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*REVIEW ARTICLE*

# Demographic Characteristics of HIV: III. Why Does HIV Discriminate by Race?

HENRY H. BAUER

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

**Abstract**—Racial ancestry influences the frequency of positive HIV-tests, F(HIV), as an independent variable. In every occupational, social, or other group tested in the United States, at all ages and for both sexes, F(HIV) increases in the order Asian → white → Native American → Hispanic → black. Data from South Africa display a similar sequence, white/Asian-Indian → Colored → black. This constant relation among the racial categories calls for an explanation in terms of genetic polymorphisms of the kind used in tracing human migration patterns. That Native Americans are closer to whites than to blacks also points to a physical cause and not a socioeconomic or behavioral one.

The relative circumstances of black and white Americans as to AIDS, by contrast to HIV ''infection'', serve as a further demonstration that HIV does not cause AIDS: Between 1981 and 2000, the ratio of black Americans to white Americans reported with AIDS trebled, while the ratio of positive HIV-tests in the two groups remained the same.

As shown in Part II of this series, F(HIV) signals a response to some sort of health challenge. The intensity of that response is evidently modified by genes associated with the immune system. Polymorphisms among these genes are well known and have been correlated with racial disparities in a variety of diseases. This view explains the peculiar and constant geographic distribution of F(HIV) in the United States as well as the extraordinary fact that Hispanics show characteristically different F(HIV) in the East and in the West. On the other hand, HIV/AIDS theory has no explanation for those, and official explanations for racial disparities as to HIV ''infection'' are plainly racist.

*Keywords:* HIV and race—HIV geography—racism and HIV/AIDS—human polymorphisms—physiological stress

## Introduction

The results of positive HIV-tests are often described as "the prevalence of HIV". This presupposes that "HIV tests" in fact detect a human immunodeficiency virus, HIV. But the commonly used tests are for *antibodies*, whose presence is merely *presumed* to indicate actual infection by HIV. To avoid confusion between the results of positive HIV-tests and the established presence of HIV, I use the term F(HIV) to denote the frequency of positive HIV-tests.

Part I of this series [1] pointed out that the distribution of F(HIV) in the United States has remained unchanged over two decades. That does not reflect a spreading epidemic of a sexual infection. Whatever the HIV tests measure, it is not a human immunodeficiency virus that entered the United States in the 1970s via New York, Los Angeles, and San Francisco to produce the AIDS outbreaks of the early 1980s among communities of drug users (IDU) and gay men (MSM) in those cities.

Part II [2] showed that F(HIV) tracks challenges to health in some fashion; it appears to be a rather non-specific indicator of physiological stress. That hypothesis explains the variation of F(HIV) between social groups and with age, sex, and population density. Those variations are, again, incompatible with the behavior of a sexually transmitted infection.

This Part III reviews the correlation between F(HIV) and racial category and argues that this, too, shows that HIV is not sexually transmitted. The correlation between F(HIV) and the racial categories used officially in the United States evidently reflects genomic polymorphisms like those used to track the course of human migrations over the last few hundred-thousand years: Genomic patterns that influence skin color are linked to genomic patterns that modify physiological responses to certain stresses. People of African ancestry display the strongest response and people of Asian ancestry the weakest.

This view readily explains yet another fact that the conventional view of HIV/AIDS cannot, namely, that the geographic distribution of F(HIV) in the United States shows a persistent weighting toward eastern and southern regions that has been evident for as long as there have been HIV tests. It also explains the even more curious fact that the level of F(HIV) among Hispanics in the western United States is comparable to that among whites whereas in the eastern United States it is comparable to that among blacks.

## HIV Discriminates by Race

All available data, from tens of millions of HIV tests on a disparate variety of sectors of the population of the United States, show that racial ancestry determines the relative level of F(HIV) as an independent variable—at all ages, in both sexes, and in groups presumed to be at low risk for AIDS or HIV infection as well as in those judged to be at high risk. As mentioned in Parts I and II, I found this, and the other regularities in F(HIV) data, so astonishing that

I consulted the Centers for Disease Control and Prevention, who responded, ''Your data 'regularities' appear to be true, and we agree that they are not 'artifacts'''[1].

I had been particularly taken aback that these data regularities seem almost quantitative with respect to race. F(HIV) is lowest among Asians, and about 50%–100% higher among white or Caucasian people. Among most Hispanic groups, it is 2 or 3 times higher than among white Americans, and among Native Americans it is between that for Hispanics and whites, rather closer to whites. F(HIV) is highest of all among black people, often 5 to 6 times higher than among white Americans, not infrequently more, rarely much less (see, for instance, [3–14]).

In general, possible reasons for variations by race include biological, cultural, geographic, and socioeconomic factors. I will argue that the evidence points to a purely physical explanation for these racial differentiations, that F(HIV) is associated with race in similar fashion as are such rudimentary attributes as skin color or hair texture or genes for sickle-cell anemia.

No one has shown, nor do I believe, that such genetic factors also determine risky sexual behavior or the sharing of needles. That, however, is the unavoidable implication of the conventional view of HIV/AIDS, for that view sees F(HIV) as the result of carelessly ''unsafe'' sexual contact, direct or indirect, with injecting drug users or men who have sex with men:

> Seroprevalence was substantially higher among blacks than among whites in nearly every serosurveillance population .... In the Western states, HIV seroprevalence was similar among Hispanics and whites, while in states along the Atlantic Coast, seroprevalence was higher among Hispanics than among whites. The marked racial and ethnic differences in HIV prevalence, even among persons treated in the same clinic, suggests that *both behavioral norms and complex social mixing patterns within racial and ethnic groups are important determinants of HIV transmission risk* (p. 37 in [15]; emphasis added).

These hand-waving generalities about behavioral norms and social mixing patterns cannot disguise that they are plainly racist, for their specific meaning is clearly this: Black Americans, and to a lesser degree Hispanics (but only in the East, not the West!), are supposed to be more intimately and constantly engaged in carelessly unsafe sex and sharing of infected needles than are whites or Asians or Native Americans. This is said to be accepted, standard behavior—''behavioral norms''. The ''complex social mixing patterns'' point to chains of sexual contact that pervade all social groups in black communities, so that HIV-infected drug users and gay men pass their infection on throughout all social groups in black communities—childbearing women, repeat donors of blood, Marines. In every sector of society, black people are supposed to behave like that on average 5 or 6 times as often as white people. Somehow, the black members of what are otherwise low-risk groups are supposed always to be contaminated to a significant extent by contact with members of high-risk groups. That is not only an absurd supposition, it also fails to explain why the

same racial disparities are seen within the high-risk groups themselves, among MSM and IDU.

## Influence of Demographic Variables

In Parts I [1] and II [2], I pointed out certain consequences of the fact that F(HIV) varies characteristically with age, race, sex, population density, and social group: Whenever comparisons are made between groups, or within groups over time, or between males and females, or in any other fashion, the precise effect of any one of these five variables could only be determined through a multivariate analysis, or by comparing groups that are matched with respect to all the other variables. Most of the published data do not satisfy these requirements, in fact I have not found any that do. Only a handful of studies report multivariate analyses, and even in these it is not certain that all the relevant variables were recognized and taken properly into account, since other studies were typically ignored. Therefore, one cannot expect precisely quantitative replication of any given observation when different social groups are compared, or when results are reported for a particular social sector for different periods of time; chance fluctuations must be expected. For example, the relative magnitude of F(HIV) among men and among women varies with age, and if this is not taken into account, incorrect conclusions could be drawn when groups are compared that are not precisely age-matched for each sex.

In the present focus on racial categories, these considerations mean that one should not expect to find exact ratios even if—to take an absurd hypothetical—there were an underlying cause as simple as Mendelian ratios[2] of 1-to-3 or 1-to-2-to-1. Even if there were a gene that, say, made F(HIV) in Caucasians 50% more frequent than in Asians, this exact proportion would not be shown in every tested group unless the compositions of those groups were matched by age, sex, geography, and social group. Since that is never the case, it is quite extraordinary that the racial disparities are so clear as to show up in virtually quantitative fashion in the great majority of studies. I have noted exceptions in only a few percent of the published reports, a proportion that can reasonably be regarded as the result of random fluctuations in the compositions of the samples. ''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 [13]); and the other two main racial groups, Asians and Native Americans, are also regularly ''in sequence'': F(HIV) among Asians is always lower than among whites, and for Native Americans it is between whites and Hispanics.

## Native Americans

Native Americans are an ideal probe for testing whether a trait is genetic or behavioral. In genetic ancestry, Native Americans are most closely related to Hispanic Americans, Asians, and Europeans. As to behavioral matters, they have

been discriminated against as much as have black Americans or any other minority, with attendant consequences in terms of poverty, demoralized youth, crime, and so forth.

Very few groups are large enough to report separately for Native Americans, since they make up less than 0.8% of the population[3]. In those few reports, F(HIV) among Native Americans falls closer to that among white Americans and Hispanics than to that for black Americans (Table 1; since column d is from so small a sample, it should probably be ignored, but it makes no difference to the argument).

Bearing in mind the caveats in the preceding section as to quantitative comparisons, the values for each racial category are remarkably consistent across these four disparate social groups. This consistency suggests that the racial disparities reflect *material* differences like skin color rather than behavioral differences. Applicants for military service are at least high-school graduates and self-screened for reasonably good health and fitness and against drug use; by almost complete contrast, the Job Corps accepts drug users among its intake of largely unemployed school drop-outs. The public testing sites cover a wide range, from family planning clinics through STD clinics to prisons; and the young MSM were sampled at such venues as dance clubs and bars, settings in which there tends to be a higher-than-average level of drug use and infectious disease.

