).

This is one of the most basic presumptions of the official view: antibodies to HIV indicate actual infection; there is no way of curing the infection, of eliminating all the virus once it has entered an individual; therefore, the total number of infected people can only increase, unless the number of infected who die exceed the number being newly infected. Among the general population—that is to say, among the low-risk populations to which these F(HIV) data refer—deaths from AIDS are quite rare. Thus, ***no decline at all*** is to be expected in these groups. How then to explain the declines indubitably observed in all the groups just cited?

These declines mean that the fresh annual recruits into each group, year after year, had a lower F(HIV) than the previous year's recruits. But since the recruits

are drawn from the wider population, that would bespeak an even more rapid decline of F(HIV) in the general population than is depicted in Figure 6.

This general decline, seen in so many population groups that it cannot be an artifact, is clearly incompatible with the current view that infection by HIV is permanent. It is not too strong to say that it disproves that view. Nor is it the only observation that stands in contradiction to the official view:

1. In every group for which there are data, F(HIV) decreases above a certain age—which differs somewhat between groups but is in the general range of what one might loosely call early middle age, say 30s to 50s [1, 4, 7, 16, 17, 21, 22, 25].

2. There have been many anecdotal reports of individuals reverting to HIV-negative even after confirmed HIV-positive tests [41; pp. 425–426 in 42; pp. 50–55 in 43].

3. In several groups, the observed incidence of new infections is of the same order of magnitude as the overall prevalence:

   — among repeat blood donors [1, 11, 12], for example, 0.012% and 0.003% respectively [44];
   — among applicants for military service [40];
   — among active-duty soldiers [16] and sailors [7], the rates of new infections turned out to be within a factor of 2 or 3 of the overall F(HIV), a surprise to the researchers [16]. (HIV-positive applicants for military service are not inducted [25].) It was conjectured that the overall F(HIV) would double in 2 or 3 years. With the benefit of hindsight, we now know that this did not happen—to the contrary, F(HIV) declined somewhat[9].

4. Among rehabilitated IDU [45] who had completed treatment and remained drug-free, F(HIV) was less by half than among IDU who had just begun detoxification treatment. Among those who had remained drug-free for more than a year, F(HIV) was only a quarter of that among former IDU who had remained drug-free for less than a year. While these are not serial observations on the same individuals, they warrant a strong inference that, for some IDU at least, HIV-positive is a reversible condition.

I have not found any published attempt to explain the clearly documented decrease of F(HIV) over the years. That it is observed in so many different social groups, and so many individual studies, marks it as a real effect. In several studies or groups, the decrease was most rapid in the mid- to late 1980s, and it has been minor since the early 1990s. Perhaps the rather rapid decline during the first few years of testing reflects changes in methodology. Crewdson [46] has described in considerable detail how unsatisfactory the first commercial test kits were (p. 249 ff.), and it has been mentioned that the criteria for the Western blot test changed in 1987 [47] and again in 1988 and 1989 [48]. Still, the magnitude

of the decline is not the salient point. The official view of HIV/AIDS as a continually spreading and increasing infection is disconfirmed by all available data from HIV tests, which reveal that F(HIV) has decreased rather than increased since 1985.

Static "Epidemic"

An official press release from the Centers for Disease Control and Prevention in June 2005 stimulated news items like the following:

"For the first time since the height of the AIDS epidemic in the 1980s, more than a million Americans are believed to be living with the virus that causes AIDS, the government said Monday"[10].

The media's memory is short, for the government had announced a million Americans infected with HIV already in 1986, 1987, 1988, and 1989: "In 1986 . . . between 1 and 1.5 million persons were infected", according to the Centers for Disease Control and Prevention[11].

In 1987, again according to the Centers for Disease Control and Prevention: "The estimate obtained by incorporating these revisions into the 1986 calculation (Table 14) —945,000 to 1.41 million-—differs little from the earlier figure" [44].

"In mid-1988 it was estimated that 1.5–2 million Americans had been infected", according to Donald Francis, AIDS Adviser, California Department of Health Services, and Richard Kaslow, Chief of Epidemiology and Biometry, National Institute of Allergy and Infectious Diseases (p. 93 in [27]).

For 1989, according to the Centers for Disease Control and Prevention[12], "Currently about 1 million persons in the United States are infected with human immunodeficiency virus (HIV). . . . it is estimated that about 750,000 persons in the United States were infected with HIV at the beginning of 1986 (Table 1)".

Thus, it seems that the number of HIV-infected Americans, about 1 million, is the same now as it was two decades ago. This estimate corresponds to 3 or 4 individuals per 1000 people in the United States. As noted earlier, this magnitude seems to be characteristic of low-risk populations, including in other countries. The agreement is not accidental, of course, because the estimates were based directly on the reported data for every tested group and on the estimated sizes of those groups. The Health and Nutrition survey of households, which surely represents something like the general population, found F(HIV) to be 3.9 per thousand for 1988–91 [49].

Actual data (Figure 6) have also shown no increase—rather a decrease—in F(HIV) in such large, continually monitored groups as the military and blood donors as recently as 2004 [6] and 2002 [1], respectively.

On all these counts, the basis for the claim in the press release of June 2005 that *"for the first time"* the number of infected Americans exceeded 1 million is far from clear. The onus should be on the Centers for Disease Control and Prevention

to explain. A full explication would have to include the manner in which these estimates are arrived at and how that differs from approaches used earlier[13].

Estimates made in the 1980s and early 1990s, all of which agreed on about 1 million infected Americans, were based on actual results of HIV-tests on various groups, as cited above. In recent years, however, "back calculation" has been used. This assumes that AIDS appears about 10 years after infection by HIV, calculates what HIV infection must have been 10 years earlier than each year's AIDS reports, and then projects that figure into the present (and future). There seems little reason to prefer this method to one based solidly on actual F(HIV), since it is more dependent on more assumptions. Moreover, the definition of AIDS has been changed several times[14], which makes back-calculations even more hazardous and doubtworthy.

Another strong reason for preferring actual reports from HIV tests to estimates from assumption-based computer models is that by now a large proportion of the population has had an HIV test. In 2001, more than 45% of 18–64-year-olds reported having had an HIV test at some time, and more than 27% reported one in the past 12 months[15].

At any rate, whatever the reasons, the estimates of F(HIV) in recent publications from the Centers for Disease Control and Prevention are confusing and not obviously connected with reports of actual F(HIV). Thus, at the end of 2003, the estimated number of persons living with HIV infection (not AIDS) was 175,000 (rounded figures), plus 406,000 (rounded) living with AIDS (Table 12, pp. 22–23 in [50]). For the end of 2002 [51], the corresponding estimates had been 145,000 and 385,000. Even at this *estimated* rate of annual increase of 30,000 and 20,000, respectively, by the end of 2005 the grand total of merely HIV-infected plus actual AIDS (which has as criterion infection with HIV) would be about 680,000, not 1 million.

