

RACIST STEREOTYPES MEET HIV/AIDS

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ABSTRACT

Anywhere and everywhere, people of relatively recent African descent test HIV-positive at a significantly higher rate than do Asians, Caucasians, Hispanics, or Native Americans. That is undisputed. However, the dilemma presented by this fact has not been addressed in mainstream discourse about HIV/AIDS: One must either accept that African heritage predisposes inevitably toward unbridled sexual activity and resort to illegal drugs; or one must accept that HIV-positive is not a contagious condition that reflects sexual transmission or sharing of infected needles.

Commentators, including representatives of public institutions, have chosen to accept HIV/AIDS theory and thereby also--often implicitly but sometimes explicitly--a deterministic link between African ancestry and the injection of illegal drugs and the highly promiscuous practice of unsafe sex. In no other connection is so direct and deterministic a link between genes and behavior or culture acknowledged; that it is accepted without question here suggests that racist stereotypes remain embedded firmly, if perhaps subconsciously, among a significant proportion of those engaged in clinical research and medical practice.

The presumption that HIV originated in Africa, even though AIDS appeared in the United States years before supposedly spreading to Africa, illustrates further that racist stereotypes continue to find ready credence.

A recent analysis of HIV-test data reveals that what these tests have been detecting is not an infectious agent. That clears the way for a non-racist interpretation of racial disparities in the tendency to test HIV-positive.

Keywords: HIV/AIDS; Racism; Stereotypes; Genes and Race; Behavior and Genes

INTRODUCTION

AIDS was first noted and described in the early 1980s. In 1984, HIV--the human immunodeficiency virus--was declared the presumptive cause of AIDS ¹. Already in 1987, Chirimuuta and Chirimuuta (1987/1989) pointed out that much of the rhetoric and speculation as to HIV, AIDS, and Africa rested on racist stereotypes. In 1989, Konotey-Ahulu (1989/1996) underscored that point, albeit from a somewhat different perspective. They emphasized that ascribing the origin of AIDS to Africa flies in the face of a number of facts, notably that AIDS first appeared in the United States. To buttress a purported African origin, mere anecdotes found uncontested publication in the medical literature: African behavior, in particular sexual practices, was described in terms both pejorative and at variance with reality. The circumstances in Africa continue to be described in similar terms; a central claim is that multiple concurrent sexual relationships are the norm there but not elsewhere (Chin 2007; Epstein 2007).

In the United States, when AIDS first appeared in the 1980s, the victims were predominantly gay men and mostly white. Increasingly since the 1990s, however, HIV/AIDS in the United States has become associated disproportionately with black communities, and in particular with black women. That is typically interpreted in ways consistent with standard racist stereotypes concerning sex and drugs, augmented by the shibboleth of “the down-low”: black men who become HIV-positive through sex with other men and then infect their unsuspecting female partners. That black men are disproportionately represented in prison populations, where sex between men is rife, has been offered as further evidence for this sort of scenario.

However, Ford et al. (2007) have pointed out that there is little evidence for, and significant evidence against, regarding the down-low as more prevalent in black communities than in others ². They also discuss the general influence of stereotypes on research about racial disparities in the incidence of HIV/AIDS. But they do not question the basic assumption that HIV is spread via sex and needles, and thereby side-step the fundamental dilemma: If HIV is spread by mechanisms that bespeak deplorable behavior, how can one avoid deploring the mores of people among whom HIV spreads to a disproportionate extent? Since HIV-positive is attributed in largest part to sexual

transmission, these racial disparities can hardly be interpreted other than as illustrating “black sexuality as generally excessive, deviant, diseased, and predatory” (Ford et al., 2007). Thus the standard rhetoric about HIV/AIDS reinforces disparaging and debilitating racial stereotyping.

The most resounding and welcome resolution of this dilemma, indeed the only seemingly possible resolution, would be if HIV-positive were not linked inevitably to transmission via sex or the use of infected needles when injecting illegal drugs. Happily, that turns out to be the case. A recent survey of more than two decades of HIV tests in the United States reveals that “HIV-positive” is not the manifestation of a sexually transmitted agent; rather, it signifies a non-specific reaction by the immune system (Bauer 2005, 2006a, b, 2007).

