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# DEMOGRAPHIC CHARACTERISTICS OF HIV: III. WHY DOES HIV DISCRIMINATE BY RACE?

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*ABSTRACT:* The relative circumstances of Black and White Americans serves 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 has increased by a factor of 3; but the relative incidence of positive HIV-tests in the two groups has remained the same.

Racial ancestry influences the frequency of positive HIV-tests, F(HIV), as an independent variable. In a variety of disparate demographic groups 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 renders a behavioral explanation less likely than 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 bespeaks a physical rather than socioeconomic-behavioral cause. F(HIV) is a response to a health challenge; the intensity of that response is evidently modified by ancestral genomic patterns among HLA 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.

*KEYWORDS:* HIV and race---human polymorphisms---physiological stress---HIV geography

## *INTRODUCTION*

The results of positive HIV-tests are often called "the prevalence of HIV". This presupposes that what is detected by "HIV tests" is in fact a human immunodeficiency virus, HIV. But the commonly used tests are for ***antibodies***, whose presence has been 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 [Bauer, 2005] revealed that the distribution of F(HIV) in the United States has remained unchanged over two decades. It did not track 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 [Bauer, 2006] showed that F(HIV) tracks challenges to health in some fashion; it appears to be an 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.

In this article, the variation of F(HIV) with racial category is shown to be associated with genetic factors. And once more, the data are incompatible with the view that F(HIV) tracks a sexually transmitted infection (STI).

The mass of available data, from tens of millions of HIV tests on a disparate variety of groups of the population of the United States, shows 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 are almost quantitative with respect to race. F(HIV) is lowest among Asians, and about 50%--70% 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 4 or 5 times greater than in White Americans, not infrequently more, rarely much less (see, for instance, [3, 4, 5, 6, 7, 8, 14, 19, 22, 25, 30]).

Possible reasons for such variation by race include biological, cultural, geographic, and socioeconomic factors. I will argue that the evidence points to a purely physical rather than behavioral 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 predispose toward risky sexual behavior or the sharing of needles. That, however, is what the conventional view of HIV/AIDS implies. It 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 [31]; emphasis added). These hand-waving generalities about behavioral norms and social mixing patterns are a euphemistic way of saying that Black Americans, and to a lesser degree Hispanics (but only in the East, not the West!), are more intimately and constantly engaged in carelessly unsafe sex than are Whites or Asians or Native Americans; "behavioral norms and complex social mixing patters" among Black Americans are presumed to bring drug users and gay men into chains of sexual contact that pervade all other social groups in Black communities--for the racial disparities in F(HIV) do show up in all social groups: Black MSM have higher F(HIV) than white MSM. Black IDU show a higher F(HIV) than White IDU. Even in the Marine Corps, even among childbearing women, even among repeat donors of blood--in every tested group, F(HIV) for Black Americans is greater than for White Americans, by a factor not far from 5.

This article reviews in detail the data on F(HIV) and racial category and argues that they are incompatible with any behavioral explanation. That is consistent with and reinforces the conclusions in Parts I and II, that HIV is not a sexually transmitted infection but rather an indication of physiological stress. The correlation between conventional racial categories and F(HIV) evidently comes about through genomic polymorphisms of the type that have been used to track the course of human migrations over the last few hundred thousand years: genomic patterns like those that influence skin color modify physiological responses to certain stresses. People of African ancestry display the strongest response and people of Asian ancestry the weakest.

This point of 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.

## *DEMOGRAPHIC VARIABLES*

In Parts I and II, 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 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. 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 results from other studies were 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]. 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 and sex. In practice, such exact matching in all except one variable is never found. It is therefore extraordinary that "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 [30]). It is even more extraordinary that 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. These regularities are so clear that the relative proportions 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 for random fluctuations in the compositions of the samples[3].

*NATIVE AMERICANS*

Native Americans are an ideal probe to test whether a trait is genetic or behavioral. Genetically, Native Americans are closely related to Hispanics, 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 on.

Very few groups are large enough to report separately for Native Americans, who constitute less than 0.8% of the population[4]. In those few reports[5], F(HIV) among Native Americans falls closer to that among White Americans and Hispanics than to that for Black Americans (Table 1).

**Table 1**

| | **a** civilian applicants to military [5] | **b** Job Corps [30] | **c** from public sites [17, 18, 23] | **d** young MSM [28] | ***Average of a--d*** |
|---|---|---|---|---|---|
| Asian | 0.59 | 0.4 | 0.63 | 0.9 | **0.63** |
| White | 1.00 | 1.00 | 1.00 | 1.00 | **1.00** |
| **Native American** | 1.47 | 1.6 | 1.23 | 2.0 | **1.6** |
| Hispanic | 2.25 | 1.6 | 2.37 | 2.1 | **2.1** |
| Black | 6.25 | 6.4 | 2.76 | 4.3 | **4.9** |

Table 1: F(HIV) in Native Americans compared to other racial categories.
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. For column (c), the ratios were calculated by first summing the actual numbers of tests in each category for each year.

Bearing in mind the caveats about quantitative comparisons (previous section, "Demographic Variables"), the values for each racial category are remarkably consistent across these three disparate social groups. This consistency suggests that they reflect ***material*** differences like skin color and not 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, a group in which there is a high 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 this 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 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 and about 28 percent of Whites . . . 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"[6].