At the end of 2003, the actual data reported from "41 areas with confidential name-based HIV infection reporting" (Table 18, p. 33 in [50]) was 221,000 (rounded). The States not so reporting had a combined population of 71 million[16] out of a U.S. total of 290 million. A reasonable estimate for the United States as a whole might then be 290/219 times 221,000 = 293,000, rather than the 406,000 shown in Table 12 in the same CDC report (see above).

But leave these details aside, and consider only the conclusions in official reports. The Centers for Disease Control and Prevention has never repudiated its published estimates that about 1 million Americans were infected in 1986, 1987, 1988, and 1989. Its latest estimate, in June 2005, is still 1 million. Such a lack of change in magnitude is consistent with the data in Figure 6 and with the unchanged geographic distribution described below. Moreover, a constant national rate of F(HIV) would not be unique to the United States: The same is true of Haiti, which drew much attention in the early 1980s because Haitians in the United States had been found to test HIV-positive to a pronounced extent. In 1986, the prevalence of HIV in Haiti was estimated at 5% [52]. In 1990, Haiti's capital city had as many as 6% infected [53]. For 1999–2000, the rate was about

Fig. 7. F(HIV) among applicants for military service, 1993–97 [5]. F(HIV) was ≤0.1 in the clear areas, 0.1–0.39/1000 in the lightly shaded, 0.4–0.69 in the more heavily shaded, and ≥0.7/1000 in the blackened States.

Fig. 8. F(HIV) among members of the Job Corps, 1993–97 [5]. F(HIV) was ≤0.4/1000 in the clear areas, 0.5–1.9 in the lightly shaded, 2.0–3.9 in the more heavily shaded, and ≥4/1000 in the blackened States.

Fig. 9. F(HIV) among child-bearing women, 1994 [9]. States with a ? reported no data or ≤3 months of data; F(HIV) was ≤0.4/1000 in the clear areas, 0.5–1.9 in the lightly shaded, 2.0–3.9 in the more heavily shaded, and ≥4/1000 in the blackened States.

Fig. 10. F(HIV) among child-bearing women, 1995 [9]. States with a ? reported no data or ≤ 3 months of data; F(HIV) was $\leq 0.4/1000$ in the clear areas, 0.5–1.9 in the lightly shaded, 2.0–3.9 in the more heavily shaded, and $\geq 4/1000$ in the blackened States.

3.8% in that capital city as well as in another region of Haiti, and both areas were classed[17] as "low risk". In 2003, the rate for the nation[18] was still 4.5–6%. In other words, the prevalence of HIV in Haiti has remained at about 5% for two decades.

Altogether, the evidence is that HIV is not a spreading infection. It seems to be an endemic condition, found in the United States in about one third of one percent of the population throughout the last two decades, but in Haiti at a level of about 5%. Part III of this series of articles will offer an explanation for that difference between the two countries.

Unchanged Geographic Distribution

Just as the magnitude of F(HIV) has not changed for two decades, or even decreased somewhat, so also has the geographic distribution not changed noticeably. Figures 1 through 5 were the earliest reports showing geographic
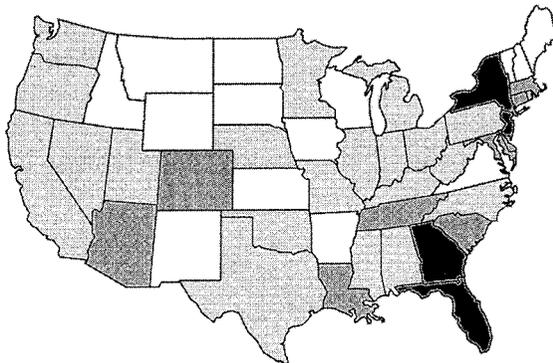

Fig. 11. F(HIV) among clients at public testing sites, 1995–98; data averaged from [4, 21, 22]. F(HIV) was $\leq 5.2/1000$ in the clear areas, 5.4–12.1 in the lightly shaded, 12.6–18.4 in the more heavily shaded, and $\geq 22.3/1000$ in the blackened States.
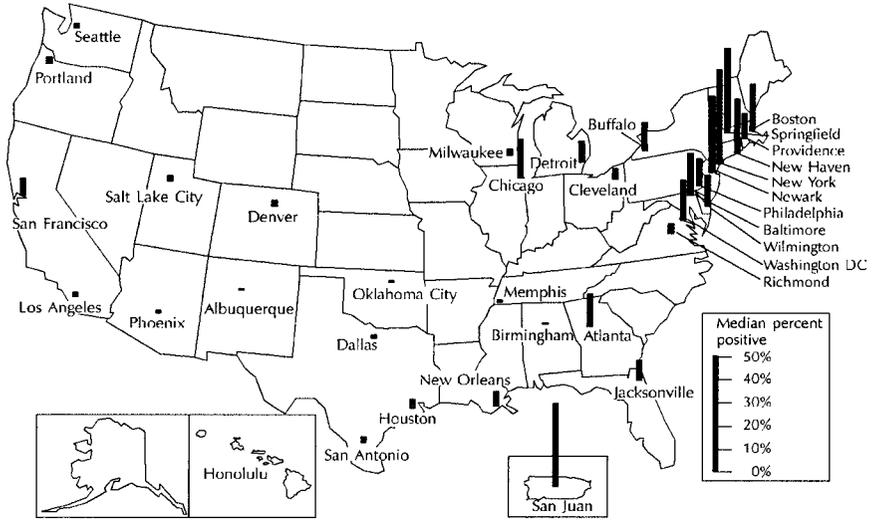
Fig. 12.   F(HIV) among IDU at drug treatment centers, 1991–92 [54].

distribution that I was able to locate for blood donors (1986–87), military applicants (1985–86 and 1985–87), child-bearing women (1988–90), and members of the Job Corps (1987–1990). The most recent such maps or data that I have found are for 1993–97 for military applicants and the Job Corps (Figures 7 & 8, respectively), 1994 and 1995 for child-bearing women (Figures 9 & 10, respectively), and 1995–98 for clients at public testing sites (Figure 11).
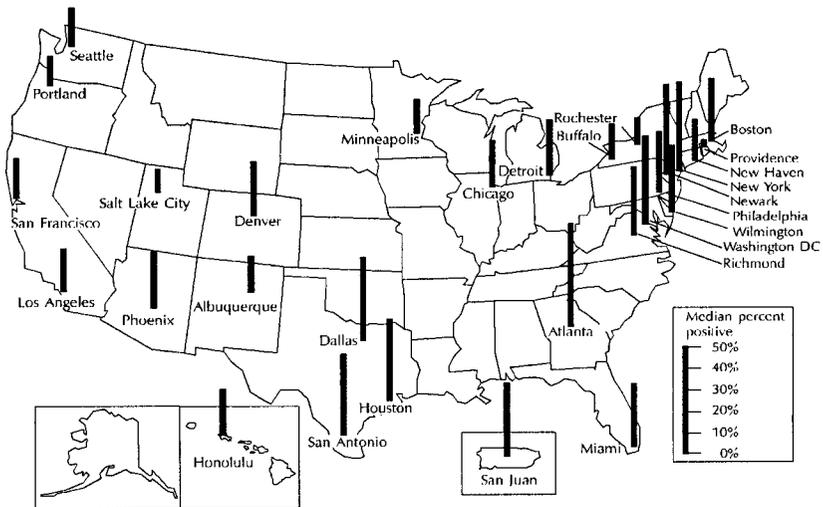

Fig. 13.   F(HIV) among MSM at STD clinics, 1991–92 [54].