It seems unlikely on the face of it that behavioral or cultural factors would produce the same racial correlations of behavior in such a variety of groups. But there is particularly strong evidence against a behavioral explanation in the circumstances of Native Americans. If these numbers reflected behavior—which would include the social, cultural, and economic consequences of discrimination

TABLE 1
Frequency of Positive HIV-tests in Native Americans Compared to Other Racial Categories

| | a Civilian applicants to military [5] | b Job Corps [13] | c From public sites [17–19] | d Young MSM [20] | Average of a–d | Average of a–c |
|---|---|---|---|---|---|---|
| Numbers of tests | 5,300,000 | 250,000 | 9,000,000 | 3,500 | | |
| Asian | 0.59 | 0.4 | 0.63 | 0.9 | 0.63 | 0.54 |
| White | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 |
| Native American | 1.47 | 1.6 | 1.23 | 2.0 | 1.6 | 1.43 |
| Hispanic | 2.25 | 1.6 | 2.37 | 2.1 | 2.1 | 2.1 |
| Black | 6.25 | 6.4 | 2.76 | 4.3 | 4.9 | 5.1 |

Note: For column (a), the ratios are the average of reported odds ratios, adjusted and unadjusted, since the adjusted ones are not necessarily more meaningful—assumptions are inherent in making the adjustments, and those assumptions were specific to the tested group; aggregated results from 1985–2000 [5] were used in preference to published data on military applicants for the smaller intervals 1993–97 [13] and 1991–92 [15]. For column (b), results aggregated for 1993–97 [13] were used rather than those for only a single year 1997 [16]. For column (c), the ratios were calculated by first summing the actual numbers of tests in each category for each of the four reported years 1995–98. MSM = gay men.

and deprivation—why would the behavior of Native Americans be closer to that of white Americans than to that of black Americans? ''Nearly half of Native American youth ages 12 to 17 will have tried an illicit drug, while only a quarter of blacks will have done so ... Over the years, the effect of substance abuse on American Indian/Alaska Native mental and physical health has been devastating. For the age group 25 to 34, American Indian males die almost three times more frequently than their non-Indian counterparts from motor vehicle crashes; they are twice as likely to commit suicide; they are seven times more likely to suffer from alcohol-related problems, such as cirrhosis of the liver''[4].

Native Americans also exceed black Americans in the rate at which they are victims of violent crime (Table 2).

Once again, as in Parts I [1] and II [2] on the basis of separate and independent considerations, the clear inference is that F(HIV) does not reflect a sexually transmitted infection. It is too constantly and uniformly associated with racial category; and within racial categories, it places Native Americans closer to socially favored groups than to other discriminated-against minorities.

## Asian Americans

In 2000, Asian Americans were 3.6% of the population of the United States[5], about 5 times more than Native Americans but still not large enough to yield separately reported data for F(HIV) in most samples.

Once again, it would be extraordinary if the relative tendency to risky sexual behavior among Asian Americans should so uniformly lead to a rate of infection significantly lower than among white Americans among groups that cover a wide range of socioeconomic circumstances (Table 1) and even as the average level of infection changes over nearly two orders of magnitude: It averaged about 0.1% among military cohorts ([5]—Table 1, column a) and not far from 10% among young MSM ([20]—Table 1, column d). Do gay men who happen to be Asian really practice ''safe sex'' more assiduously than gay white men, whereas gay Hispanics are twice as likely as gay whites to be ''unsafe'' and gay blacks more than 4 times more likely to be irresponsible? To offer a behavioral

TABLE 2
Native Americans as Victims of Violent Crime in Comparison to Other Racial Groups

| Racial category of victims | Violent victimizations per 1000 persons $\geq$12 years |
|---|---|
| Asian | 20.7 |
| White | 39.2 |
| Hispanic | 42.9 |
| Black | 48.9 |
| American Indian | 97.2 |

Note: From Table 4, ''Race and ethnicity of victims of violent crime ... 1993–2001'', *National Crime Victimization Survey, 1993–2001*, U.S. Department of Justice Special Report, September 2003, NCJ 194820; www.ojp.usdoj.gov/bjs/abstract/wuvc01.htm, accessed 26 June 2005.

explanation for these regularities would be not merely politically incorrect or racist, I suggest, it would be absurd.

## Black Americans

F(HIV) has been reported separately for black Americans in most reports. Table 3 summarizes the rates from many studies, relative to those of white Americans in the same groups.

The entries in Table 3 are arranged in decreasing size of the population samples, so the range of reliable values is no narrower than between 2.8 and 14; dropping the low outlier, 1.5 for college students, from the smallest sample of known size, would make no significant difference. Recalling once again the limitations in attempting to make quantitative comparisons among groups whose composition varies by age, sex, and geography, the numbers from all these different groups are quite consistent with one another, so much so that I calculated a standard deviation as noted in the Table.

But the precise numbers are not of primary interest, nor even whether they are exactly constant. What is certainly constant in all groups is that F(HIV) always follows the same sequence, Asian < white < Native American < Hispanic <

TABLE 3
Ratios of Frequency of Positive HIV-tests, F(HIV), for Black Americans Relative to White Americans Among Various Groups

| Group | Black-to-white ratio of F(HIV) | Numbers tested (in millions) | Sources |
|---|---|---|---|
| Childbearing women | 12.2 | 14.5 | [14] |
| Various public sites | 2.8 | >8 | [17–19] |
| Active Army | 3.8 | 7.7–0.17 | [3, 4, 21, 22] |
| Applicants for military service | 7.9 | 6.9–0.31 | [5, 6, 13, 16, 23, 24] |
| Blood donors | 14 | 2.2–0.82 | [25, 26] |
| Teenage applicants for military service | 4.7 | 1.1–0.044 | [7, 27, 28] |
| Navy | 4.3 | 1.1 | [8] |
| Army Reserve components | 6.4 | 0.68 | [29] |
| Job Corps | 5.1 | 0.36–0.14 | [10, 11, 13, 16, 30] |
| Marriage-license applicants | 4.3 | 0.1 | [31] |
| College students | 1.5 | 0.017 | [9] |
| National Health and Nutrition Survey | 4.0 | ? | [32] |
| Hospitals and outpatient clinics | 3.4 | ? | [13, 16, 33] |
| STD clinics | 2.8 | ? | [12, 34, 35] |
| Prisons | 5.5 | ? | [36] |
| IDU | 4.0 | ? | [37] |
| MSM | 2.7 | ? | [13, 20, 38–40] |
| Average | 5.5 | | |
| Standard deviation | 3.4 | | |

Note: No attempt was made to weight for numbers of tests in each sample, nor for variations in composition by age and sex (which were not usually reported in any case). The quality of methodology is likely to have been comparable in all cases since the tests were carried out under auspices of the Centers for Disease Control and Prevention or by the Army HIV Research Group or the American Red Cross. IDU = drug users; MSM = gay men.

black. The question is, do these racial disparities arise from behavioral differences or from physical or physiological ones? That the qualitative relationships are universal is in itself more than suggestive. That the relative values are semi-quantitatively constant adds further weight. But finally convincing is surely the fact that the same disparities are seen at all ages, for both sexes, and in all social groups and socioeconomic sectors of society. This is so unexpected for something supposedly infectious that illustrative examples are called for.

Within a given group, the racial differences are much the same at different ages ([4]—Figure 1, [10]—Figure 2), as well as for both sexes ([5]—Figure 3). This is particularly striking in the case of teenagers (Figure 2), because the male-to-female ratio changes significantly during the teenage years: As noted in Part II [2], it is often $<1$ among young teenagers but $>1$ at all other ages, both higher and lower.

Moreover, the same relationships are seen for *annual incidence* of new "infections" as for overall (cumulative) F(HIV) ([8]—Figure 4).

As already mentioned, even when the magnitude of F(HIV) increases by an order of magnitude, from a few per thousand (in the military or the Job Corps) to a few percent or more (among MSM), one continues to see the same relative proportions for Asian, white, Hispanic, and black Americans ([20]—Figure 5).

And again, with gay men just as with sailors, the incidence of new HIV-positive tests among HIV-negative men shows the same racial disparity. In a study of gay men during 1984–89, the overall rate of new "infections" for black Americans was 2.23 and that for Hispanics 1.77 greater than that for white Americans [41]. In another study of MSM aged 23–29 during 1998–2000, the incidence ratios were 5.88 and 1.4, respectively [20].

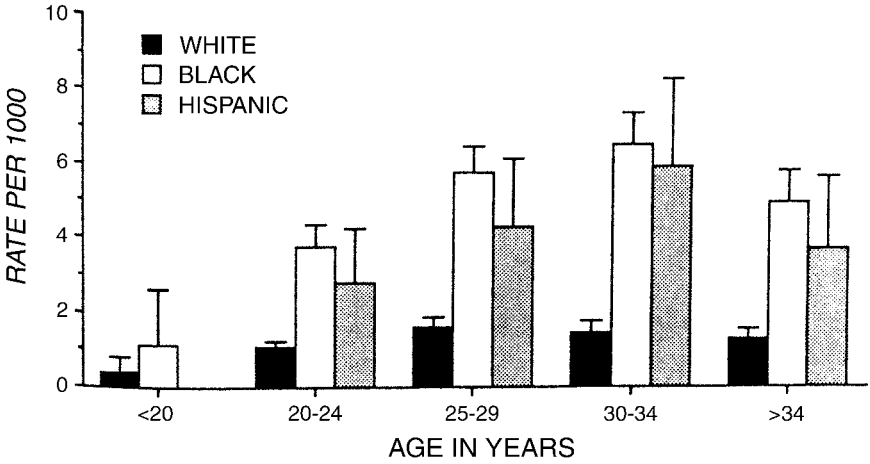


Fig. 1. The same racial disparities are seen at all ages; US Army personnel, 95% confidence interval bounds shown; HIV prevalence per 1000 [4].