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

**Table 2**

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

Table 2: Native Americans as victims of violent crime in comparison to other racial groups. 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

Once again, as in Parts I and II 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[7], 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 seems remarkable that 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, even as the average level of infection changes over nearly two orders of magnitude, from about 0.1% among military cohorts to not far from 10% among young MSM; and among groups that cover a wide range of socioeconomic circumstances. 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 Whites to be "unsafe" and Blacks nearly 5 times as likely to be irresponsible? To offer a behavioral 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 reported in many studies, relative to those of White Americans in the same groups.

**Table 3**

| Group | Black-to-White ratio of F(HIV) | Sources |
|---|---|---|
| Blood donors | 14 | 10, 122 |
| College students | 1.5 | 14 |
| Marriage-license applicants | 4.3 | 15 |
| Active Army | 3.8 | 3, 4, 94, 95 |
| Applicants for military service | 7.9 | 1, 5, 6, 2, 30, 27 |
| Teenage applicants for military service | 4.7 | 7, 75, 67 |
| Army Reserve components | 6.4 | 70 |
| Navy | 4.3 | 8 |
| National Health and Nutrition Survey | 4.0 | 119 |
| Job Corps | 5.1 | 19, 20, 22, 27, 30 |
| Various public sites | 2.8 | 17, 18, 23 |
| Hospitals and outpatient clinics | 3.4 | 27, 30, 98 |
| STD clinics | 2.8 | 25, 26, 85 |
| Prisons | 5.5 | 115 |
| IDU | 4.0 | 88 |
| MSM | 2.7 | 29, 30, 34, 114, 118 |
| | | |
| AVERAGE | 5.1 | |
| Standard deviation | 3.0 | |

Table 3: Ratios of F(HIV) for Black Americans relative to White Americans among various groups. 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). The quality of methodology was likely 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

The low outlier, 1.5 for college students, is from the smallest sample (17,000) as well as from one of the earliest surveys, and one would have good reason to neglect it, but that would change the average value very little. The next smallest number, 2.8 from public sites, is from one of the largest samples (>8 million), as is the high outlier for blood donors (800,000 and 2.2 million tests in the two sources), so the range of values is certainly no narrower than from 2.7 to 14.

That the high outlier of 14 represents 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. Under circumstances where the health challenge is severe, most of any given group is affected, and the influence of any modifying factors such as individual or racially correlated resistance or fitness would become less apparent. Consider a situation where vaccination for flu, say, has been only partly effective, because the vaccine was prepared for a different strain than the epidemic one. One would then expect, in a group hospitalized for flu, not much difference in recovery rate for vaccinated and unvaccinated individuals; whereas among people known to have been exposed to the flu but not ill enough to be hospitalized, one would expect a rather high ratio of vaccinated to unvaccinated. Similarly, in the case of F(HIV), 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 very ill, say MSM and IDU showing symptoms of pre-AIDS or AIDS. This expectation is supported, for example, in a study by Torian et al. [114]: 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, 2.55, was correspondingly higher; similarly, the Hispanic-to-White ratio was only 1.38 when F(HIV) was 34% for Whites and 47% for Hispanics (in 1990) but higher at 1.73 when the overall F(HIV) was lower, 11% and 19% respectively (in 1999). That the high outlier is for blood donors

not only in the case of Black Americans but also among Hispanics (further below, Table 4) is therefore consistent with an explanation of F(HIV) in terms of physiologic stress.

The groups considered here are even more varied as to social and economic circumstances than in the case of Native Americans, above. Recalling once again the limitations in attempting to make quantitative comparisons among groups whose composition varies by age, sex, and population density of residence, these numbers are quite close to one another--so much so that I calculated a standard deviation. If the high and low outliers are omitted, the average becomes 4.6 with a standard deviation of 1.5.

But the actual numbers are not of primary interest, nor even their constancy. What is certainly constant in all groups is that F(HIV) always follows the same sequence, Asian < White < Native American < Hispanic < Black. The question is, do these racial disparities arise from behavioral differences or from physical or physiological ones? That the relative values for the racial categories are reasonably constant over a range of social groups is suggestive. Perhaps even more compelling is the fact that the same disparities are seen at all ages and for both sexes: within a given group, the ratio is much the same at different ages ([4]--Figure 1, [19]--Figure 2), as well as for both sexes ([5], Figure 3):

**Figure 1**

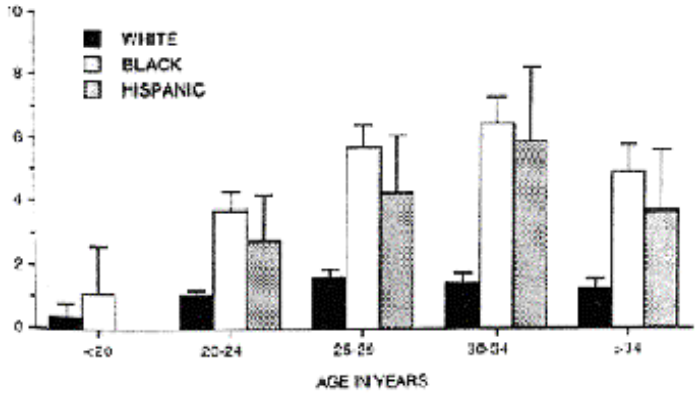


Figure 1: The same racial disparities are seen at all ages. US Army personnel, 95% confidence interval bounds shown; HIV prevalence per 1000 [4]