There is no obvious difference between the distributions for the various social groups, nor for a given social group over time.

In every study that remarks as to the geographic distribution, even where actual maps are not given, it is universally the case that the level of F(HIV) is greater in the East and South than in the West and lowest of all in the North-Central regions, and this asymmetry of F(HIV) has often been the subject of specific comment [2, 3, 5, 9, 17, 18, 20, 24, 28, 54–58].

It bears noting that this Eastward weighting shows up even among IDU (Figure 12) and MSM (Figure 13): "While HIV prevalence among men reporting sexual activity with men was high in all areas, it was the highest in states along the Atlantic Coast, Texas, and Puerto Rico. ... Among female and heterosexual male injecting drug users and among persons who denied male homosexual contact and injecting drug use, rates were generally highest in the Atlantic Coast states, including Florida, and Puerto Rico and lowest in the Mountain and Pacific Coast states" (p. 24 in [54]). Articles reviewing many individual studies remark on the same pattern [32, 59].

The conventional HIV/AIDS view is unable to explain the East–West asymmetry of F(HIV): "The reasons for the persistent geographic heterogeneity in seroprevalence are unknown" [54]. Indeed, one would hardly expect a sexually transmitted infection to display so unvarying a geographic distribution.

This fact adds to the conundrum that F(HIV) has not increased in magnitude since 1985: If HIV arrived around 1970 in Los Angeles, San Francisco, and New York, it would have needed to spread explosively in just a few years to produce a geographic pattern that then remained stable since 1985; and if it had indeed spread so rapidly at first, why did it then stop spreading almost immediately?

HIV Is Not Readily Transmissible

That conundrum can be resolved as soon as it is posed: HIV did not spread rapidly, because it could not. The conventional wisdom—purveyed and entrenched by the popular media, by charitable organizations, and even by authoritative bureaucracies—has not kept pace with what has been learned over the years in direct studies of the transmission of HIV: HIV is unlike a sexually transmitted infection[19] (STI) in being not very transmissible. The probability of being infected when stuck by a contaminated needle is less than 1%; via unprotected intercourse the probability is something like 1 per 1000.

Needles

It is a shibboleth, but also a myth, that needle-sharing spreads HIV. One study found F(HIV) nearly twice as high among IDU who did *not* share needles (34%) as among those who did (19%), at the same clinic [60]. Similarly, an independent study in Montreal found that clean needles were associated with decreased transmission of hepatitis B but with *increased* F(HIV) [61]. A study of IDU prisoners in Maryland found no spread of HIV infection during 2 years [62].

At first, it had been feared that health-care workers would become infected through accidental needle-punctures. Actual data have shown the fear to be baseless. Surveys of medical personnel in military service found no occupational risk of HIV infection. Indeed, the annual incidence of HIV infection was actually lower among medical personnel than non-medical personnel for females (0.68/1000 vs. 0.86) and for married males (0.09 vs. 0.10). Only among male nurses was this reversed, indicating that the risk here was owing to some non-occupational factor [25, 63].

The risk of transmission of HIV from needle-stick was found to be low or negligible in another study in which as many as 14% of subjects reported such contact with needles that had made contact with AIDS patients [64]. Quantitative estimates of risk in still other studies were on the order of 0.3% to 0.4% [65–70]. For comparison, transmission of hepatitis B via needle-stick has an efficiency of 20–40% [67]. Among needle-sharing IDU in Britain, hepatitis B and C were transmitted with efficiencies of more than 10% and 20%, respectively, while the efficiency for HIV was well under 1% (Coreslide 8 in [71]).

Despite initial concerns, health-care workers have not contracted AIDS even after inadvertent exposure to HIV in blood [72]. Altogether there are very few instances, if any, of becoming HIV-positive through occupational hazards [73]: only 40 documented and 83 possible cases up to 1993 (Table 16, p. 19 in [74]), by which time there had been 360,000 reported cases of AIDS; revised to only 57 possible cases up to December 2001 [75], by which time the total number of AIDS cases was nearly 790,000.

Intercourse

It is also a shibboleth, and again also a myth, that unprotected intercourse brings great danger of spreading HIV. The risk of sexual transmission of HIV has been found in several independent studies to be well under 1%, more like 0.1% or even less:

- on the order of 1 per 1000 acts of intercourse [76], or, more precisely, 0.8–1 per 1000 [77] or 0.5–2.3 [78];
- in a 10-year study [79], rather less than 1 per 1000 for male-to-female transmission (0.0009 was the actual figure) and much less than that (0.00011) for female-to-male transmission;
- less than 1 per 1000 for male-to-female and half that for female-to male [80];
- for male-to-female, 0.8–1 per 1000 [81], or 0.6–2.6 [82], or 0.6–0.9 [83], or 0.6–0.8 [84], or 0.5–1.2 [85];
- the risk of becoming HIV-positive was only 7% during a year of unprotected intercourse with an infected partner [86] (which would correspond to only 70 acts of intercourse in a year at a transmission rate of 1 per thousand; if there had been more frequent intercourse, then the transmission rate was correspondingly even lower);
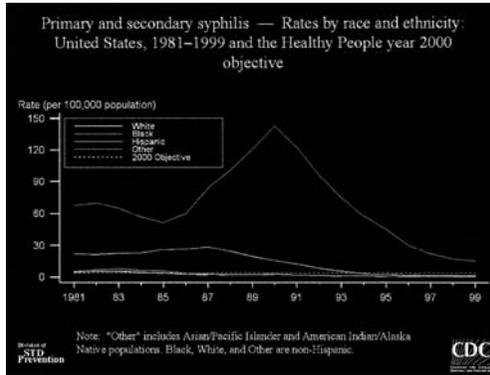
Fig. 14.   Racial disparities in incidence of primary and secondary syphilis, 1981–99 (Top curve with peak is for 'Black'); re-drawn from slide 10 in *Syphilis—Sexually Transmitted Disease Surveillance 1999*, Division of STD Prevention, Centers for Disease Control and Prevention; Available at: www.cdc.gov/nchstp/dstd/Stats_Trends/1999Surveillance/Syph-ilis/sld010.htm. Accessed 22 June 2005.
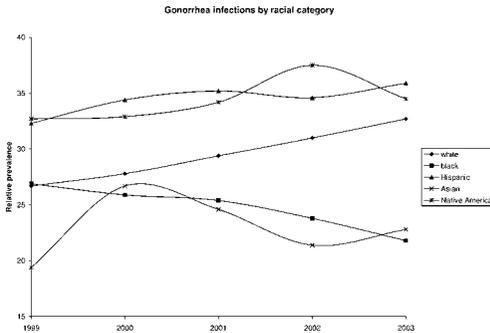

Fig. 15.   Racial disparities in incidence of gonorrhea. Data from Table 21B, page 109 in *Sexually Transmitted Disease Surveillance, 2003* (Centers for Disease Control and Prevention, September 2004. Available at: www.cdc.gov/std/stats/).