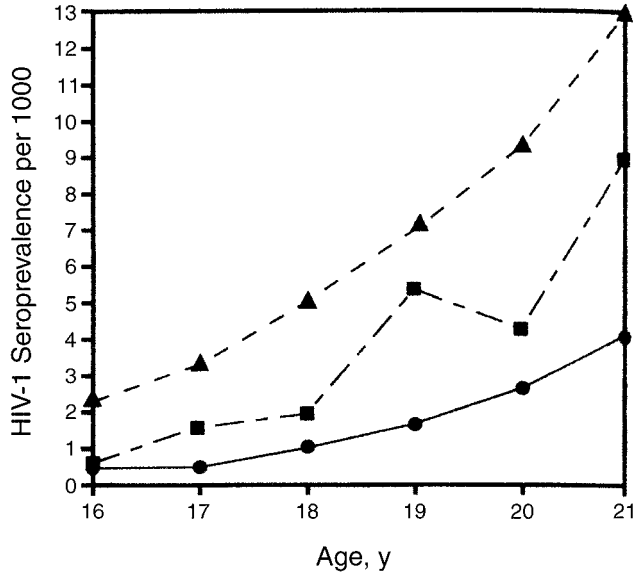


Fig. 2. The same racial disparities are seen even among teenagers [10]; ▲ = black, ■ = Hispanic, ● = white.

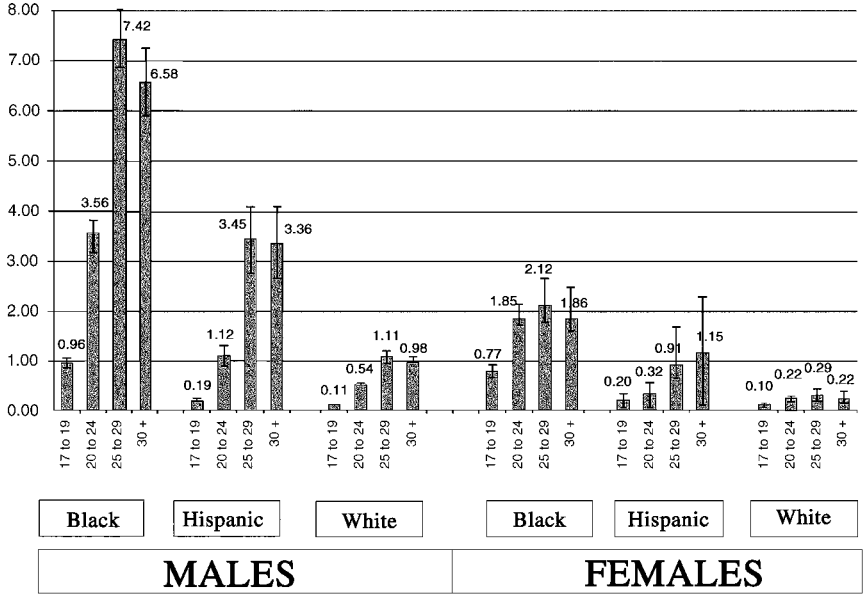


Fig. 3. The same racial disparities are seen at all ages in both sexes; civilian applicants for military service; 95% confidence intervals shown; HIV prevalence per 1000; ''other'' category omitted [5].

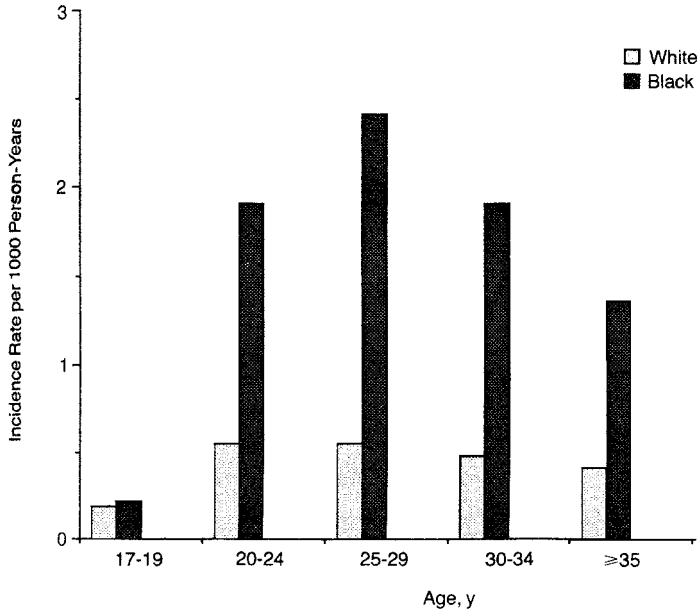


Fig. 4. The same racial disparities are seen at all ages for *annual incidence* of "HIV infection" just as for overall "prevalence", F(HIV); active-duty US Navy personnel, 1986; "other" and "all" categories omitted [8].

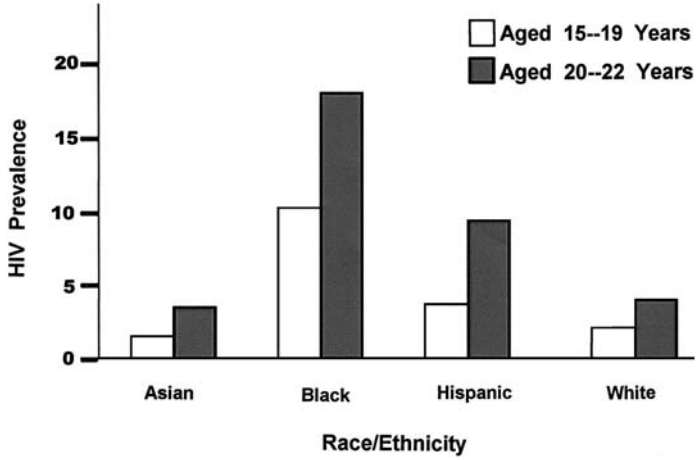


Fig. 5. The same racial disparities are seen even when F(HIV) is as high as a few percent to nearly 20%; young (15–22 years) MSM in seven cities, 1994–98; "mixed" and "total" categories omitted [20].

The F(HIV) ratios vary by race in the same manner for heterosexual men (Figure 6) as for MSM (Figure 7) even though the level of F(HIV) differs by an order of magnitude (all the tests were carried out at the same STD clinics [13]). The scales on the horizontal axes in these two Figures differ by a factor of 10.

These relations have remained the same during two decades even as the overall F(HIV) declined steadily ([23]—Figure 8).

The same pattern applies within each State [16]; for example, Figure 9 for childbearing women. This Figure also illustrates the usual geographic trend, described in detail in Part I: F(HIV) is significantly higher in the Atlantic Coast region than elsewhere.

This pattern among childbearing women was the same in each of the six years during which the survey was conducted (Table 4).

Examples could be multiplied *ad lib*:

- The Centers for Disease Control and Prevention noted in 1989 that the ''racial/ethnic disproportion is also observed in . . . blood donors, applicants for military service, and sentinel hospital patients''; among homosexual and bisexual men; among migrant farm workers; and in Belle Glade (FL). Among female prostitutes, F(HIV) was 15.4% for black and Hispanics, 6.7% for whites and ''others'' (ratio, 2.3); the ratio was 2.5 among those who admitted injecting drugs, 3.3 among those who did not [42].
- In the mid-1980s among 5000 MSM in Baltimore, Chicago, Los Angeles, and Pittsburgh, black men were infected 60%–80% more often than others [43].
- In 1991, the Centers for Disease Control and Prevention again reported F(HIV) of 1.9% for black, 1.0% for Hispanic, and 0.3% for white women who had no known risk factors (ratios black-to-white 6.3, Hispanic-to-white

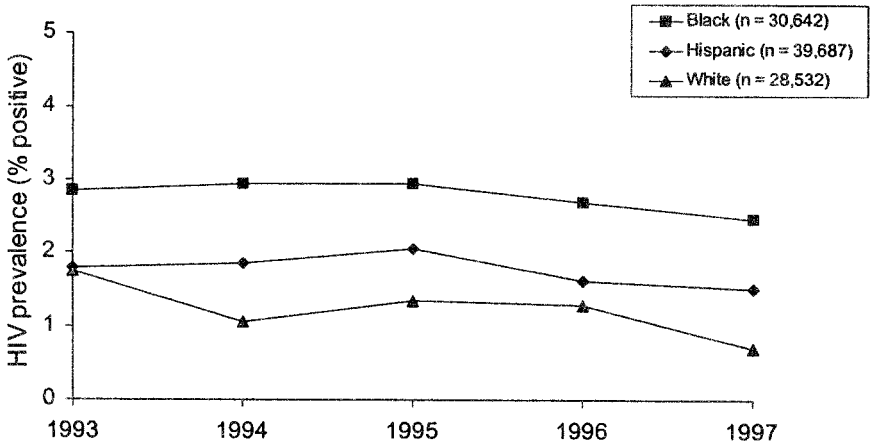


Fig. 6. The same racial disparities are seen among heterosexual patients at STD clinics, 1993–97 [13].

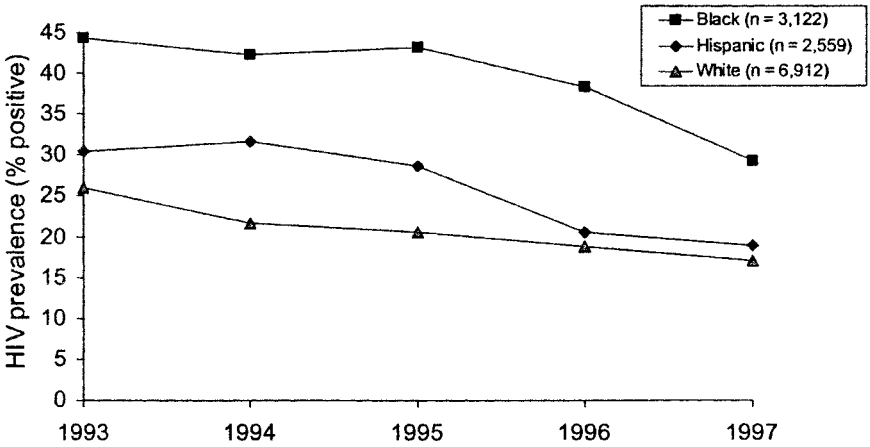


Fig. 7. The same racial disparities are seen among MSM at STD clinics, 1993–97 [13].