**Figure 2**

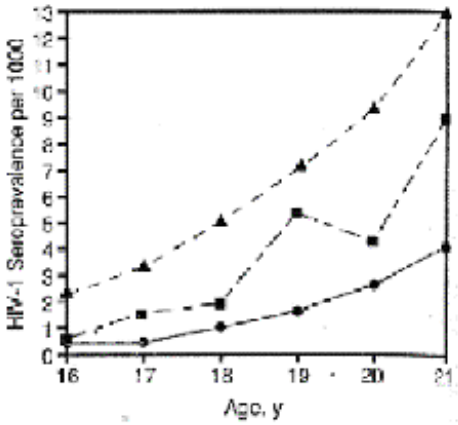


Figure 2: The same racial disparities are seen even among teenagers [19] (where the relative values for the sexes often change from higher for females among younger teenagers--usually 16 and below--to higher among males in older teenagers--typically above 17 or 18) [Bauer 2006]
▲ = black, ■ = Hispanic, ● = white

**Figure 3**

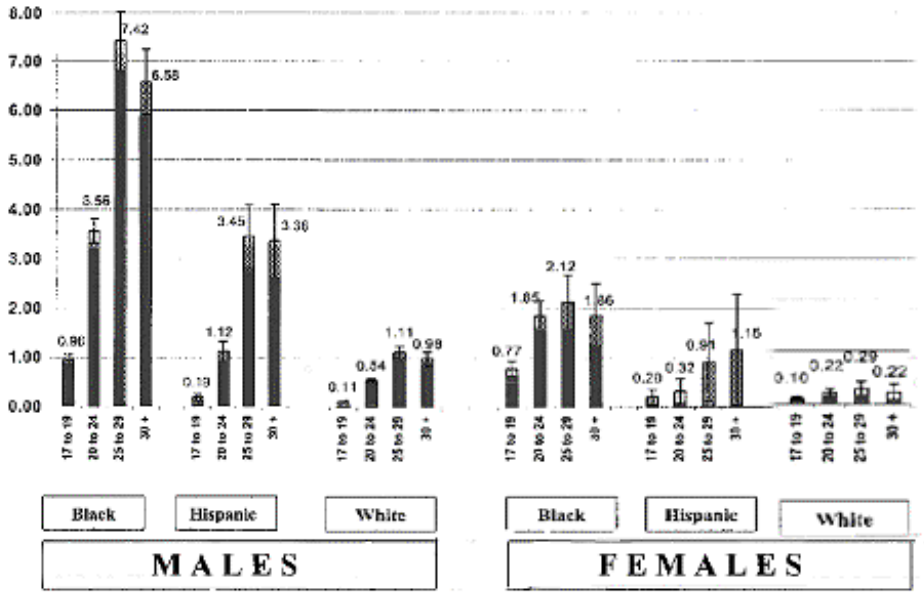


Figure 3: The same racial disparities are seen in both sexes and at all ages; 95% confidence intervals shown; HIV prevalence per 1000; "other" racial category omitted. Civilian applicants for military service [5]

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

**Figure 4**

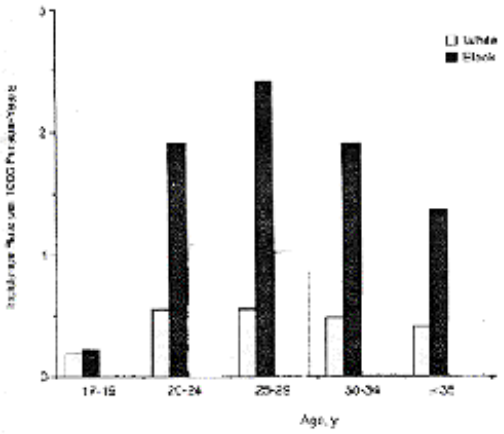


Figure 4: The same racial disparities are seen for ***annual incidence*** of "HIV infection" as for overall "prevalence", F(HIV), at all ages; active-duty US Navy personnel, 1986; "other" and "all" racial categories omitted [8]

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 ([34], Figure 5).

**Figure 5**

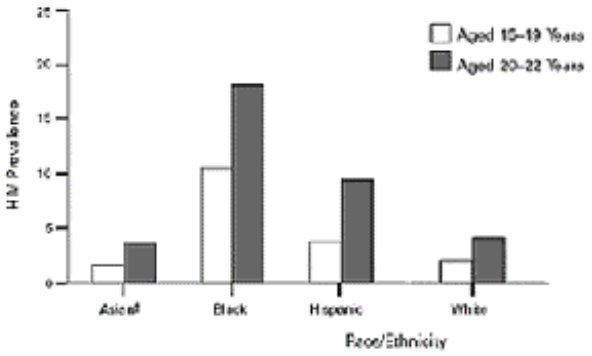


Figure 5: The same racial disparities are seen even when F(HIV) is as high as a few percent to nearly 20%. Data for young (15--22 years) MSM in seven cities, 1994--98 (Baltimore MD, Dallas TX, Los Angeles CA, Miami FL, New York City NY, San Francisco CA, Seattle WA; "Asian" includes Asian/Pacific Islander); "mixed" race and "total" categories omitted [28]

And again, with gay men 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" was 1.4 per 50 years; the rate for Black Americans was 2.23 and that for Hispanics was 1.77 greater than that for White Americans [144].