This figure is intended to portray only relative ***changes***, not relative magnitudes, so the vertical scale is not the same for all groups. The actual prevalence per 100,000 in 2003 was—rounded to two significant figures—23 for Asian, 33 for white, 72 for Hispanic, 104 for Native American, and 660 for black.
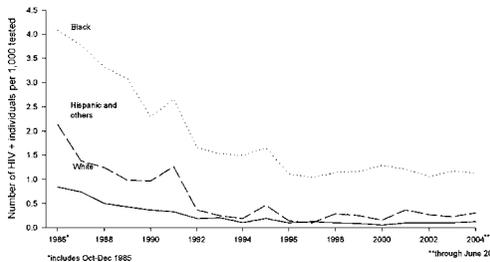

Fig. 16.   Uniform declining trend of F(HIV) in the three largest racial groups among applicants for military service, 1985–2004 [6].

- in Africa (Mwanza), a study of 1802 couples found infection rates among discordant couples (one partner initially HIV-positive and the other initially negative) of 10 per 100 person-years male-to-female and 5 per 100 person-years for female-to-male [87]. For 100 acts of intercourse per year, that corresponds once again to about 1 per 1000 acts for male-to-female and half of that for female-to-male transmission.

Thus *the risk of transmitting HIV is hundreds of times less than that of transmitting STDs*:

"the likelihood of transmitting gonorrhea during vaginal intercourse ranges from 20 percent to 80 percent for female-to-male transmission and from 50 percent to 70 percent for male-to- female transmission.[6] The chance of infection for syphilis is about 30 percent with each sexual exposure to syphilis sores.[7] In general, herpes, HPV, trichomoniasis, gonorrhea, chlamydia, and syphilis are the most highly transmissible STDs" [88]. Other estimates for gonorrhea transmission are 50% likelihood for male-to-female transmission and 25% for female-to-male [89], or 80% male-to-female and 20–25% for female-to-male [90].

In any case, "the transmission probabilities presented are so low that it becomes difficult to understand the magnitude of the HIV-1 pandemic" [91] — difficult to understand if HIV is sexually transmitted; indeed, *impossible* to understand. It remains only to draw the obvious inference that HIV is not sexually transmitted.

Several studies have reported a positive correlation between seroconversion and receptive anal intercourse [92, 93], yet even there the estimated risk of seroconversion is low, for example, 4% per year, with low risk for the insertive partner and only *rare* infection via other types of intercourse [92]. That studies in San Francisco [94] found F(HIV) essentially the same in monogamous gay couples as in other MSM brings into question even the risk of anal intercourse, as well as being most curious for an STI. Also puzzling for an STI would be the fact that there was no correlation between exposure to prostitutes and probability of testing HIV-positive among patients at STD clinics in New York [95]. Though sex between men is common in prisons, a maximum seroconversion rate of 1.7 per 1000 was reported in a 3-year study of Nevada inmates [96]; and in a military prison, no seroconversion occurred over several years during which there was a 2% transmission rate of hepatitis B [97].

Demographics of Sexually Transmitted Diseases

The demographics of HIV differ in a number of respects from those of the well known STDs. The Centers for Disease Control and Prevention continues to estimate that about 1 million Americans are now "living with HIV", as mentioned above. This is far fewer than the estimated 4 to 8 million *new infections annually* with chlamydia, 500,000 with genital herpes, and 400,000 with gonorrhea[20]. This huge numerical disparity between annual rates for HIV and for STDs also makes a mockery of the oft-mooted hints that infection with HIV may be catalyzed by infection with STDs.
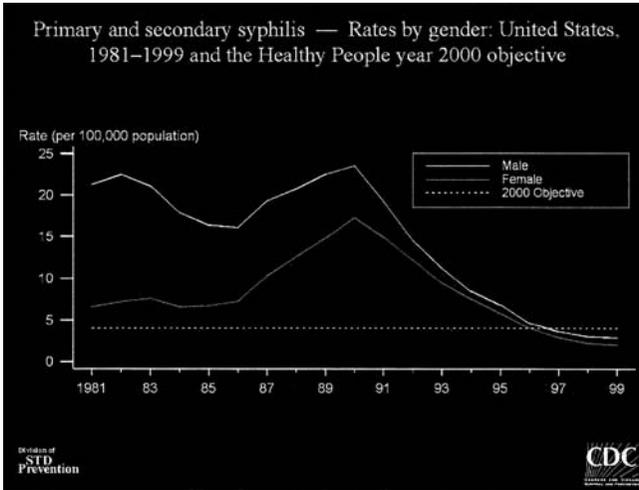
Fig. 17. Four-fold change over the years in male-to-female ratio for incidence of primary and secondary syphilis, 1981–99; re-drawn after slide 9 in *Syphilis—Sexually Transmitted Disease Surveillance 1999*, Division of STD Prevention, Centers for Disease Control and Prevention; Available at: www.cdc.gov/nchstp/dstd/Stats_Trends/1999Surveillance/ Syphilis/sld009.htm. Accessed 22 June 2005.

The extraordinarily uniform regularities that F(HIV) displays are not shown by chlamydia, gonorrhea, or syphilis. Instead, the rates of those infections vary up and down over time, the periodic outbreaks are often concentrated disproportionately in a few social groups, and the geographic foci change.

For example, there was a significant epidemic of syphilis about 1990 that
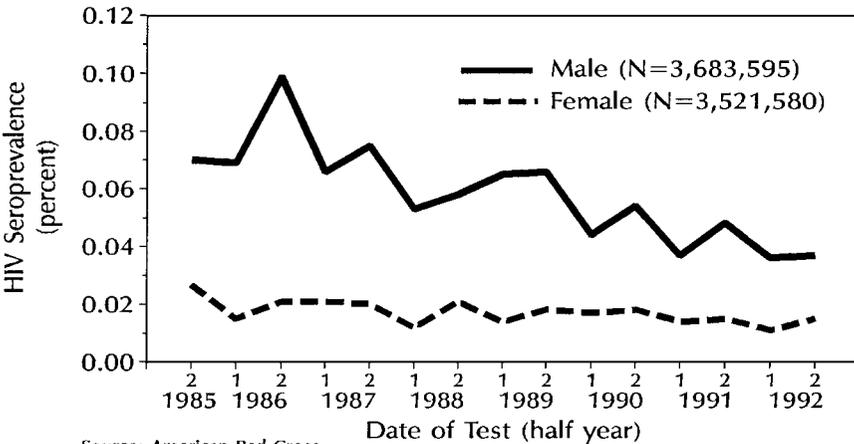
Fig. 18. Male-to-female ratio for F(HIV) hardly changes over a decade; data for first-time blood donors [8].