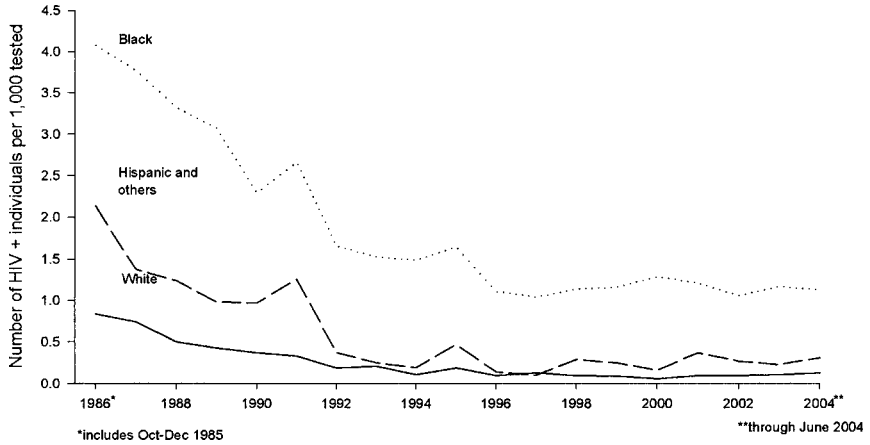


Fig. 8. The same racial disparities were seen among military applicants in every year for two decades (1985–2004), even as the overall magnitude of F(HIV) declined significantly [23].

3.3); among female IDU, the rates were 16.7%, 15%, and 3.8%, respectively (ratios B/W 4.4 , H/W 3.9); among female sex partners of "persons at risk", the rates were 8.2%, 3.6%, and 1.5%, respectively (ratios B/W 5.5, H/W 2.4) [44].

- The national surveillance summary [15] up to 1992 noted that black women were 3–28 times more likely than white women to be seropositive, and black IDU were nearly 5 times as likely to be HIV-positive as white IDU (18.4% as against 3.8%).
- A review of 92 studies [45] found F(HIV) among black IDU about 4 times greater than among white IDU (varying from 1+ to about 6 in different

TABLE 4
Ratio of Black-to-White Frequency of Positive HIV-tests, F(HIV), Among Childbearing Women

| | 1989 | 1990 | 1991 | 1992 | 1993 | 1994 | Average |
|---|---|---|---|---|---|---|---|
| AL | | 5.4 | 6.7 | 6.0 | 6.5 | 17.6 | 8.4 |
| CA | 7.4 | 13.2 | 7.3 | 13.2 | 11.5 | 8.1 | 10.1 |
| CT | 13.6 | 13.5 | 9.9 | 17.5 | 8.9 | 11.4 | 12.5 |
| FL | 10.6 | 10.8 | 9.3 | 7.7 | 13.3 | 16.0 | 8.7 |
| KS | | | 12.8 | 2.8 | 4.6 | 9.7 | 7.5 |
| MI | | 13.4 | 13.7 | 29.5 | 9.7 | 11.2 | 15.5 |
| MN | | 12.5 | 15.4 | 15.6 | 16.4 | 30.0 | 18 |
| MS | | 7.8 | 12.8 | 5.9 | 10.7 | 12.8 | 10 |
| NJ | | | 16.9 | 14.1 | 12.2 | 14.6 | 14.4 |
| NY | 15.7 | 16.2 | 15.1 | 17.0 | 19.0 | 21.5 | 17.4 |
| SC | | 17.2 | 11.9 | 14.3 | 11.1 | 8.4 | 12.6 |
| TN | 4.5 | 8.3 | 8.9 | 8.5 | 12.2 | 22.4 | 10.8 |
| TX | 7.3 | 9.8 | 10.8 | 12.3 | 11.2 | 18.5 | 11.6 |

Note: Data from [14].

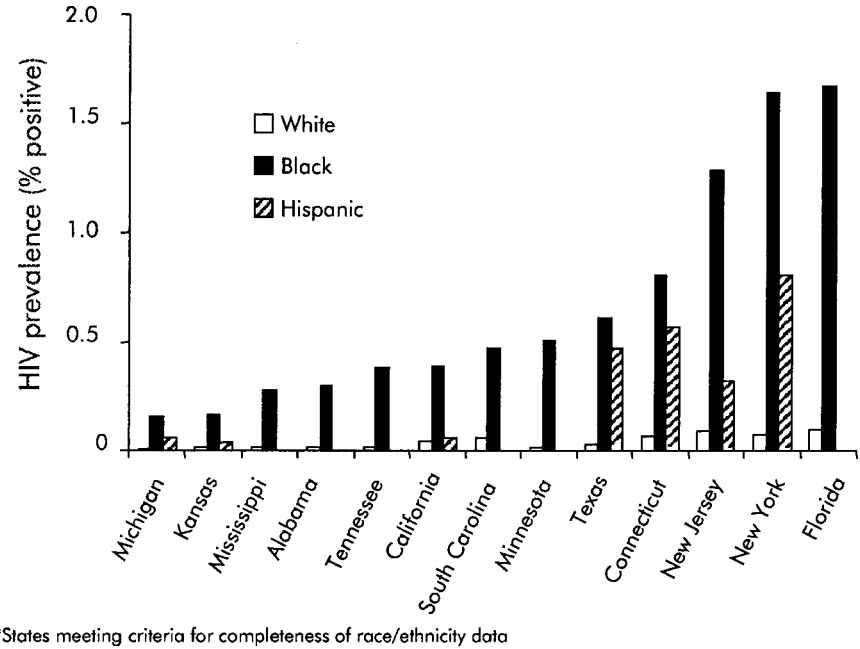


Fig. 9. The same racial disparities are seen in every State; childbearing women, 1994 [16].

cities), and among Hispanic IDU about 5.8 times greater than among whites (varying from 1+ to about 16).

IDU and MSM are the groups at highest risk[6], and thus indicted in the conventional view for practicing most assiduously the foolish and risky behaviors of needle-sharing and unsafe sex. Are we to believe that even within these communities of promiscuous and irresponsible people, black Americans exceed by a large factor the promiscuity and irresponsibility of white Americans? Surely it is abundantly obvious, that these relative levels of F(HIV) must reflect differences in physiological response to a given set of circumstances, not differences in chosen behavior.

### *HIV and AIDS*

The relative circumstances of black Americans as to HIV and as to AIDS constitute yet another demonstration that there is no connection between HIV and AIDS: The racial disparities between black and white Americans are quite different for F(HIV) and for AIDS.

The percentage of AIDS cases who are black has almost doubled from the first appearance of AIDS to the present time; it increased from 25.5% in 1981–87 to 31.2% in 1988–92, to 38% during 1993–95, and to 44.9% for 1996–2000 [46]. The percentage of AIDS cases who were white decreased correspondingly, from 59.7% to 50.4% to 42.4% to 34%. The ratio of black percentage to white

TABLE 5
Data for HIV From the Same Sources as in Table 3, Re-ordered Chronologically and Re-averaged as Required; AIDS Ratio Changes by Factor of 3, Frequency of Positive HIV-tests [F(HIV)] Ratio Does Not Change

| | Ratio of black-to-white cases of AIDS and F(HIV) | | |
|---|---|---|---|
| Year[a] | AIDS [46] | F(HIV) average (individual reports) | Sources for F(HIV) data |
| 1984 | 0.43 | | |
| 1985 | | | |
| 1986 | | 7.4 (15.5, 4.4, 2.3) | [3, 6, 26] |
| 1987 | | 4.4 (5.4, 4.3, 4.3, 4.2, 3.9) | [4, 7, 8, 12, 31] |
| 1988 | | 4.6 (6.6, 4, 3.1) | [28, 29, 37] |
| 1989 | | 3.3 (6, 4.4, 4.1, 2.6, 1.5, 1.3) | [9, 10, 21, 25, 35, 47] |
| 1990 | 0.62 | 5.5 (8.5, 4.1, 4) | [24, 30, 32] |
| 1992 | | 8.4 (12.2, 4.6) | [14, 22] |
| 1993 | | 5.9 (6.3, 5.5) | [5, 11] |
| 1994 | 0.90 | 1.7 | [34] |
| 1995 | | 4.6 (6.4, 2.7) | [16, 23] |
| 1996 | | 2.8 | [18] |
| 1997 | | 2.7 | [16] |
| 1998 | 1.32 | 4.7 (5.9, 5.5, 2.7) | [19, 36, 40] |
| 1999 | | 5.3 | [20] |

[a] Or mid-year of range.

percentage thus changed from 0.43 to 0.62 to 0.90 to 1.32, an overall factor of 3. In complete contrast, the ratio of F(HIV) among black Americans to that among white Americans shows no continuing trend (Table 5). There is no correlation between the data for HIV and the data for AIDS.

## Hispanic Americans

All the general considerations about black Americans apply also to Hispanics, but there is an additional point worth noting. A similar geographic pattern has been reported among Hispanics as for the overall population of the United States: F(HIV) among Hispanics is persistently highest in the Atlantic and southern regions and lowest in the north-central areas.

In the late 1980s [4], F(HIV) among Hispanic soldiers was 5.6 per 1000 for those from the East (NY, NJ, Puerto Rico) but only 2.2 for those from the West (AZ, CA, NM, TX). More generally, for several population groups (clinics, Job Corps, military), it was remarked that "In the Western states, HIV seroprevalence was similar among Hispanics and whites, while in states along the Atlantic Coast, seroprevalence was higher among Hispanics than among whites" (p. 37 in [15]). The same asymmetry is seen among Hispanic childbearing women; but not among black mothers (Table 6).