This article summarizes the undisputed evidence for racial disparities in rates of HIV-positive; reviews data and opinion as to sexual transmission; reports some of the overtly or implicitly racist interpretations that have been offered, including by official sources; cites studies that undermine those interpretations; and draws attention to some of the implausible inferences proposed to bolster the notion of sexually transmitted HIV. Finally, attention is drawn to a non-racist interpretation of racial disparities in testing HIV-positive.

RACIAL DISPARITIES IN TESTING HIV-POSITIVE

Every comparison of HIV-positive rates across racial lines finds significantly higher rates among black people than among others, typically by factors of 5 or more. The central question then is this: Is testing HIV-positive associated directly in causal fashion with race--like, say, dark skin or sickle-cell anemia--or is the association an indirect correlate stemming from cultural or social influences? Such an indirect association might be, for example, carry-over from slavery and later persistent discrimination in the United States, whereby African Americans remain under-represented among economic and academic elites, over-represented among prison inmates, and generally more prone to unfortunate and undesirable circumstances.

The available data provide an unequivocal answer to this question. African ancestry is linked to a propensity to test HIV-positive just as directly as it is linked to sickle-cell anemia, in probabilistically causal fashion: not everyone of African ancestry carries the sickle-cell genes, but in any group of people, those of African ancestry carry sickle-cell genes far more often than do

people of non-African or non-Mediterranean ancestry. The salient evidence from HIV tests is this:

- In the United States, where the most detailed and most reliable data on HIV tests are available, similar racial disparities are seen in every social group, at all ages, in both sexes, in all localities. Irrespective of cultural or social milieu, in other words, the propensity to test HIV-positive is linked to racial ancestry.
- Similar racial disparities as to HIV tests are seen in other countries.
- Global figures reveal the same thing. On average, about 7% of sub-Saharan Africans test HIV-positive. For Caribbean residents, the average is around 1.5 to 2%, but as high as about 5% in Haiti. In other parts of the world, the rates are below 1%, usually well below. The differences between those three global regions correlate with the relative proportions there of people whose ancestry is African: in the Caribbean, the proportions of people of African ancestry vary between about 40% in the Cayman Islands and in Trinidad and Tobago up to 95% in Haiti (CIA 1998).

UNITED STATES

The Centers for Disease Control and Prevention (CDC) state unequivocally that black Americans test HIV-positive more often than white Americans in every tested group: blood donors, applicants for military service, hospital patients, homosexual and bisexual men, migrant farm workers, female prostitutes, residents of Belle Glade (FL), female drug abusers, female sex partners of persons at risk, and women with no known risk factors (*MMWR* 1989, 1991).

The national surveillance summary up to 1992 found black women 3 to 28 times more likely than white women to be HIV-positive (CDC 1992). Black drug abusers were nearly 4 times (Hahn et al. [1989], reviewing 92 individual reports) or nearly 5 times (CDC 1992) as likely to be HIV-positive as white drug abusers. A survey of most of the available data for the United States found the black-to-white ratio for tendency to test HIV positive to average between 5 and 6 (Bauer 2007, p. 51, Table 8). A report at the 14th Conference on Retroviruses and Opportunistic

Infections claimed that in Colorado, “black men and women were 4 and 24 times more likely, respectively, to acquire HIV than their white counterparts” (Young 2007).

In noting such actual numbers, however, it must be borne in mind that the propensity to test HIV-positive depends not only on race but also on the risk level of the tested group as well as on sex and age. Quantitative estimation of the influence of any one of these variables therefore requires multivariate analysis. Few of the available data have been so analyzed, therefore all reported numbers carry an inevitable range of uncertainty. Moreover, in high-risk groups where the average rate of HIV-positive is already high, it becomes mathematically impossible to find black-to-white ratios as great as those cited in the previous paragraph. For example, one study found a rate of HIV-positive of about 35% among white gay men and about 55% among black gay men (Chmiel et al., 1987), a ratio of only 1.6.