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 [30]). The scales on the horizontal axes in these two Figures differ by a factor of 10:

**Figure 6**

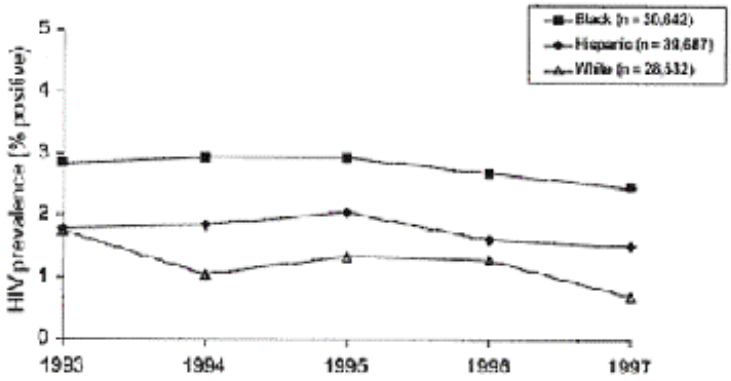


Figure 6: The same racial disparities are seen among heterosexual patients at STD clinics; data from 1993--97 [30]

**Figure 7**

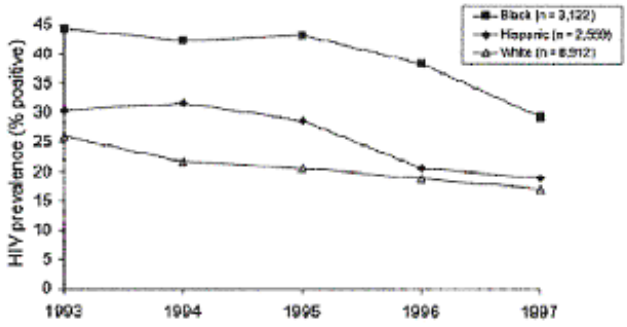


Figure 7: The same racial disparities are seen among MSM at STD clinics; data from 1993--97 [30]

These relations have remained semi-quantitatively the same during two decades even as the overall F(HIV) declined steadily ([1], Figure 8).

**Figure 8**

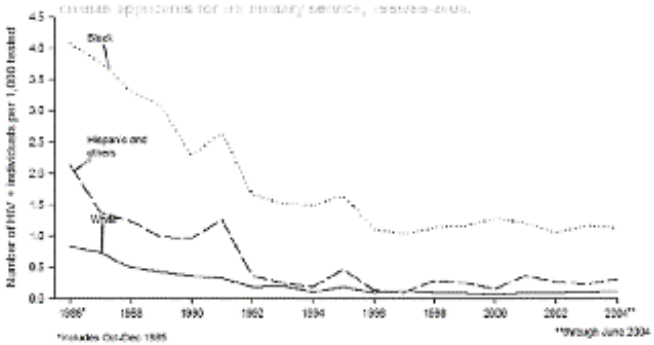


Figure 8: The same racial disparities were seen among military applicants in every year for two decades (1985--2004), even as the overall magnitude of F(HIV) declined significantly [1]

The same pattern persists within each State [27]; for example, Figure 9 for child-bearing 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.

**Figure 9**

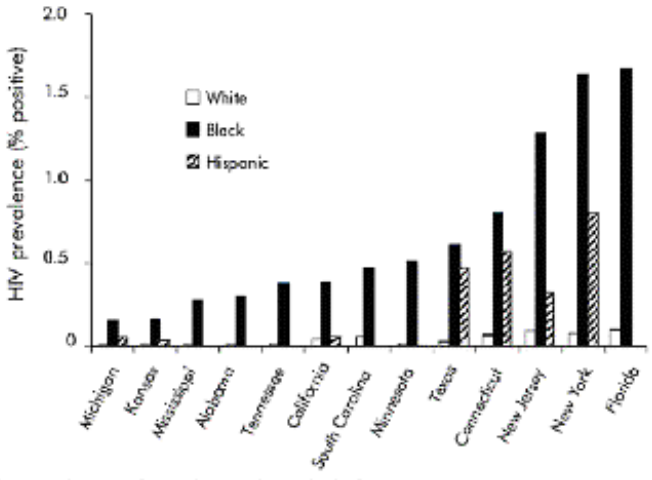


Figure 9: The same racial disparities are seen in every State. Data for childbearing women, 1994 [27]

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; in Belle Glade (FL); among female prostitutes (F(HIV) was 15.4% for Black and Hispanics, 6.7% for Whites and "others"; the ratio was 2.5 among those who admitted injecting drugs, 3.3 among those who did not [160]. In the mid-1980s among 5000 MSM in Baltimore, Chicago, LA, and Pittsburgh, Black men were infected 60--80% more often than others [69]. 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 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) [164]. The national surveillance summary [31] up to 1992 noted that Black women were three to 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 [77] found F(HIV) among Black IDU about 4 times greater than among White IDU (varying from 1+ to about 6 in different cities), and among Hispanic IDU about 5.8 times greater than among Whites (varying from 1+ to about 16).

MSM and IDU are the groups at highest risk[8], 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 irresponsibly promiscuous people, Black Americans exceed by a large factor the irresponsibility and promiscuity of White Americans? Surely it is 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 to 44.9% for 1996--2000 [168]. The percentage of AIDS cases who were White decreased in proportion, from 59.7% to 50.4% to 42.4% to 34%. The ratio of Black percentage to White percentage thus changed from 0.43 to 0.62 to 0.90 to 1.32, a factor of 3. In complete contrast, the ratio of F(HIV) among Black Americans to that among White Americans has remained constant at ~5--6[9].

The relative incidence of AIDS among Blacks and among Whites has changed by a factor of 3. The relative incidence of HIV has not changed. HIV and AIDS are not correlated.

*HISPANIC AMERICANS*

All the considerations about Black Americans apply as well to the case of Hispanics, but there are a few additional points worth noting.

Of particular interest is the fact that 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 [31]).

Now, the official classification of "Hispanic" does not parallel the others: it is ethnic, not racial. In some circumstances, official figures (for example, from the Census Bureau) make distinctions between white Hispanics and black Hispanics. If racial variations in F(HIV) reflect physical differences between the racial categories, then this strange geographic differentiation within the category of Hispanics makes sense: In the West, "Hispanics" are largely of Mexican ancestry, whereas in the East a high proportion of "Hispanics" are from the Caribbean and have on average a significant proportion of African ancestry.