TABLE 1
Locales with Highest Reported Incidence of Gonorrhea in Various Social Groups,
1999–2003 (numbers are percentages)

Job Training 2003 16–24s	Juvenile detention 2003	Adult corrections 2003	Adult corrections 2001	Juvenile detention 2001	Army recruits 17–37s 1999–2000
OK 6.3	OH 3.3	PA 6.6	WI 5.7	CA 3.2	TN > 1
WV 6.2	OK 3.2	NE 5.2	IN 2.8	GA 2.7	GA > 1
MO 3.8	IL 2.5	WI 4.7	PA 2.2	MD 2.3	AL 0.9
PA 3.6	NE 1.9	WV 3.4	MI 2.1	CO 2.1	OH 0.7
MI 2.9	KY 1.7	CA 1.7	CA 1.6	TX 2.1	LA 0.6
NV 2.7	WV 1.6	IA 3.0	IL 1.4	IL 1.9	SC 0.6
IN 2.5	TX 1.5	IL 1.4	NH 0.8	KY 1.6	TX 0.5
KY 1.9	NJ 1.4	NY 0.5	TN 0.5	PA 0.9	IL 0.5
FL 1.8	NY 1.3			WI 0.8	IN 0.2
CA 1.4	GA 1.0			UT 0.7	
	CA 0.6			NM 0.5	
	MD 0.6				
	OR 0.3				

Note: Data for Job Training from www.cdc.gov/std/stats/adol3.htm, Figure R, accessed 16 June 2005; for 2003 corrections from www.cdc.gov/std/stats/corrections2.htm#figmm, accessed 16 June 2005; for 2001 corrections from www.cdc.gov/std/stats01/2001SFCorrections2.htm#FigJJ, accessed 16 June 2005; for Army recruits from www.cdc.gov/std/stats00/2000SFAdol&YAdults3.htm#FigP, accessed 16 June 2005.

affected chiefly black communities (Figure 14), whereas ''in the late 1970's and early 1980's . . . [primary and secondary syphilis] primarily occurred in homosexual white men''[21].

In recent years, the rates of infection with gonorrhea, too, have changed quite differently over the years among different racial communities: During as few as 5 years, among white Americans there was a 22% *increase*; among black Americans, a 19% *decrease*; and the other groups varied up and down in individual fashion (Figure 15).

By contrast, F(HIV) shows a decline over the last two decades to a similar extent among black, Hispanic, and white Americans in every studied group [see, among others, 2, 3, 5–9, 16–19, 23–25], for example, for civilian applicants for military service (Figure 16) [6].

This regular difference by race is like something determined physically or genetically rather than behaviorally. Sexual behavior involves choices and constraints that are influenced by socioeconomic and cultural circumstances and lead to the sort of fluctuations seen with syphilis and gonorrhea.

Relative rates of infection among men and women, too, may indicate changes in relative behavior over the years. During two decades, the rates of infection of males and females with syphilis changed progressively by a factor of more than 2 (Figure 17).

By contrast, the relative rates of F(HIV) among men and women have remained constant for two decades across several disparate social groups[22], for example, among blood donors (Figure 18).

Once again, such a constant ratio of male to female indicates something physical, something that differs ***inherently*** and not behaviorally between men and women.

The geographic distribution of F(HIV) has remained without significant change for well over a decade, and has been the same in all social groups. By stark contrast, STIs tend to break out in different communities at different times. For example, Table 1 shows large and random variations in the incidence of gonorrheal infections, both over time and between social groups.

In 2003, the 13 States in which juvenile inmates showed the highest rates of infection included only half of the 11 States that had shown the highest rates in 2001; and among the 6 in both rankings, 2 went up and 4 went down. The same among adult inmates: only half of the top 8 States in 2001 were in the top 8 in 2003; one was higher, one lower, and two about the same.

In 2003, there was little in common between the Job Training Corps and juvenile detention centers so far as prevalence of gonorrhea was concerned; only in Oklahoma was the prevalence high in both groups, and that state was not in the top 8 for gonorrhea among adult inmates. A similar lack of concordance is seen between Army recruits (1999–2000) and juvenile inmates (2001).

Outbreaks of syphilis are also quite local: "Despite the decline in national syphilis rates to historic lows, syphilis remains a severe public health problem in a small number of U.S. counties. In 1998, over 50% of infectious . . . syphilis cases were reported from only 28 (<1%) counties, the majority of which are in the South"[23].

Altogether, with gonorrhea, syphilis, and other indubitable STDs, there is not the uniformity and regularity within and between social groups, and over time, and in geographic distribution, that F(HIV) displays.

Conclusion: HIV Is Not a Sexually Transmitted Infection

Direct observations, reported above, have shown that HIV is not efficiently transmitted either by infected needles or by sexual intercourse. It could not have spread by 1985 from the AIDS epicenters into the general population, with the wide distribution already found in all social groups when testing began. The demographic characteristics of HIV are quite different from those of gonorrhea or syphilis in chronology, geography, racial disparities, and gender differences. The lack of change in its geographic distribution marks it as somehow endemic, with a marked East–West asymmetry.

All this shows that HIV did not cause the outbreaks of AIDS of the early 1980s.

Prolegomenon

The conclusion just reached is diametrically opposed to "what everyone knows". "HIV, the virus that causes AIDS", can be heard and read every day in the popular media. So my conclusion will immediately incite a host of dis-believing rhetorical questions: So what ***is*** HIV? So what does cause AIDS? What

is AIDS? How could everyone have been so wrong for so long about HIV causing AIDS?

Quite reasonable, substantive answers are available to all those questions, but they can hardly be given in short order. Yet some sort of response seems needed here to make this paper's conclusion seem less incredible. Therefore, the following paragraphs outline those parts of the rest of the story that are already clear.

What Is HIV?

Part II of this series of papers will discuss the enormous variation in F(HIV) among different social groups, ranging from a few per 100,000 among repeat blood donors to 50% or even more among IDU and MSM. The reason is that F(HIV) is not specific for HIV but indicates some sort of general physiological challenge to health. That has been argued for many years on entirely different grounds—explicitly biological grounds, not epidemiological ones as here. In particular, the Perth Group [98, 99] argues that F(HIV) is a non-specific indicator of oxidative stress.