Another matter that encumbers interpretation of infection rates is that, increasingly over the last decade or so, data are being reported as “HIV/AIDS” cases, which lumps together HIV-positive people with no symptoms of illness and AIDS patients who are manifestly sick. Thus the Centers for Disease Control and Prevention reported national averages for “HIV/AIDS cases” for 2007 in the United States as 7.6 per 100,000 for Asians, 9 for whites, 28.5 for Hispanics, and 72.8 for blacks; for Navajo Native Americans, the numbers were 7.6 in 2005 and 7.8 in 2006 (Kaiser 2007c). But it is vital that a distinction between HIV and AIDS be maintained, because data for HIV and data for AIDS are gathered differently and in several respects point in different directions. For example, the rate of HIV-positive among black Americans has remained about 5 times that of white Americans since testing began more than two decades ago; by contrast, the relative occurrence of AIDS has changed dramatically, by a factor of more than 6, from a black-to-white ratio of 0.2 in 1981 to 1.3 by the late 1990s (Bauer 2007, p. 107, Table 28).

(Incidentally, this illustrates the lack of correlation between HIV and AIDS, but the focus of this article is on racial disparities in testing HIV-positive and AIDS will be mentioned only as it impinges on that main theme.)

An extraordinary fact is that, among those classed in the United States as Hispanic, the rates of testing HIV-positive are characteristically higher in the East than in the West. “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” (CDC 1992, p. 37). This difference has been reported for a range of social groups, including soldiers (Kelley et al., 1990), newborns (Davis et al., 1998), and drug abusers (Prevots et al., 1996). This points rather directly to the conclusion that the tendency to test HIV-positive is intimately linked to ancestry. “Hispanic” is an ethnic and not a racial category. Hispanics in the western United States are predominantly of Mexican lineage, having on average a much lower proportion of African ancestry than do Hispanics in the eastern United States where people from Puerto Rico and other Caribbean locales are strongly represented.

OUTSIDE THE UNITED STATES

In South Africa there are similar racial disparities on HIV tests, albeit the ratios are rather larger than in the United States. Among regular blood donors in South Africa, more than 1% of black women tested HIV-positive while fewer than 1 in 10,000 white women did so, a ratio of 100 (Barnes 2005). Other reports that did not separate men and women gave the black-to-white ratio for positive HIV-tests among blood donors as 12.6 (Martin et al., 1990) or about 25 (Manto 2004). For women at prenatal clinics a ratio of about 15 was reported (Grobbelaar 1992). For the South African population as a whole, Africans are 22 times more likely to be HIV-positive than whites (Tomlinson 2007).

Scientists in India ascribed to racial ancestry that HIV infection rates are higher in the South than in the North, and higher for Indians than for Caucasians (*Times of India* 2006).

European data are not as plentiful as from the United States, and they do not always include race as a studied variable. Still, whenever race is considered, similar disparities have been noted. For example, at an HIV clinic in London (UK), the rate of HIV-positives was about 4 times greater for black Caribbeans and black sub-Saharanans than for others (Sinclair et al., 2004). Among child-bearing women in the United Kingdom in 1997-98, the HIV-positive rates among women born in East Africa and in Central Africa were respectively 16 and 14 times higher than among the other women (Ades et al., 1999).