Now, the official classification of "Hispanic" does not parallel the others: It is ethnic, not racial. In some circumstances, the Census Bureau makes a distinction between black Hispanics and non-black Hispanics. If racial variations in F(HIV) reflect physical differences between the racial categories, that suffices to explain the strange, just-noted, geographic differentiation within the category of His-

TABLE 6
Ratio of Hispanic-to-White Frequency of Positive HIV-tests, F(HIV), Among Childbearing Women Is Vastly Different Across the United States, Whereas Black-to-White Ratio Is Almost Unchanged [14]

| | | | | | | | | Regional averages | |
|---|---|---|---|---|---|---|---|---|---|
| | 1989 | 1990 | 1991 | 1992 | 1993 | 1994 | State average | Hispanic-to-white | Black-to-white[a] |
| NY | 11.1 | 10.3 | 7.6 | 8.7 | 10.0 | 10.7 | 9.7 | | |
| NJ | | | 7.6 | 4.9 | 3.2 | 3.6 | 4.8 | | |
| CT | 6.8 | 6.9 | 4.8 | 9.9 | 4.0 | 8.0 | 6.8 | | |
| | | | Atlantic Coast | | | | | 7.1 | 14.8 |
| KS | | | 5.1 | | 3.1 | 2.4 | 3.6 | | |
| MI | | 4.6 | 4.1 | 8.6 | 3.1 | 4.6 | 5.0 | | |
| | | | Mid-West | | | | | 4.3 | 11.5 |
| CA | 1.3 | 1.3 | 0.7 | 1.3 | 2.3 | 1.2 | 1.4 | | |
| TX | 1.0 | 0.7 | 0.8 | 1.0 | 1.0 | 1.4 | 1.0 | | |
| | | | West | | | | | 1.2 | 10.9 |

[a] From Table 4.

panics: In the West, ''Hispanics'' are largely of Mexican ancestry, with little recent African ancestry, whereas in the East a high proportion of ''Hispanics'' are from the Caribbean and have on average a significant proportion of relatively recent African ancestry.

Why is there any variation at all in the black-to-white ratio in different regions of the country? Under the present interpretation, HIV-positive is a response to stress. Background causes of environmental stresses are likely to vary with geography because of differences in climate, say. As noted in Part II [2], population density certainly exerts an influence. In fact, F(HIV) among white women who have just given birth does show significant regional differences [14]: lowest in the Mid-West (about 0.15/1000 in KS, MI, MN), highest in FL (1.6/1000), almost as high in the North-East (about 1/1000 in CT, NJ, NY), and intermediate in AL, MS, SC, TN (0.3/1000) and CA, TX (0.4/1000). In part, these apparently regional differences may be owing to differences in average age of the mothers, however, or differences in medications or in frequency of drug abuse. At any rate, there is an overall ten-fold regional variation of F(HIV) within the category of white women, which dwarfs the 10–15 range for the black-to-white ratio. Such a minor range would be readily explicable: If the F(HIV) responses to stress are not linear with respect to the amount of stress, then the black-to-white ratio will be different at different levels of stress.

However, the six-fold variation in the Hispanic-to-white ratio cannot be explained away in this fashion. For Hispanics, then, geography—or racial ancestry—is an additional factor, beyond age and sex, that can give rise to chance fluctuations when F(HIV) is compared between groups. It is to be expected, therefore, that the observed, unadjusted, ratio of F(HIV) for ''Hispanics'' to that of Asians, whites, Native Americans, and blacks will be less constant than the ratios within those other four groups. That is indeed the case (Table 7): The standard deviation of 2.0 on an average of 2.8 reflects a greater variability than in the black-to-white ratios in Table 3.

## Biology and Sociobiology

Centuries of controversy surround questions of the degree to which mental, emotional, or behavioral characteristics may be ''instinctive'', influenced significantly by an individual's genetic constitution. If someone chooses to believe that people whose skin happens to be black are always, in every social setting or group, more promiscuous and reckless than whites; and that Hispanics behave in that manner more than whites but less than blacks; and that Asians are always and everywhere less promiscuous or reckless than any other group—nothing one can say is likely to shake that belief. But it is without evidential foundation: ''Prof Mhlongo pointed out that the data presented could be interpreted as suggesting that the HI virus is highly selective in terms of race. The high prevalence of HIV positivity in the black population of South Africa would therefore have to imply that black people were more promiscuous than white

TABLE 7
Ratios of Frequency of Positive HIV-tests, F(HIV), for Hispanics Relative to White Americans Among Various Groups

| Group | Hispanic-to-white ratio of F(HIV) | Sources |
|---|---|---|
| Blood donors | 8.8 | [25, 26] |
| College students | 1.6 | [9] |
| Childbearing women | 4.6 | [14] |
| Active Army | 2.9 | [3, 4, 22] |
| Applicants | 2.3 | [5, 6, 13, 16, 23, 24] |
| Teenage applicants | 1.5 | [7, 27, 28] |
| Reserve components | 4.5 | [29] |
| Various public sites | 2.4 | [17–19] |
| National Health and Nutrition Survey | 1.33 | [32] |
| Job Corps | 1.7 | [10, 11, 13, 16, 30] |
| Hospitals and outpatient clinics | 1.4 | [13, 16] |
| STD clinics | 1.2 | [34, 35] |
| Prisons | 3.4 | [36] |
| MSM | 2.0 | [13, 20, 38, 40] |
| Average | 2.8 | |
| Standard deviation | 2.0 | |

Note: As in Table 3, the ratios reported in the various sources were averaged without attempting to weight for numbers of tests in each sample. The quality of methodology is likely to have been comparable in all cases since the tests were carried out under auspices of the Centers for Disease Control and Prevention or by the Army HIV Research Group or the American Red Cross. MSM = gay men.

people. He went on to point out that there is no evidence to support such a conclusion'' (p. 31 in [48]).

Under the standard view of HIV/AIDS, the data on F(HIV) would also require one to believe that Hispanics in the western United States, who come largely from Mexico, behave in sexual and drug-related matters much like white Americans, whereas in the eastern United States they behave much like black Americans. Why would that be? Because so many of them come from the Caribbean? Is needle-sharing and sexual promiscuity an hereditary cultural commonality in those places, by contrast with Mexico?

If behavioral variables exhibited the regular trends shown by F(HIV), then the social sciences would long ago have become mathematical ''hard'' sciences. Even the most confirmed sociobiologist might hesitate to suggest that risky sexual behavior and the sharing of needles for injecting illegal drugs is always several times more common among black people than among white, irrespective what group they belong to: repeat blood donors, Marines, soldiers, Job Corps, MSM, childbearing women, prisoners, drug abusers in treatment centers, or those attending public clinics for adolescents or for family planning.

As a matter of actual fact, research in the context of HIV/AIDS has failed to find racial differences in sexual behavior. Among drug users, no significant differences in behavior by race were found as to number of sexual partners, frequency of intercourse, number of sexual partners who were IDUs, number of non-IDU sexual partners, prostitution, or intercourse with people then or later

diagnosed as AIDS [49]. Samuel and Winkelstein [50] found no significant racial differences in behavior among gay men in San Francisco and concluded that the black-to-white ratio of F(HIV) could not be explained by differences in major risk factors. The San Francisco Department of Health [51] found no differences between races as to anal intercourse, measured via the incidence of rectal gonorrhea. Bausell et al. [52] found white Americans ***less*** likely to take protective measures during sex than black Americans.

The interpretation seems clearly indicated, that F(HIV) has something to do with deep-seated genetic patterns as ancient as those commonly used to distinguish people as ''Caucasian'', ''African'', ''Asian'' (or ''Mongoloid''), and so on. But F(HIV) is not simply like skin color or hair texture: It also varies greatly with differing states of overall health, as shown in Part II [2]. F(HIV)—the prevalence (or possibly the strength) of antibody reactions that are assumed to detect HIV—seems to be an indicator of physiological stress; deep-seated ''racial'' genomic patterns modify the physiological response to a variety of health challenges.

## Race and Genomes

Cavalli-Sforza [53, 54] was a pioneer in showing that human migration patterns for the last 200,000 years or so can be traced using mutations accumulated in the human genome. Remarkably and controversially, the patterns so derived seem to be consistent with those derived on the entirely independent grounds of historical linguistics, which infers how human languages branched as peoples moved and lost contact with one another—''controversially'' because the time scale for language differentiation is vastly shorter than the time scale of migrations and physical evolution. However, the chief claim, that the human genome contains certain patterns that parallel conventional racial classifications, has found general acceptance. DNAPrint Genomics offers ancestry-tracing services based on what they call ''Ancestry Informative Markers''[7]. Medical research has begun to take into account health-related racial differences and to consider the possibility of drugs that are more effective in some racial groups than in others: BiDil was approved by the Food and Drug Administration in June 2005 after tests specifically on African Americans, who had failed to benefit fully from existing medications for heart disease[8]; lower doses of statins are recommended for Asians than for other racial groups[9].

Presumably because well-known evils and tragedies in human history have been associated with racial pseudo-science and abusive racial stereotyping, the discussion of ***any*** real distinctions between human ''races'' has been hampered. But as to HIV/AIDS, a recognition of physical correlates of ''races'' would be the very opposite of racist, in direct contrast to the inescapably racist implications of the theory that HIV is sexually transmitted.

A great brouhaha erupted in South Africa in 2004 when it became public knowledge that the National Blood Service (SANBS) was not using blood donated by black donors for transfusions; such blood was categorized as ''high

risk'' because ''the average risk of a black South African being HIV-positive was 100 percent greater than a white South African and, depending on the specific group, could be about 150 percent higher'' [55]; ''white females who donate blood regularly (at least once a year) had an HIV infection rate of three in 113,000 units donated. In contrast, . . . black females who donate regularly have an HIV infection rate of 90 in 7500 units of donated blood'' [56].

The SANBS of course was interpreting F(HIV) as the presence of a virus that causes AIDS and that is transmitted through blood as well as by needle-sharing and unsafe sex. Their data included the relative levels of F(HIV) among blood donors from different racial groups (Table 8).

The same sequence had been reported for women at prenatal clinics in 1990 by the South African Department of National Health ([58]—Table 9).

Although these several sources differ on the precise ratio of black-to-white F(HIV), they are consistent in several respects with data from the United States:

- F(HIV) is highest in blacks and lowest in whites.
- Within each racial category, it is lower for repeat donors than for first-time donors.
- For white repeat donors, the South African figure of 1.12 [57] or 2.7 [56] per 100,000 is in the same ball-park as the US figures for 2002 [59], age-averaged at 1 per 100,000 (highest being 3.7 for 20–29-year-olds and less at both greater and smaller ages).
- F(HIV) for pregnant women (Table 9) is significantly higher than for blood donors (Table 8).