Part III of this series deals with the fact that F(HIV) varies by race with such regularity that *semi-quantitative* differences emerge. In the overwhelming majority of reports, the ratio of F(HIV) to that among whites is: Asians, ~0.5–0.9; Native Americans, ~1.1–1.6; Hispanics, ~1.5–3; and blacks, ~2.5–6. One has the choice of seeking for these observations a behavioral explanation or a non-behavioral one. Any behavioral explanation raises ghosts of such long-discarded and properly discarded theories as phrenology, physiognomy, or Cesare Lombroso's *Criminal Anthropology*, which asserted a strictly determinist connection between behavior and physique (or genome). Fortunately, a less racist and more scientifically (as well as politically) correct explanation is available. It points to the well-established fact of genomic polymorphisms that run broadly parallel with the usual racial categories. Such genetic markers have been used to track human migration patterns over the last few hundred thousand years. Racial patterns of this sort have been found among the genes that influence immune responses (p. 180 in [100]). These racial differences are beginning to be attended to in medicine—the best medication for people in one racial category is not necessarily the best for those in other racial categories. This explanation for racial differences in F(HIV) could also mitigate the recent fuss in South Africa over the classification—roundly denounced as racist—of "black" blood as too HIV-risky to be used for transfusion [101, 102].

Taken together, the data in Parts II and III of this series can account for the East-over-West distribution of HIV in the United States, for which the official view offers no explanation.

What Is AIDS and What Causes It?

"Acquired Immune Deficiency Syndrome" was devised as a label for cases of opportunistic infections indicating that the immune system had been damaged, seen in young men with no obvious cause for such damage. Over the years the definition has changed into something quite different, including a positive HIV

test (see note 14). Under the original definition, for instance, hemophiliacs should never have been classed as AIDS cases, since the immune system does not function in a *"normal"* fashion in hemophilia (pp. 145 f. and 271 ff. in [43]). Much material about HIV and AIDS in hemophiliacs is available on several reliable web-sites (note 5).

Because AIDS was first seen in young gay men, it was assumed to have something to do with gay sex. Not then recognized was the fact that AIDS struck only those gay men who were heavy users of recreational and other drugs, by no means only injected ([103], especially Chapter I and pp. 191–193). Most or all aspects of AIDS are explicable in terms of drug abuse, including why different opportunistic infections are found in those who (ab)use different drugs [104].

AIDS in Africa is an entirely different matter than AIDS in the USA—especially under the original definition of AIDS. In Africa, AIDS is almost certainly the result of endemic malaria, tuberculosis, and many other bacterial, parasitic, and viral diseases common in the tropical regions. Hodgkinson [105] and Root-Bernstein [43] have given detailed narrative accounts, and many pertinent articles are available on dissident web-sites (note 5). Malan [106] has described the fallibility of official estimates of HIV infections and of AIDS deaths in South Africa. An incisive critique of hypotheses about an African origin of HIV or AIDS, still pertinent almost two decades later, was published by the Chirimuutas [107].

How Could "Everyone" Have Been Wrong for So Long?

Recall first (note 5) that not everyone has been wrong: many competent, indeed distinguished people with relevant credentials have persistently denied that HIV has been proven to be the cause of AIDS.

As to the hold that HIV/AIDS theory has exercised, the outline of an account is already available. History of science offers innumerable instances in which a mainstream consensus turned out to be wrong [108]. In recent decades, bureaucratic institutions have come to control some fields of science and medicine to the extent that official views seem impervious to plain fact: there now exist ***knowledge monopolies*** [109].

It is an illustration of such a knowledge monopoly over HIV/AIDS that so few people among the general public are even aware that expert molecular biologists, including Nobelists, deny—on detailed, substantively argued grounds—that the theory has been proved. The HIV/AIDS case also offers a cogent illustration of the all-too-common willingness to accept indications of correlation as proof of causation.

Notes

[1] "AIDS" has been re-defined a number of times over the years. The term was originally introduced in connection with the outbreaks, first recognized in New York, Los Angeles, and San Francisco in the early 1980s, of unusual opportunistic infections—*Pneumocystis carinii*, Kaposi's sarcoma, candidia-

sis, and others as well—that rather quickly killed predominantly young gay men and drug abusers.

2 In newborns, it is presumed that a positive HIV-test reflects passive antibodies transferred from the presumably infected mother and possibly active infection as well.

3 Hemophiliacs constitute another group supposed, at least for a time, to be at high risk. Haitians were classed as high risk for a brief time in the mid-1980s.

4 The largest samples reported are 22 million tests at public sites, 1989–98 [4]; tests on 6,900,000 applicants for military service, 6,500,000 on active-duty soldiers, 2,600,000 National Guard personnel, and 1,600,000 Army reservists, 1985–2004 [6]; 1,100,000 sailors and Marines, 1986–88 [7]; tests on about 500,000 members of the Job Corps, 1987–90 [2, 3, 5, 8, 9, 13]; and tests on more than 10 million blood donors [1, 5, 10–12, 14]. Various cohorts of most of these samples were analyzed in a number of separate publications. Many other publications have reported HIV-test results from prisons, hospitals, child-bearing women, and from high-risk groups.

5 Convenient introductions to the considerable volume of dissenting opinion are the web-sites www.virusmyth.net/aids and healtoronto.com. Some dissident as well as mainstream opinions are given space at www.aegis.com.

The virusmyth site lists more than 2000 signatories to a petition that HIV/AIDS theory be re-examined. The site's home page has this quote:

> If there is evidence that HIV causes AIDS, there should be scientific documents which either singly or collectively demonstrate that fact, at least with a high probability. There is no such document.
> —Dr. Kary Mullis, Biochemist, 1993 Nobel Prize for Chemistry.

Mullis's Nobel Prize was for inventing the polymerase chain reaction (PCR), which is universally used in DNA studies. Mullis has described as invalid estimates of "viral load" of HIV made by this technique (chapter 18 in [15]).

6 Letter to the author, dated 19 May 2005, from Shari Steinberg, Divisions of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention, Centers for Disease Control and Prevention.

The letter continues, "The 'characteristic differentiation by race' that you note **is compatible** [emphasis in original] with a behavioral explanation", an opinion I do not share, since it implies that the same genomic patterns that determine skin color also determine sexual behavior, and in a similarly decisive manner. This will be discussed further in Part III of this series of articles.

7 In the Job Corps [3, 5, 8], F(HIV) for males showed a steady decline, but for females it fluctuated from year to year in a manner that could be interpreted as a slow decline or as remaining fairly constant over time. Variations in the racial composition of Job Corps entrants have been proposed as a possible reason for those fluctuations [3, 13].

8 Within the random fluctuations expected, particularly in States with small populations. Only two States showed a possible slight increase in F(HIV) in the late 1990s (Table 5 in reference [4]).

[9] These published data do not, however, answer definitively the question of possible reversion in a given individual, although there are some pertinent observations. In 1996, 324 HIV-positive soldiers remained on active duty, of whom most were senior enlisted, more than half were married, 50 had been diagnosed some 10 years earlier, and 25 were female [39]. In June 2004, there were still 325 HIV-positive soldiers on active duty [6]. One would like to know what decisions had been made with respect to new infections: for example, whether serial testing continued for these soldiers and, if so, whether some reverted to HIV-negative status; and, if so, whether it was with or without anti-retroviral treatment.

[10] Associated Press, Atlanta, Updated: 3:05 p.m. ET, 13 June 2005; http:// msnbc.msn.com/id/8203052/, accessed 3 August 2005.