Therefore, when data concerning "Hispanics" are considered, there is an additional factor beyond age and sex to give rise to chance fluctuations. Consequently, the observed 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 illustrated by the data in Table 4; the standard deviation of 2.1 on an average of 2.7 reflects a greater variability than in the Black-to-White ratios in Table 3.

**Table 4**

| Group | Hispanic to White ratio of F(HIV) | Sources |
|---|---|---|
| Blood donors | 8.8 | 10, 122 |
| College students | 1.6 | 14 |
| Active Army | 2.9 | 3, 4, 95 |
| Applicants | 2.3 | 1, 2, 5, 6, 27, 30 |
| Teenage applicants | 1.5 | 7, 67, 75 |
| Reserve components | 4.5 | 70 |
| Various public sites | 2.4 | 17, 18, 23 |
| National Health and Nutrition Survey | 1.33 | 119 |
| Job Corps | 1.7 | 19, 20, 22, 27, 30 |
| Hospitals and outpatient clinics | 1.4 | 27, 30 |
| STD clinics | 1.2 | 26, 85 |
| Prisons | 3.4 | 115 |
| MSM | 2.0 | 29, 30, 34, 118 |
| | | |
| AVERAGE | 2.7 | |
| Standard deviation | 2.1 | |

Table 4: Ratios of F(HIV) for Hispanics relative to white Americans among various groups.
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 was likely 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

A standard deviation so close to the mean value--78% of it--conveys little confidence that a constant is being measured (though if the high outlier is deleted, the average becomes 2.3 with a standard deviation of 1.0). But recall [Bauer 2005] that a geographic correlation between HIV and AIDS was asserted [110] when the ratio between the two in 52 regions was 11.2 with a standard deviation of 8, 78% of the mean value. In that case, no confounding variables were in play, whereas in the present case there are three: age, sex, and racial ancestry within the Hispanic category. The evidence is much better for a constant ratio of F(HIV) between racial and ethnic categories than is the evidence for a correlation between HIV and AIDS.

*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. Some would call the belief racist, but I prefer to view it simply from an intellectual viewpoint: 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 people. He went on to point out that there is no evidence to support such a conclusion" (p. 31 in [Mbeki report]).

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 they come largely from the Caribbean and South America? Is needle-sharing and sexual promiscuity a 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: Marines, soldiers, Job Corps, MSM, child-bearing women, prisoners, drug abusers in treatment centers, or those attending public clinics for adolescents or for family planning.

As a matter of fact, research 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 [141]. Samuel & Winkelstein [161] 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 [162] found no differences between races as to anal intercourse, measured via the incidence of rectal gonorrhea. Bausell [163] found White Americans ***less*** likely to take protective measures as to 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. F(HIV)--the antibody reactions that are assumed to detect HIV--seems to be an indicator of physiological stress: deep-seated "racial" genomic patterns modify the physiologic response to certain health challenges.

*RACE AND GENOMES*

Cavalli-Sforza[10] has shown that human migration patterns for the last 200,000 years or so can be traced using mutations accumulated in the human genome; and that the patterns so derived are reassuringly 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. The chief claims of that work, that the human genome contains certain patterns that parallel conventional

racial classifications, have found general acceptance. 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--for example, BiDil was approved by the Food and Drug Administration in June 2005 after tests on African-Americans, who had failed to benefit fully from existing medications for heart disease. DNAPrint Genomics offers ancestry-tracing services based on what they call "Ancestry Informative Markers"[11].

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; for instance, in the early 1990s a scheduled conference on "The Biological Basis of Crime", sponsored by the National Institutes of Health, was canceled because of protests that such discussions would inevitably have racist under- or over-tones[12].

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" [38]; "white females who donate blood regularly (at least once a year) had an HIV infection rate of three in 113000 units donated. In contrast, . . . black females who donate regularly have an HIV infection rate of 90 in 7500 units of donated blood" [39].

The SANBS 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 5).

**Table 5**

| Classification by South African National Blood Service | Nature 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 |

Table 5: F(HIV) in blood in South Africa, by racial classification [MANTO]. "Colored" is the South African term for mixed ancestry

The same sequence was reported for women at prenatal clinics in 1990 by the South African Department of National Health ([120]; Table 6).

**Table 6**

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

Table 6: F(HIV) among women at prenatal clinics, 1990, South African Department of National Health [120]

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 [MANTO] or 2.7 [39] per 100,000 is quite similar to the US figures for 2002 [13], age-averaged at 1 per 100,000 (from 3.7 for 20--29-year-olds to ≤1 for those over 50).
- F(HIV) for pregnant women (Table 6) is significantly higher than for blood donors (Table 5).

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

As already noted, this view affords an explanation for the East-over-West prevalence of F(HIV) among Hispanics in the USA; 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" [40]. 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 7).