TABLE 8
Frequency of Positive HIV-tests, F(HIV), Among Blood Donors in South Africa, by Racial Classification [57]

| Classification by South African National Blood Service | Racial categories of blood donors | F(HIV) per 100,000 |
|---|---|---|
| Category 1 (''safe'') | Regular donors (all white or Indian) | 1.12 |
| Category 2 | Coloreds, & first-time Indian and white | 2.2 |
| Category 3 | First-time coloreds, & blacks (not first-time) | 25.8 |
| Category 4 | Blacks (first-time) | 58.97 |

Note: ''Colored'' is the South African term for mixed ancestry.

TABLE 9
Frequency of Positive HIV-tests, F(HIV), Among Women at Prenatal Clinics, 1990, South African Department of National Health [58]

| Racial category | F(HIV) per 1000 |
|---|---|
| White | 0.6 |
| Colored | 1.6 |
| Black | 8.9 |

All this fits the view that F(HIV) is a physiological response to stress whose strength is modified by somatic race-associated factors.

As already noted, this view affords an explanation for the East-over-West prevalence of F(HIV) among Hispanics in the United States; it is consonant with their respective ancestries: ''Carribean [sic] Hispanics tend to show significant European, Native American and African admixture. Non-African Hispanics tend to show relatively even European/Native American admixture with some showing more (even all) European, and others more (even all) Native American'' [60]. Since F(HIV) is highest among (''black'') people of African ancestry, Caribbean Hispanics show a higher level of F(HIV) than do non-Caribbean Hispanics. For example, the average Puerto Rican ancestry includes 5 or 6 times more African heritage than does the average Mexican ancestry (Table 10).

These genetic markers indicate that contemporary Native Americans share something like 30% or 40% European ancestry; again this is consonant with the F(HIV) ratios (Table 1).

So are the data in Table 8. The ratio of black-to-white F(HIV) among South African blood donors is about 25, larger than that (about 14, Table 3) for blood donors in the United States. That is consonant with the present hypothesis since ''African Americans'' on average share 14% European ancestry and some Asian, while ''European Americans'' on average share some African ancestry (Table 10); in South Africa, there has been less racial mixing—or, rather, the slightest sign of mixing shifted a person into the ''colored'' group.

That the Indian category in South Africa is similar to their ''white'' category is also consistent with the last two rows in Table 10.

The dark skins of Africans have a known physiological function. Sunlight absorbed through the skin catalyzes the formation of vitamin D, which is essential, but in too large amounts it is also harmful. In equatorial regions, unfiltered sunshine produces too much vitamin D. Humans evolved in Africa with (presumably) the optimum degree of sunshine-filtering by the skin. As humans

TABLE 10
Ancestral Connections to Four ''Racial'' Sets of Genetic Markers, as Percentages

| Ethnic group | European | Sub-Saharan African | East Asian | Native American |
|---|---|---|---|---|
| Mexican | 43 | 6 | 4 | 47 |
| Puerto Rican | 55 | 33 | 4 | 9 |
| American Indian[a] | 29 | 2 | 8 | 61 |
| American Indian[b] | 42 | 4 | 7 | 48 |
| African American | 14 | 80 | 3 | 3 |
| European American | 90 | 3 | 3 | 4 |
| South Asian Indian | 59 | 5 | 27 | 9 |

Note: From AncestryByDNA©; www.ancestrybydna.com/Ethnicities.asp, accessed 25 June 2005 (numbers rounded and standard deviations omitted).

[a] Includes individuals from US Government recognized tribes only (Sioux, Cheyenne, Cherokee, Arapaho).

[b] Includes individuals from US Government recognized tribes and unrecognized tribes.

migrated out of Africa into northern and temperate regions, it became necessary to absorb more of the incident sunshine in order to manufacture sufficient vitamin D, and the average skin color lightened. As a corollary, dark-skinned people who now live in temperate regions may be at risk for vitamin D deficiency: ''black race had a strong association with vitamin D deficiency . . . black women are at higher risk than are white women'' [61].

An analogous albeit speculative chain of reasoning can be constructed relating to immune function. Tropical regions harbor a great variety of endemic bacterial, fungal, parasitic, and viral diseases—great enough to warrant special Departments of Tropical Medicine in many places, as well as a Royal Society of Tropical Medicine and Hygiene. It would therefore be curious if humans, evolving in Africa, had not acquired very strong immune responses against that wide range of challenges to health. As humans migrated to other, non-tropical parts of the world where the immune system is less fiercely challenged, it seems reasonable that the responses generated by the immune system would have become modified, less wide-ranging or somewhat weaker. In Part II [2] of this series of articles, F(HIV) was shown to behave like a response to a physiological challenge. So it would be reasonable to expect that response to be stronger in people of relatively recent African ancestry than in people whose ancestors migrated out of Africa about 200,000 years ago, whose immune systems had evolved more or less in tandem with the hue of their skin. One piece of evidence to support this reasoning is that ''A unique African HLA haplotype may identify a population at increased risk for kidney graft rejection [*in other words, an exceptionally strong immune response*] . . . . Unique HLA alleles and MHC haplotypes have been identified in the Cape Colored and in the black South African populations . . . . HLA haplotypes are inherited 'en-bloc' as ancestral haplotypes that vary considerably between races'' [62].

These HLA genes associated with the immune system may well be particularly relevant to the present concerns. Much work has been done on the relation between race and HLA genes and the consequences in terms of illness. A search for ''HLA race'' in the database PubMed in August 2005 retrieved 2487 citations. A review in the context of human migration and evolution remarked that ''the combination *A1B8DR3* . . . is relatively common in Northern Europe. It perhaps represents a type that was present in Mesolithic, pre-Neolithic populations . . . [and] is . . . an extraordinarily good marker of European migration to other parts of the world, . . . inevitably found at a relatively high frequency . . . in Australia, Canada, and the United States . . . . The combination is not at all common in southern Europe. *A1* alone is not only a marker for Europeans, but is also found almost uniquely in all Caucasoid populations, including those in India. It is possible to find other combinations that are, for example, distinctive of African populations or Oriental populations, and the data clearly show that these haplotype distributions are the most distinctive HLA frequency markers for characterising [sic] different human populations'' (p. 180 in [63]).

That a propensity to test HIV-positive is characteristic of tropical regions is also consistent with the manner in which the average prevalence of HIV varies around the world (Table 11).

Would it not be quite extraordinary for a sexually transmitted infection to lodge and create havoc in Sub-Saharan Africa, for 25 years or so, without seeping even into North Africa and the Middle East? Particularly an infection that caused deaths that were first noted in the United States and not in Africa? Moreover, deaths difficult to overlook, since they result from an unusual number of otherwise very rare opportunistic illnesses?

Note—and far from by the way—that these estimates from UNAIDS reflect no significant change over these four years. All the slight differences are considerably smaller than the ranges of uncertainty. If anything, there has been a decline in prevalence in Sub-Saharan Africa and in the Caribbean.

## Geography of F(HIV) in the United States

In Part I [1], a persistent geographic weighting of F(HIV) in the United States toward the East and South was noted. It has often been remarked on, but no explanation has been offered under the official theory that HIV is a sexually transmitted infection, and it is difficult to conceive of one under that theory. On the other hand, this well-attested asymmetry follows rather obviously from the dependence of F(HIV) on race, discussed above, and on population density, reported in Part II [2].

The two variables of race and population density were combined in the following manner to calculate relative magnitudes of F(HIV) to be expected in each State if those two variables are all that matters:

The variation of F(HIV) by race was taken to be in the ratio of 0.65 to 1 to 1.5 for Asian, white, and Native Americans (see Table 1) to 2.8 for Hispanics (see

TABLE 11
Prevalence of HIV Among Adults Around the World

| Region | F(HIV), % 2001 [64] | F(HIV), % 2003 [64] | F(HIV), % 2003 [65] | F(HIV), % 2005 [65] |
|---|---|---|---|---|
| World | 1.0 (0.9–1.1) | 1.1 (1.0–1.2) | 1.1 (1.0–1.2) | 1.1 (1.0–1.3) |
| Sub-Saharan Africa | 7.6 (7.0–8.5) | 7.5 (6.9–8.3) | 7.3 (6.7–8.1) | 7.2 (6.6–8.0) |
| Caribbean | 2.2 (1.5–3.5) | 2.3 (1.4–4.1) | 1.6 (1.1–2.7) | 1.6 (1.1–2.7) |
| Latin America | 0.5 (0.4–0.7) | 0.6 (0.5–0.8) | 0.6 (0.4–0.8) | 0.6 (0.5–0.8) |
| North America | 0.6 (0.3–1.0) | 0.6 (0.3–1.0) | 0.7 (0.3–1.1) | 0.7 (0.4–1.1) |
| South & South-East Asia | 0.6 (0.4–0.9) | 0.6 (0.4–0.9) | 0.6 (0.4–0.9) | 0.7 (0.4–1.0) |
| Central Asia & Eastern Europe | 0.4 (0.3–0.6) | 0.6 (0.4–0.9) | 0.7 (0.4–1.3) | 0.9 (0.6–1.0) |
| Western Europe | 0.3 (0.2–0.4) | 0.3 (0.2–0.4) | 0.3 (0.2–0.4) | 0.3 (0.2–0.4) |
| North Africa & Middle East | 0.2 (0.1–0.5) | 0.2 (0.1–0.6) | 0.2 (0.1–0.7) | 0.2 (0.1–0.7) |
| Oceania | 0.2 (0.1–0.3) | 0.2 (0.1–0.3) | 0.4 (0.2–0.6) | 0.5 (0.2–0.7) |
| East Asia | 0.1 (0.1–0.2) | 0.1 (0.1–0.2) | 0.1 (0.04–0.1) | 0.1 (0.05–0.2) |

Note: Data from United Nations HIV/AIDS program [64, 65].

Table 7) to 5.5 for black Americans (see Table 3). For each State, a number R was calculated by weighting in these ratios the number of people in each racial category, as reported in the 2000 Census[10]. To take account of population density, the relative levels of F(HIV) were taken to be 1 in rural areas, 4 in cities over 1 million, and 2 for in-between areas (see Part II [2]). For each State, a number D was calculated by weighting the urban, rural, and in-between populations in these ratios, using data from the Demographia databases[11] and the Census 2000 figures for total populations in each State. The product RD yields the ranking of States shown in Table 12.