[11] Centers for Disease Control and Prevention. (1987). *Morbidity and Mortality Weekly Report, 36*(49), 801–804.

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[13] Dennis H. Osmond, "Epidemiology of HIV/AIDS in the United States", HIV InSite Knowledge Base Chapter, March 2003. http://hivinsite.ucsf.edu/, accessed 3 August 2005.

[14] For three comprehensive discussions of these changes in definition and their import, see www.virusmyth.net/aids/index/definition.htm, accessed 6 August 2005.

[15] www.statehealthfacts.kff.org/, category "HIV/AIDS", sub-category "HIV Testing", accessed 6 August 2005.

[16] States not included (population in millions): CA (29.8), OR (2.8), WA (4.9), MT (0.8), IL (11.4), KY (3.7), HI (1.1), DE (0.7), DC (0.6), MD (4.8), MA (6), RI (1), NH (1.1), ME (1.2), VT (0.6); total population = 70.5.

[17] U.S. Census Bureau. (2001). *HIV/AIDS Surveillance Data Base Summary Tables*, revised December 2001. www.census.gov/ipc/www/hiv3lati.html, accessed February 2004.

[18] Embassy of the Republic of Haiti. (2003). *The Aids Crisis And Health Care*, 7 February 2003, Washington, DC.

[19] STI and STD are often used interchangeably, though "infection" and "disease" are not strictly the same thing.

[20] www.niaid.nih.gov/factsheets/stdinfo.htm, dated July 1999, accessed 15 June 2005.

[21] http://edcenter.med.cornell.edu/Pathophysiology_Cases/STDs/STD_06.html, accessed 28 May 2005.

[22] Within a given age range. F(HIV) changes with age differently for males than for females, but in all groups for which data are available—with the usual necessary caveats about chance fluctuations—the ratio of male to female F(HIV) at a given age is the same [2, 3, 6, 7, 16–18, 23, 24, 28, 40, 55, 56].

[23] Nilesh Chatterjee, "A community-based intervention with popular opinion leaders (cpol) to achieve syphilis elimination in two urban high morbidity areas in Texas", Centers for Disease Control and Prevention Award No. U65/CCU622268-01. http://cpol.tamu.edu, accessed 4 July 2005.

[24] Sonnabend recalls his efforts while accepting an award, at www.amfar.org/cgi-bin/iowa/amfar/record.html?record=22, accessed 27 July 2004. His activities are also described in Michael Callen. (1990). *Surviving AIDS*. HarperCollins.

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4. Centers for Disease Control and Prevention. (2001). *HIV counseling and testing in publicly funded sites—Annual report 1997 and 1998.*

5. Centers for Disease Control and Prevention. (2001). *HIV prevalence trends in selected populations in the United States—Results from national serosurveillance, 1993–1997.* 1–51.

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Appendix

Drawing and Comparing Maps

Comparing shaded maps, like those in this article, is subject to pitfalls, and it may deceive, unwittingly or deliberately. Therefore, a discussion is mandatory of how the maps in this article were prepared. However, I want to emphasize that the conclusions as to HIV drawn from these maps in this article do not depend on fine distinctions: the telling point is simply that there exists an unchanging East–West asymmetry.

Choice of the numbers that separate the data into discrete groups significantly affects the visual impression, and such a choice must always be made.

What should be done when drawing maps for groups in which the range of F(HIV) differs? For example, for 1993–97, among military recruits F(HIV) is less than 0.1% (Figure 7 [5]), whereas in the Job Corps the prevalence is as high as 0.5% (Figure 8 [5]). Should one of those figures be re-drawn, and if so, how? One might decide that they should be compared as is; or that one should be

re-drawn so that the numbers separating discrete groups are in the same proportion in both figures; or one might decide to have in both figures the same numbers of States in each of the discrete groups.

The question is moot in the several cases in which the published sources show numbers only for the ranges of shading and not for individual States—Figures 4, 9, and 10 for child-bearing women. Those use the heaviest shading for only 2 or 3 States and the second-heaviest shading for only 6, 9, and 5 States, respectively.

Where full numerical data were provided, I used the criterion that each figure should place the same number of States in each category. (Only the 48 mainland States were considered. Some sources report separately for Washington, DC, and some give data for Puerto Rico; both are almost always in the highest F(HIV) category.) The 5 mainland States highest in F(HIV) are blackened, the next 14–16 are heavily shaded, the next 16–18 are lightly shaded, and the remainder are left clear. (The ranges 14–16 and 16–18 reflect an unwillingness to place in separate shadings 2 States with equal magnitudes of F(HIV); where 3 or more States had the same F(HIV), assignments were made arbitrarily.)

All maps were re-drawn to use the same types of shading.

This issue of choices in drawing the maps adds a further caveat to the earlier one as to random fluctuations among samples. *Any* division of the States into discrete groups is somewhat arbitrary, and States differing but little in F(HIV) may differ significantly in shading. The differences shown for military applicants between 1985–86 (Figure 2—310,000 tests) and 1985–87 (Figure 3—1.25 million tests) illustrate how small sample sizes magnify the influence of chance fluctuations. F(HIV) (for low-risk groups) is on the order of ≤ 5 per 1000, and 310,000 represents not much more than 1 per 1000 of the population of the United States. Therefore, one cannot expect data from States with small populations to be reliable. As noted in the text, the States from which no HIV-positive applicants were recorded in these studies are indeed the States of low population; and correspondingly, the only States whose shading between these two figures differs by more than one unit do have small populations (Delaware and West Virginia).

One reasonable way to compare any two such maps is to count the numbers of States whose shadings differ. For example: the two successive years shown for child-bearing women (Figures 9 & 10) illustrate the expected effect of chance year-to-year fluctuations when sample sizes are about the same. Eight States have different shadings for those 2 years. From 1994 to 1995, 3 States—Kansas, Oregon, and Washington—moved from "0.05 to 0.19" into "0.2 to 0.39"; while 5 States—Georgia, New Hampshire, Minnesota, Virginia, and West Virginia—moved in the opposite direction. In all cases, a change as small as 0.005, or 3%, in the rate of F(HIV) could prompt such a switch—or it could have been a much greater change, of course. Unfortunately, these two maps had unequal numbers of States in the second-highest range of shading: 9 and 5 States, respectively. With equal numbers of States in each range, there might have been as many 12 changes between the two maps.

This comparison indicates that changes in as many as 12 of the 48 States—1 in 4, or 25% of States—should not be regarded as significant; particularly not, of course, when a roughly equal number of changes are in opposite directions, as in this case.

At any rate, the visual impression created by these comparisons should be treated as no more than semi-quantitative at best. But the point to be made here is primarily a qualitative and not a quantitative one. Under any manner of illustrating the data, it is clear that the distribution of F(HIV) is heavily weighted toward the East and South. That weighting is seen in the most recent data, just as it was in the earliest data. It is similarly so among quite disparate social groups: blood donors, child-bearing women, military applicants, Job Corps, clients at public testing sites. The conventional HIV/AIDS theory offers no explanation for it. Nor would one expect a sexually transmitted infection to have such an unvarying geographic distribution.