**Table 7**

| ETHNIC GROUP (NUMBER OF CASES) | EUROPEAN | SUB-SAHARAN AFRICAN | EAST ASIAN | NATIVE AMERICAN |
|---|---|---|---|---|
| Mexican (60) | 43 | 6 | 4 | 47 |
| Puerto Rican (64) | 55 | 33 | 4 | 9 |
| American Indian* (223) | 42 | 4 | 7 | 48 |
| American Indian** (170) | 29 | 2 | 8 | 61 |
| African American (136) | 14 | 80 | 3 | 3 |
| European American(207) | 90 | 3 | 3 | 4 |
| South Asian Indian (56) | 59 | 5 | 27 | 9 |

Table 7: Ancestral connections to 4 "racial" sets of genetic marker, as percentages; from AncestryByDNA©; www.ancestrybydna.com/Ethnicities.asp, accessed 25 June 2005 (numbers rounded and standard deviations omitted)
* includes individuals from US Government recognized tribes (Sioux, Cheyenne, Cherokee, Arapaho) as well as unrecognized tribes, without regard to "blood" percentage
** includes individuals from US Government recognized tribes only (Sioux, Cheyenne, Cherokee, Arapaho), without regard to "blood" percentage

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

The data in Table 5 are also in line with these genetic trends. The ratio of Black-to-White F(HIV) among South African blood donors is about 25, larger than the ratio of about 14 (Table 3) 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 7); in South Africa, there has been less racial mixing.

That the Indian category in South Africa is similar to their "White" category is also consistent with the last two rows in Table 7.

Among the genetic factors that have been studied in relation to race, particularly relevant to the present concerns are the HLA genes associated with the immune system. For example, "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 [Bodmer]).

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. For instance, "A unique African HLA haplotype may identify a population at increased risk for kidney graft rejection. . . . Unique HLA alleles and MHC haplotypes have been identified in the Cape Colored and in the black South African populations. . . . Because HLA haplotypes are inherited 'en-bloc' as ancestral haplotypes that vary considerably between races" [167].

The dark skins of Africans have a known physiologic function. Sunlight absorbed through the skin catalyzes the formation of vitamin D, which is both essential and also harmful at too large doses. In equatorial regions, unfiltered sunshine produces too much vitamin D. Humans evolved in Africa with the optimum degree of sunshine-filtering by the skin. As humans 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. That is well established. The following is speculation that has not so far been tested, but it is based on the fact that a great variety of bacterial, microbial, parasitic, and viral diseases are endemic in tropical regions. It would therefore be curious if humans, evolving in Africa, had not acquired very strong immune responses against a wide range of those challenges to health. As humans migrated to other, non-tropical parts of the world where challenges to the immune system were less frequent, it seems reasonable that the responses generated by the immune system would become somewhat weaker. In Part II of this series of articles, F(HIV) was shown to behave like a response to a physiologic challenge. It would be reasonable to expect that 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.

### *GEOGRAPHY OF F(HIV) IN THE UNITED STATES*

In Part I, 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 could find no explanation under the official theory that HIV is a sexually transmitted infection. This well attested asymmetry actually follows rather obviously from the dependence of F(HIV) on race, discussed above, and on population density, reported in Part II.

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 0.6 to 1 to 1.6 for Asian, White, and Native Americans (see Table 1) to 2.7 for Hispanics (see Table 4) to 5.1 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[13]. 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 [Bauer 2006]). 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[14] and the Census 2000 figures for total populations in each State. The product RD yields the ranking of States shown in Table 8.

**Table 8**

| | R from race ratios | D from population densities | **CALCULATED F(HIV)** RD/10, rounded | | | R from race ratios | D from population densities | **CALCULATED F(HIV)** RD/10, rounded |
|---|---|---|---|---|---|---|---|---|
| DC | 351.0 | 27.58 | **970** | | AL | 206.0 | 1.55 | **32** |
| NY | 185.9 | 4.08 | **76** | | NM | 181.8 | 1.75 | **32** |
| RI | 129.9 | 4.59 | **60** | | OR | 116.6 | 2.71 | **32** |
| IL | 178.7 | 3.21 | **57** | | MN | 116.7 | 2.68 | **31** |
| PR | 286.7 | 2.00 | **57** | | NC | 193.3 | 1.60 | **31** |
| MD | 215.8 | 2.65 | **57** | | IN | 138.2 | 2.11 | **29** |
| GA | 222.0 | 2.57 | **57** | | CT | 150.1 | 1.88 | **28** |
| CA | 175.1 | 3.25 | **57** | | WI | 127.7 | 2.17 | **28** |
| TX | 198.6 | 2.72 | **54** | | TN | 168.1 | 1.64 | **28** |
| FL | 185.1 | 2.91 | **54** | | OK | 157.7 | 1.65 | **26** |
| LA | 233.0 | 2.18 | **51** | | AR | 167.2 | 1.52 | **26** |
| NV | 156.7 | 3.23 | **51** | | UT | 116.4 | 1.88 | **22** |
| AZ | 155.6 | 3.02 | **47** | | NE | 124.1 | 1.70 | **21** |
| PA | 143.9 | 2.89 | **42** | | KY | 130.5 | 1.56 | **20** |
| MA | 129.6 | 3.18 | **41** | | AK | 120.2 | 1.65 | **20** |
| MI | 160.3 | 2.53 | **41** | | ID | 113.8 | 1.66 | **19** |
| CO | 142.3 | 2.77 | **39** | | WY | 113.7 | 1.65 | **19** |
| VA | 183.3 | 2.12 | **39** | | IA | 111.8 | 1.61 | **18** |
| OH | 147.6 | 2.55 | **38** | | NH | 104.2 | 1.60 | **17** |
| MS | 246.9 | 1.49 | **37** | | WV | 113.0 | 1.46 | **17** |
| KS | 132.7 | 2.72 | **36** | | ND | 105.1 | 1.56 | **16** |
| MO | 146.6 | 2.44 | **36** | | MT | 105.4 | 1.54 | **16** |
| SC | 221.0 | 1.61 | **36** | | SD | 106.7 | 1.52 | **16** |
| NJ | 173.3 | 1.94 | **34** | | ME | 102.1 | 1.40 | **14** |
| WA | 120.4 | 2.74 | **33** | | HI | 74.6 | 1.92 | **14** |
| DE | 183.0 | 1.79 | **33** | | VT | 102.2 | 1.38 | **14** |

Table 8: Relative values of F(HIV) expected if race and population density are the only determining factors. See text for how R and D were calculated

This should be compared to actual data from the general and generally healthy population. The nearest available such data is from applicants for military service, 1985--2000 [5], in which unfortunately the geographic distribution is given only by regions, not by individual States. Table 9 compares the odds ratios for F(HIV) in that source with the averaged RD values from Table 8 for the States in each of those regions; and the RD values for the 4 States mentioned in the cited source as those with the highest F(HIV).