This ranking should be compared to actual data from the general and generally healthy population. The nearest available such data are from applicants for military service, 1985–2000 [5], in which unfortunately the geographic distribution is given only by regions, not by individual States. Table 13 compares the odds ratios for F(HIV) in that source with the averaged RD values from Table 12 for the States in each of those regions; and the RD values for the four States mentioned in the cited source as those with the highest F(HIV).

The agreement seems satisfactory, bearing in mind how simple-minded is the basis for the RD calculation and how rudimentary and approximate the calculation of population densities. Note particularly that neither race alone (R) nor population density (D) alone is as good a match to the actual F(HIV) as the product RD is: As Table 12 shows, neither R nor D ranks States in the same order as RD does. It is the *combination* of population density (health challenge) and inherent racially modified response or capacity that matches the facts rather well.

Another way to make this comparison is by maps shaded for the different levels of F(HIV), as in Part I [1]. There, ten maps (Figures 1–5 and 7–11) for various periods of time displayed the geographic distribution of F(HIV) for military applicants, people tested at public sites, blood donors, and members of the Job Corps. They were all quite similar (with the usual caveat concerning random fluctuations from sample to sample). Here, Figure 10 displays the geographic distribution that is averaged from the ten maps in Part I [1]. Figure 11 is drawn from the calculated numbers in Table 12. In both cases, the most heavily shaded States number 6, the next two groups 16 each, and the unshaded group 10.

Again the agreement is quite good. Thirty-one States are shaded the same in both Figures, eight States are more heavily shaded by 1 unit in one of the Figures, and nine are more heavily shaded by one unit in the other Figure. That 2/3 of the States are shaded the same, and that equal numbers of States differ in opposite directions in shading, and by only one unit, represents satisfactory agreement under this type of comparison, bearing in mind that the grouping into four categories is arbitrary, as discussed at greater length in the Appendix in Part I [1].

One could refine the calculation by taking into account the fact that F(HIV) for Hispanics differs from West to East, as discussed earlier. The calculation of population density in each State is also very crude and could be refined. But such

TABLE 12
Relative Values of Frequency of Positive HIV-tests, F(HIV), Expected if Race and Population Density are the Only Determining Factors

| | R from race ratios[a] | D from population densities[a] | Calculated relative F(HIV) RD/10[a] |
|---|---|---|---|
| DC | 380 | 27.6 | 1050 |
| NY | 190 | 4.1 | 80 |
| RI | 130 | 4.6 | 61 |
| MD | 230 | 2.7 | 61 |
| GA | 240 | 2.6 | 61 |
| IL | 190 | 3.2 | 60 |
| PR | 290 | 2.0 | 59 |
| CA | 180 | 3.3 | 59 |
| TX | 210 | 2.7 | 56 |
| FL | 190 | 2.9 | 56 |
| LA | 250 | 2.2 | 54 |
| NV | 160 | 3.2 | 52 |
| AZ | 160 | 3.0 | 48 |
| PA | 150 | 2.9 | 43 |
| MA | 130 | 3.2 | 42 |
| MI | 170 | 2.5 | 42 |
| VA | 190 | 2.1 | 41 |
| CO | 150 | 2.8 | 40 |
| OH | 150 | 2.6 | 39 |
| MS | 270 | 1.5 | 39 |
| SC | 240 | 1.6 | 38 |
| KS | 140 | 2.7 | 37 |
| MO | 150 | 2.4 | 37 |
| NJ | 180 | 1.9 | 35 |
| DE | 190 | 1.8 | 35 |
| WA | 120 | 2.7 | 34 |
| AL | 220 | 1.6 | 34 |
| NC | 200 | 1.6 | 33 |
| NM | 180 | 1.8 | 32 |
| OR | 120 | 2.7 | 32 |
| MN | 120 | 2.7 | 32 |
| IN | 140 | 2.1 | 30 |
| CT | 160 | 1.9 | 29 |
| TN | 180 | 1.6 | 29 |
| WI | 130 | 2.2 | 28 |
| OK | 160 | 1.7 | 27 |
| AR | 180 | 1.5 | 27 |
| UT | 120 | 1.9 | 22 |
| NE | 130 | 1.7 | 21 |
| KY | 130 | 1.6 | 21 |
| AK | 120 | 1.7 | 20 |
| ID | 110 | 1.7 | 19 |
| WY | 120 | 1.7 | 19 |
| IA | 110 | 1.6 | 18 |
| NH | 110 | 1.6 | 17 |
| WV | 120 | 1.5 | 17 |
| ND | 110 | 1.6 | 17 |
| HI | 90 | 1.9 | 17 |
| SD | 110 | 1.5 | 16 |
| MT | 110 | 1.5 | 16 |
| ME | 100 | 1.4 | 14 |
| VT | 100 | 1.4 | 14 |

Note: See text for how R and D were calculated.
[a] Rounded for easier viewing; no rounding was used in calculating RD.

TABLE 13
Comparison of Calculated and Actual Relative Values of Frequency of Positive HIV-tests, F(HIV)

| States highest in F(HIV) | Actual F(HIV) in highest States | Calculated (Table 8) | R alone highest States | D alone highest States |
|---|---|---|---|---|
| DC | 8.7 | 1050 | 380 | 28 |
| PR | 3.6 | 59 | 290 | 2.0 |
| NY, NJ | 2.2 | 57.5 (80, 35) | 185 (190, 180) | 3.0 (4.1, 1.9) |
| Other States ranked by regions | | Odds ratios for F(HIV) in reported regions | Average for regions | Average for regions | Average for regions |
| PA | | Mid-Atlantic[a] 3.5 | 53 | 175 | 2.9 |
| DE, FL, GA, MD, NC, SC, VA, WV | | South Atlantic[b] 2.5 | 44 | 200 | 2.1 |
| AR, LA, OK, TX | | West South Central 1.9 | 41 | 200 | 2.0 |
| AL, KY, MS, TN, AK, CA, HI, OR, WA | | East South Central & Pacific 1.7 | 32 | 160 | 2.1 |
| CT, IL, IN, MA, ME, MI, NH, OH, RI, VT, WI | | North-East 1.4<br>East North Central 1.3 | 34 | 140 | 2.4 |
| AZ, CO, ID, MT, NM, NV, WY | | Mountain 1.1 | 32 | 140 | 2.2 |
| IA, KS, MN, MO, ND, NE, SD | | West North Central 1.0 | 27 | 120 | 2.0 |

Note: ''Actual'' among military applicants, 1985–2000 [5] (''odds ratios'' are the average of the adjusted and unadjusted ratios given in the cited source, since the adjustments may not have considered properly all the variables).

[a] Also includes New Jersey and New York, two of the highest States, already shown above.

[b] Also includes Washington DC, already shown above as highest.

complications are not warranted by the point at issue, which is simply this: Can the actual geographic distribution of F(HIV) be accounted for reasonably well by considering only race and population density? The answer is, ''Yes''. By contrast, the standard view of HIV/AIDS has offered no explanation at all for the East-over-West weighting of F(HIV).

But is this just a circular argument, a tautology? Is this no more than taking the empirical dependence of F(HIV) on two variables, race and population density, and then recombining them to reproduce the reported pattern? Of course they should match! How could they not?

In a sense, yes, it is a rather circular argument. But in an important sense, no, it is not.

For one thing, the empirical data on race and population density used in these calculations were not derived just from those sources that reported geographic distributions around the United States; the averages used were from ***all*** the sources that reported quantitatively about race ratios and variations by population density, and they did so for disparate sectors of the population and for a variety of periods of time. Second, the calculations presume implicitly that the subject of interest, F(HIV), is unchanging over time, and present in the general population rather than only in some specific sub-groups for limited times. If those cal-

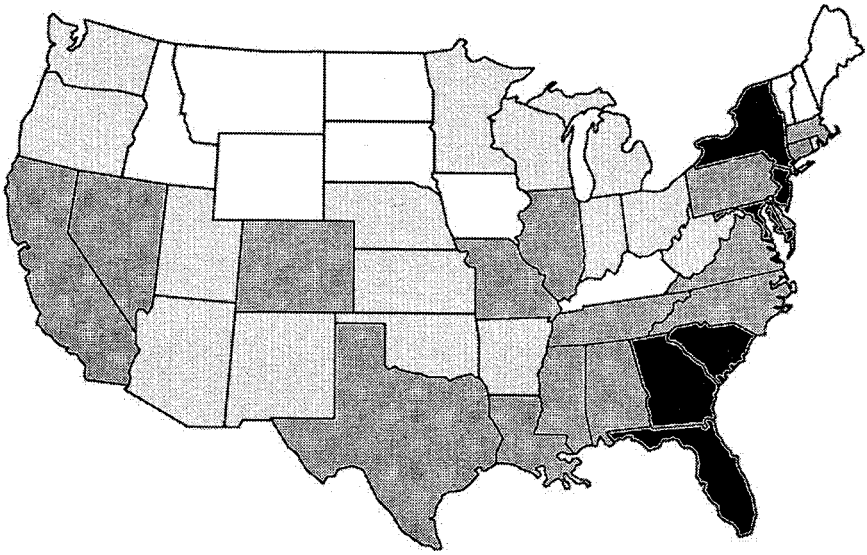

Fig. 10. Observed geographic distribution of F(HIV) averaged over time and social groups; from Figures 1–5 and 7–11 in [1].