Maps of AIDS and Maps of HIV

These remarks about valid comparisons between maps are pertinent to an early claim [110] of quantitative correlation between HIV prevalence and AIDS cases. It was asserted that ''The geographic distribution of HIV prevalence in military applicants is similar to that for reported cases of AIDS with the adjusted prevalence of HIV in recruit applicants usually three to ten times as high as the cumulative incidence of reported AIDS (Fig. 2)''. Figure 19 is a reproduction of the cited ''Fig. 2''. (Figure 19A is for the same data as Figure 3, which was re-drawn in the manner discussed above, for comparable shading with the other figures in the main text.) But how similar are those two maps in Figure 19 really?

No details were given as to how Figure 19 was drawn, but the inserted numbers indicate that the five ranges of shading in each were chosen with cut-off numbers in ratios of 2. That resulted in very different numbers of States in each shading range in the two parts of the figure—see Table 2.

This sharpens the question, is there really a similarity of geographic distribution here?

First, bear in mind that any visual impression of similarity may be misleading.

Second, if one compares the shading of each State between the two maps, it turns out that fewer than half the States are in the same category of shading in both figures. (If the maps are re-drawn with comparable numbers of States in each category, with the same criteria as for other figures in the main text [except those for child-bearing women], again, only about half of the 48 mainland States are shaded similarly.) Recall from the earlier discussion that random variations may reasonably be assumed when as many as a quarter of the States are in different categories. (That criterion was based on only four categories of shading, whereas here there are five, which should allow a more accurate representation and fewer misleading, random, variations.) Therefore, fewer than half the States in the same category hardly indicates that the distributions are significantly similar.
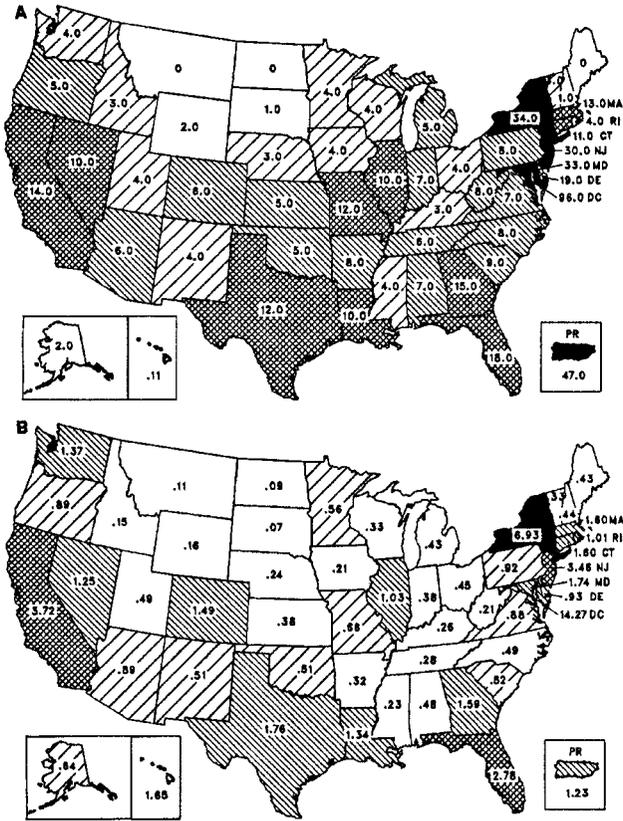
Fig. 19. Reproduced from [101]. Map A is for F(HIV) in military applicants, November 1985 to September 1987; map B is cumulative AIDS cases reported through 2 November 1987.

Third, a direct test of whether or not there is a linear correlation between the numbers in the two maps is to calculate their ratios. The results are shown in Table 3.

It would be a stretch to take this as a constant ratio with chance variations around a meaningful average. The actual mean is 11.2. The standard deviation,

TABLE 2
The Numbers of States in Each Shading Category of Figure 19A & B

Figure 19A ranges	Number of states in each range, 19A	Figure 19B ranges	Number of states in each range, 19B
≥20	5	≥4	2
10–20	11	2–4	3
5–10	15	1–2	13
2.5–5	13	0.5–1	11
≤2.5	8	≤0.5	23

TABLE 3
Ratio of HIV Prevalence (Figure 19A) and Cumulative Reported AIDS Cases (Figure 19B)

DC	38.2	CT	16.3	AZ	9.1	WI	6.8
PR	38.0	UT	14.6	CO	8.9	OH	6.7
NY	25.0	LA	14.3	OR	8.7	MS	6.7
MD	21.4	IL	13.2	TN	8.7	RI	6.5
NJ	20.4	SC	12.5	KS	8.2	ID	5.6
DE	20.0	AR	12.5	OK	8.1	NE	4.9
FL	19.0	NC	12.1	WA	8.0	VT	4.0
GA	19.0	WV	11.6	MI	8.0	KY	4.0
CA	18.4	PA	11.5	UT	7.8	WY	3.8
MA	18.2	IN	9.8	NM	7.5	AK	3.1
TX	17.4	AL	9.7	MN	7.1	SD	2.9
MO	17.3	VA	9.4	IA	6.9	NH	2.3

a common measure of how tightly numbers are clustered around the mean, is 8, which marks this more as a random scattering of numbers between 0 and 40 than as a normal "bell" curve centered at about 11—let alone somewhere between 3 and 10.

Fourth, this calculation exposes as false, the assertion that the numbers in each State are "usually three to ten times as high" for Figure A as for Figure B; only 25 out of 52 fall in that range (50 States plus Washington, DC and Puerto Rico). The median of about 9 does at least fall inside that range, although the average, 11, does not.

Doubts about the mathematical or statistical care with which this comparison was made are exacerbated by the patently unwarranted number of significant figures shown in map A: all numbers except ".11" for Hawaii end in ".0", yet it is hardly believable that the other 47 measurements (those not zero) were all within 0.05 of a whole number. (Stating 14.0, after all, means between 14.04 and 13.95. If such accuracy is not claimed, then there should be no decimal point followed by a "0".)

As noted in the main text, the geographic distribution of F(HIV) is the same for all sub-groups of the population. Thus, Figure 19A is representative not only of military applicants but also of blood donors (Figure 1), child-bearing women (Figures 4, 9, & 10), the Job Corps (Figures 5 & 8), and the average of public testing sites (Figure 11); in other words, it is a reasonable reflection of the general population. Thus, there was little if any geographic correlation between F(HIV) and AIDS cases up to November 1987. In more recent work, numerical comparisons have typically taken into account a presumed latent period of about 10 years between determination of HIV-positive status and development of AIDS. But this is moot when comparing maps of AIDS and of HIV, since the geographic distribution of HIV has not changed, as illustrated in the main text of this article.

The fact that the maps in Figure 19 were published as support for a correlation between HIV and AIDS is in itself a reason to doubt that there is a correlation.