**Table 9**

| States highest in F(HIV) | **Actual** F(HIV) highest States | | **Calculated** (Table 8) | R alone highest States | D alone highest States |
|---|---|---|---|---|---|
| DC | 8.7 | | **970** | 350 | 28 |
| PR | 3.6 | | **57** | 290 | 2.0 |
| NY, NJ | 2.2 | | **55 (76, 34)** | 180 (190, 170) | 3.0 (4.1, 1.9) |
| | | | | | |
| | | | | | |
| other States ranked by regions | odds ratios for F(HIV) for reported regions | | **average for regions** | average for regions | average for regions |
| PA | Mid-Atlantic* | 3.5 | **42** | 140 | 2.9 |
| DE, FL, GA, MD, NC, SC, VA, WV | South Atlantic** | 2.5 | **41** | 190 | 2.1 |
| AR, LA, OK, TX | West South Central | 1.9 | **39** | 190 | 2.0 |
| AL, KY, MS, TN AK, CA, HI, OR, WA | East South Central & Pacific | 1.7 | **30** | 150 | 2.1 |
| CT, IL, IN, MA, ME, MI, NH, OH, RI, VT, WI | North East<br>East North Central | 1.4<br>1.3 | **33** | 130 | 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 | **25** | 120 | 2.0 |

Table 9: Comparison of calculated and actual relative values of F(HIV). Calculated from Table 8; 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)
* also includes New Jersey & New York, two of the highest States, already shown above
** also includes Washington DC, already shown above as highest

The agreement is gratifying, 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 a good match for the actual F(HIV) (as Table 8 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 [Bauer 2005]. 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 the same (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. Figure 11 is drawn from the calculated numbers in Table 8. In both cases, the most heavily shaded States number 6, the next two groups 16 each, and the unshaded group 10.

**Figure 10**

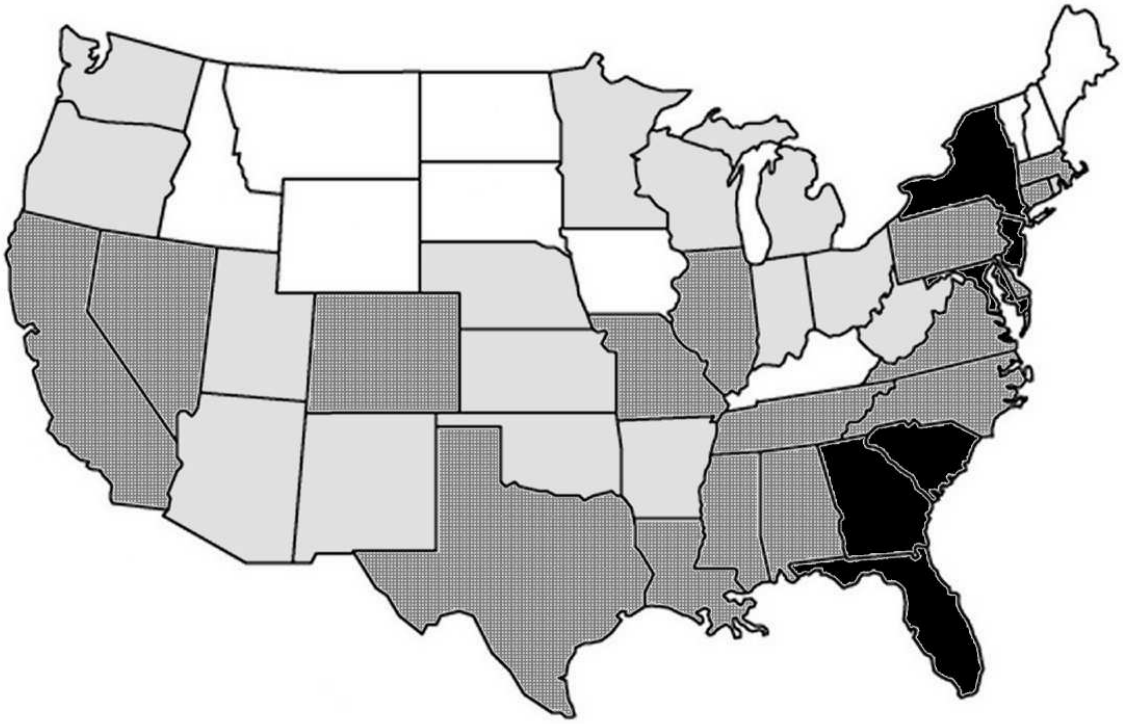

Figure 10: Observed geographic distribution of F(HIV), averaged over time and social groups from Figures 1--5 and 7--11 in [Bauer 2005]