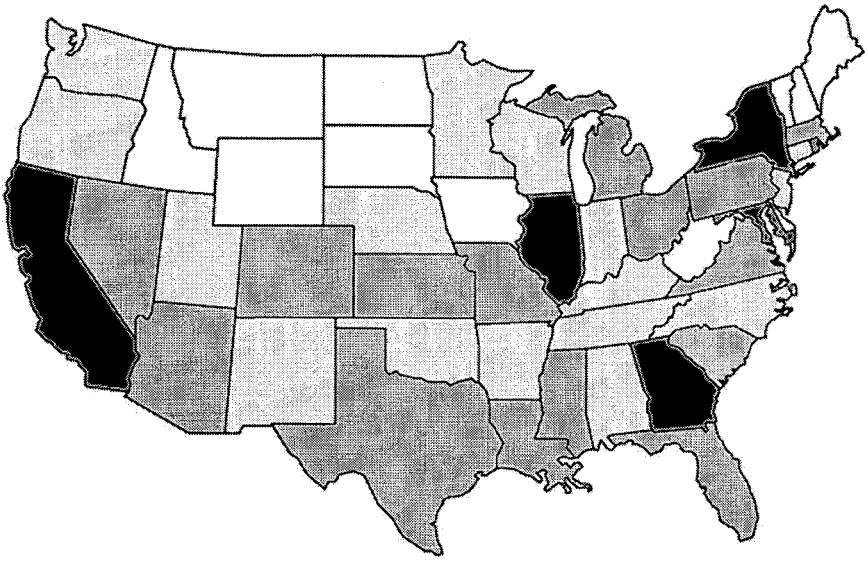

Fig. 11. Geographic distribution of F(HIV) if race and population density are all that counts, using numbers from Table 12.

culations yield a good fit with actual data on smaller groups studied at different periods of time (the ten various maps in Part I [1]), as they do, then those implicit assumptions would seem to be good ones.

But, again, the main point at issue does not depend on such detail. The question simply is, does F(HIV) reflect the spread of a sexually transmitted infection, or does it not? Now, if one were asked to speculate about the distribution in the United States of syphilis or gonorrhea or any other sexually transmitted infection, the attempt to do so by combining race ratios and population densities would rightly be dismissed out of hand as mumbo-jumbo numerology. If there were a spread of HIV owing to unsafe sexual practices and the sharing of unclean infected needles, if the distribution of HIV in the United States depended on such chosen behavior, one would not find a geographic pattern that could be matched so easily with just the two variables of race and population density. That the matching works at all is thus yet another point against the HIV/AIDS theory and in favor of the interpretation in terms of physiological stress.

That the high outliers of 14 in Table 3 and 8.8 in Table 7 represent blood donors, the healthiest group, makes sense if race influences F(HIV) as an independent variable and if F(HIV) is proportional to a health challenge. If a health challenge is severe enough, all or most members of every group of people will be affected, and the influence of any modifying factors such as individual or racially correlated response would not be apparent. So one would expect to note differences in F(HIV) that are owing to age, sex, and race most clearly in the healthiest groups—blood donors—and least clearly in people who are most challenged, say MSM and IDU showing symptoms of pre-AIDS or AIDS. This expectation is supported, for example, in a study by Torian et al. [39]: When F(HIV) was relatively high—34% for whites and 56% for blacks in 1990 in New York—the black-to-white ratio was relatively low, 1.65; whereas when F(HIV) was lower—11% and 28%, respectively—the ratio, 2.6, was correspondingly higher. Similarly, the Hispanic-to-white ratio was only 1.4 when F(HIV) was 34% for whites and 47% for Hispanics (in 1990) but higher at 1.7 when the overall F(HIV) was lower, 11% and 19%, respectively (in 1999).

## Conclusions

In Part I [1], it was shown that HIV tests do not track a virus that spread from the original centers of the AIDS epidemic; HIV is endemic, not epidemic. Part II [2] found that F(HIV) correlates with the average general level of health or fitness; the production of what are taken to be antibodies to HIV seems to be a non-specific sign of some sort of physiological stress. In Part III, it has been demonstrated that the tendency to produce these antibodies runs parallel to certain ancient patterns in the human genome.

These conclusions, based solely on the epidemiology and demographics of HIV tests, are fully concordant with conclusions reached on the basis of retrovirology and molecular biology by Duesberg [66, 67] and by the Perth

Group [68]. Duesberg has argued for two decades that HIV is a harmless component of the human genome that is ''transmitted'' primarily from mother to child; and the Perth Group has argued that ''HIV antibodies'' reflect oxidative stress and not necessarily antibodies to a retrovirus. That a positive HIV-test does not necessarily denote any serious health problem is illustrated by a variety of evidence described in Part II [2]. Another example is the fact that the fertility of HIV-positive women is no different from that of HIV-negative ones [14].

Many other researchers and writers have explained why the theory that HIV causes AIDS is neither proven nor sound, and have done so with full attention to all the points typically raised whenever anyone questions whether HIV causes AIDS: AIDS in hemophiliacs, AIDS in Africa, ''life-saving'' AIDS medications, and more [66–74]. Many authors have pointed out how misleading are the periodic press releases from authoritative organizations about the numbers of people supposedly infected by HIV or suffering from or having died from AIDS (for example, [75])—numbers derived only from computer models that are unverified and whose very authors emphasize that the models need further refinement [76]. Every point among the popular shibboleths supposedly reinforcing the HIV/AIDS dogma has been fully answered in these writings—say, the notorious case of the Florida dentist who supposedly infected several people [77]. Many authors have explained why the official view that Africa is being ravaged by AIDS is not only unproved but provably incorrect (for example, [72, 78, 79]).

It remains for students of the sociology of science and medicine to fill in the details of how this mistaken view came to be so dominant for so long even as ample evidence against it has long been available in the peer-reviewed literature and in official reports of actual data. Steven Epstein [80] has already described the degree to which non-scientific pressures influenced the initially defining course of HIV/AIDS science and practice. Michelle Cochrane [81] has demonstrated that the assumption of an infectious cause of AIDS had been made by the Centers for Disease Control and Prevention from the beginning, and that this resulted in misleading classification of AIDS cases. Actual evidence of an infectious agent has never been adduced. Speculation about sexual transmission had been based on the presumption of a period of months rather than years between infection and symptoms; that presumption was soon found to be wrong, it was soon realized that any such latent period must be years rather than months (the currently accepted figure being nine or ten years), but this has not lead to re-consideration of the hypothesis—actually the dogma—of sexual transmission. Bruce Nussbaum [82] has described how the Establishment approach to AIDS was dominated by virology, to the neglect of treatment of opportunistic infections which community doctors had shown to be effective. As to the general situation of science and medicine in the 21st century that makes possible such mis-steps, the dominance of knowledge monopolies and research cartels [83] plays a large role.

The analysis of HIV data in the three papers in this series points to needed research in several directions. There are persistent hints in the data that black

mothers are unusually prone to show this physiological response to stress (Figure 9; Tables 4 & 6)—the B/W ratio for new mothers is 2 or 3 times the average overall B/W ratio of about 5.5. Perhaps most needed is an investigation of the apparently more frequent appearance of the HIV-positive response among gay men, to determine whether this signifies anything about their health, and if so, whether it is connected solely with the fast-lane life, as the occurrence of AIDS was.

## Implications

The facts and conclusions set out in this series of papers hold significant implications for social and political attitudes and actions. The mainstream dogma that HIV causes AIDS has made it standard practice to treat HIV-positive people with highly toxic, debilitating chemotherapy (''anti-retrovirals''). Even against expressed wishes of parents, infants have been subjected to this iatrogenic damage. Parents who refused HIV tests for their children have been threatened with prosecution for child neglect. Incessant propaganda urges that as much as possible of these chemotherapeutic poisons be distributed throughout Sub-Saharan Africa. All these actions and practices should cease forthwith, since HIV does not cause AIDS.

Gay men who test HIV-positive face a special dilemma. Mainstream medicine and the most influential organizations of gay people urge them to take treatment, and the federal government makes that affordable for everyone in the United States. But most gay men are also aware of the dissident professional views and of the many organizations of gay men and others who have forsworn or abandoned anti-retroviral treatment and who offer numerous testimonials that one can remain entirely healthy and active for decades as an untreated HIV-positive person. Gay men are also likely to have observed the dreadful side-effects suffered by many or most of those who accepted mainstream treatment. HIV-positive gay men therefore have a choice of only two options, both of them psychologically stressful. They know that accepting retroviral treatment is likely to be physically debilitating and that there is no real evidence that it is life-extending. But if they refuse treatment, they can hardly avoid worrying continuously that they may have made the wrong choice.

Official acknowledgment that HIV does not cause AIDS is long overdue.

## Notes

[1] 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.

[2] Gregor Mendel was the first to show that such obvious hereditary traits of pea plants as size or color arise in successive generations in simple numerical proportions: 1 to 3, or 1 to 2 to 1, or the like. Nowadays this is understood to reflect the influence of single genetic factors. It is also understood that such simple genetic influences are rare, and have no relevance to anything sig-

nificant about the behavior of human beings; and moreover that it is an oversimplification to think in terms of isolated individual genes at all.

[3] From CensusScope, a product of the Social Science Data Analysis Network. Native Americans were 0.63% of the US population in 1980, 0.72% in 1990, and 0.74% in 2000; www.censusscope.org/us/chart_race.html, accessed 14 June 2005.

[4] National Household Survey on Drug Abuse, Health Promotion and Substance Abuse Prevention Among American Indian and Alaska Native Communities: Issues in Cultural Competence, SAMHSA, 2001; http://ncadi.samhsa.gov/govpubs/prevalert/v5/10.aspx, accessed 14 June 2005.

[5] http://www.censusscope.org/us/chart_race.html

[6] It was pointed out in Part II [2] that MSM for whom information about HIV is available from surveys and studies are only a small proportion of all gay men, largely or perhaps even entirely those who practice the ''fast-lane'' lifestyle that includes overindulgence in alcohol and drugs. There is no evidence that having gay sex constitutes in itself any danger to health.

[7] www.dnaprint.com/2003/science/science.html, accessed 18 June 2005

[8] www.BiDil.com

[9] Prescribing information for Crestor, Rev 08/05, at http://www.crestor.com/#important, accessed 12 February 2006.

[10] quickfacts.census.gov/qfd/states/02000.html, accessed 18 June 2005.

[11] www.demographia.com/db-uza2000.htm for large urban areas and www.demographia.com/db-usa-staterural.htm for rural areas, acessed 18 June 2005.

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