**Figure 11**

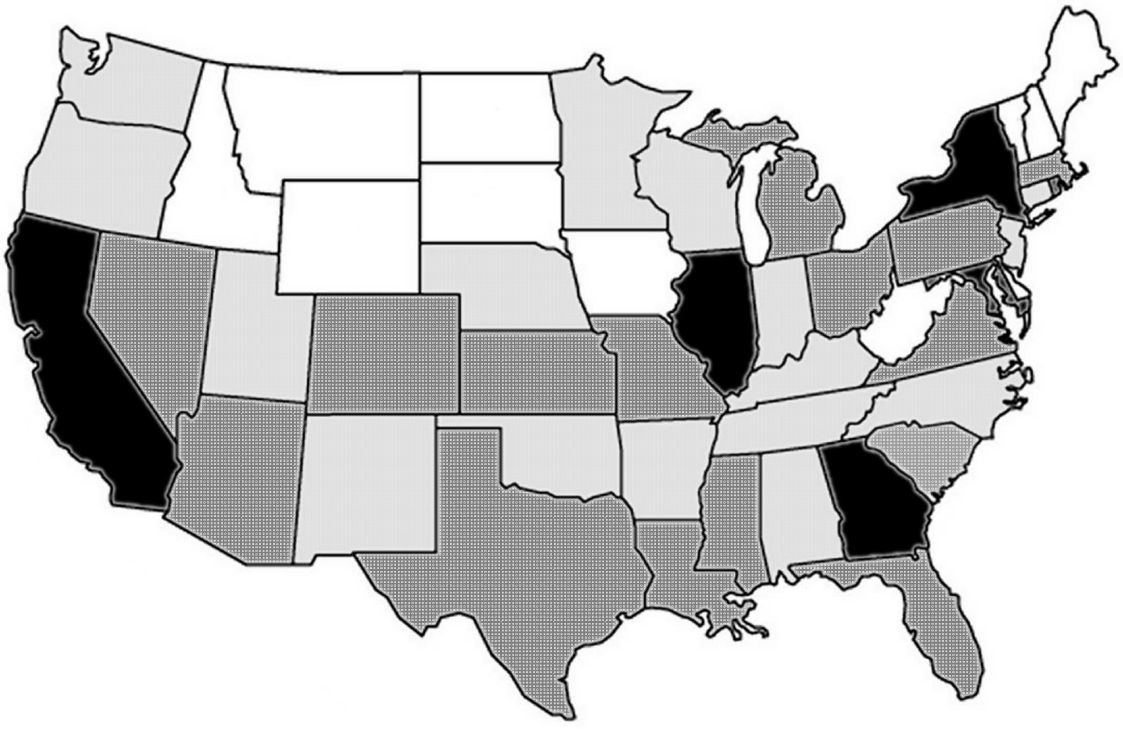

Figure 11: Calculated geographic distribution of F(HIV) if race and population density are all that counts; numbers from Table 8.

Again the agreement is rather good, bearing in mind that the grouping into 4 categories is arbitrary, as discussed at greater length in Part I. 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 9 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[15].

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 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". So this comparison supports the hypothesis that F(HIV) represents a physiologic response to a rather non-specific health challenge. 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 calculations yield a good fit--as they do--with actual data on smaller groups studied at different periods of time (the 10 various maps in Part I), 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.

### *HIV IS ENDEMIC IN THE HUMAN GENOME*

In Part I, 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 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 sign 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, are fully concordant with conclusions reached on the basis of retrovirology and molecular biology by Duesberg [36, 42] and by the Perth Group [41]. 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.

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 [35, 36, 37, 41--43, 62, 63, 65]. 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, [Malan])--numbers derived only from computer models that are unverified [Crichton] and whose very authors emphasize that the models need further refinement [126]. 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 [53]. Many authors have explained why the official view that Africa is being ravaged by AIDS is not only unproved but provably incorrect (for example, [62, 123, 124]). 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. As to the general situation of science and medicine in the 21st century that makes such mis-steps possible, the dominance of knowledge monopolies and research cartels [45] plays a large role.

The recognition that F(HIV) is both an indicator of physiologic stress and of "racial" heritage means that further research to understand the governing mechanisms and parameters may even lead to useful applications of HIV tests, as adjuncts to the monitoring of public health and to the tracing of human migration patterns. There are persistent hints in the data of several specific points worth following up: for one, it seems that Black women are unusually prone to show this physiologic response to stress--see Figure 9 for women who have just undergone the stress of childbirth; for another, it seems that the Black-to-White ratio is exceptionally high among the most carefully screened groups, blood donors. Investigation of such hints could shed more light on the whole business.

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

[3] Reports from public testing sites in two years had F(HIV) for Hispanics greater than that for Blacks--6.0% vs. 4.4% in 1990 [165], 4.6% vs. 3.6% in 1991 [166].

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

[5] Table 1 uses aggregated results for military applicants between 1985 and 2000 [5] in preference to published data on applicants for the smaller intervals 1993--97 [30] and 1991--92 [31]; and aggregated for 1993--97 for the Job Corps [30] in preference to the analysis for only a single year 1997 [27].

[6] 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.

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

[8] It was pointed out in Part II 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.

[9] Supporting references for this assertion, by years that represent the mid-points of ranges, are: 1986 [3, 6, 122]; 1987 [4, 7, 8, 15, 25]; 1988 [67, 70, 88]; 1989 [10, 14 , 19, 85, 94, 121]; 1990 [2, 20, 119]; 1992 [95]; 1993 [5, 22]; 1994 [26]; 1995 [1, 27, 30, 114]; 1996 [17, 18, 23, 29]; 1997 [27]; 1998 [115, 118]; 1999 [34]. The Black-to-White ratio of F(HIV) shows no trend over this period. As already pointed out, it is the same in all social groups.

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