SEXUAL TRANSMISSION: DATA AND SPECULATION

Although the demonstration by Bauer (2005, 2006a, b, 2007) that HIV tests do not track an infectious agent is not yet generally accepted, researchers--if not the media or the general public--have long been aware of what amounts to essentially the same thing, namely, that HIV is “distinctively difficult to transmit” (Gallo 1991, p. 131). A score or more of studies in Africa, Haiti, Thailand, and the United States find uniformly that the risk of transmitting HIV via unprotected intercourse is on the order of 1 per 1000 acts (see citations in Bauer 2007, p. 44 ff.), prompting the judgment that “the transmission probabilities presented are so low that it becomes difficult to understand the magnitude of the HIV-1 pandemic” (Chakraborty et al., 2001). Such a rate of transmission seems indeed to preclude the possibility of an epidemic, since the latter requires a basic reproduction number (R_0) greater than 1: a given infected individual must, within a rather short time, infect more than one other individual if there is to be the rapid rise in total infections that marks an epidemic. At a transmissibility of 1/1000, each infected individual would have to have sex with more than a thousand others *within a short time* to make $R_0 > 1$.

When asked about this (Rehm 2006), Anthony Fauci³ was both misleading and unrealistic. He misled in saying, “it is not a one to one ratio by any means. It’s not you have one sexual contact, and therefore you’ll get infected. It’s a relatively low efficiency”; “not one to one” is a misleadingly far cry from 1 per 1000. Fauci was unrealistic in saying “since there is so much sexual activity . . . , when you compound all of the sexual contacts among people, one of who might be HIV infected, then you get the infection rates that we just spoke about where you windup getting five million new infections per year. There has to be a lot of sexual contact for that to occur. But, in fact, there is a lot of sexual contact going on everyday in the world”. The latter assertion is misleading as well as unrealistic since the probability of 1 per 1000 applies when one of the sex partners is already HIV-positive. Now the global average infection rate is only 1% (UNAIDS 1997, 2004, 2005, 2006). Since the overwhelming majority of people are in low-risk groups, the rate of new infections outside high-risk groups would expectably be negligible, and that expectation is ratified by the established facts: AIDS never spread into the general population (Fumento 1990) outside Africa or the Caribbean; “for most heterosexuals, the

risk from a single act of sex was smaller than the risk of ever getting hit by lightning” (Bennett and Sharpe, 1996).

To make the persistently reported $\sim 1/1000$ average probability jibe with the possibility of an epidemic, it was speculated that there must be occasions when the transmissibility is much higher. The occurrence of such hypothetical high-transmissibility periods is sharply delimited, however, by the observation that little if any actual virus can be found in most HIV-positive people--“HIV-positive” indicates the presence of *antibodies* thought to be specifically anti to HIV. Therefore the hypothesis became that there is a relatively short period immediately after infection during which the virus reproduces rapidly, thereby increasing greatly the probability of transmitting it to someone else (for example, Anderson and May, 1988; Pedersen et al., 1987). That hypothesis has remained a mainstay of orthodox opinion: “the virus’ ability to be transmitted peaks during the weeks after a person becomes infected” (Kaiser 2007a).

Note that this suggestion cannot be disproved, for there is no way of testing it directly. There are no signs by which researchers could become aware that someone has just become newly infected. Attempts to study transmission indicate that infection by HIV typically brings no symptoms with it, or at most such non-specific or flu-like symptoms as slight fever--“acute retroviral symptoms occur in only one-half of patients, and the signs and symptoms are nonspecific” (Daar et al., 2001); and it seems also that it takes a matter of weeks after infection before the infected person “seroconverts”--generates the antibodies that produce a positive HIV-test. Therefore, only circumstantial evidence is ever available as to possible dates and possible sources of infection. Any inference as to when an HIV-positive person became infected is based on a search for past experiences that might have been risky, yet that were associated either with no symptoms at all or at most with slight flu-like symptoms. These uncertainties are exacerbated by the fact that “HIV-positive” can be mimicked by dozens of other circumstances, even by pregnancy or by vaccination (Johnson 1996)--the relied-on tests for HIV “have been dogged by concerns with specificity” (Cohen and Pilcher, 2005).

Nevertheless, the notion has become accepted doctrine, that there is immediately upon infection a short period of high infectiousness, followed by a long period--about 7 to 10 years--of

very low infectiousness, followed again by a period of greater infectiousness when actual symptoms of illness manifest. To repeat, this is believed not because it has been directly demonstrated, but because no other scenario accommodates the facts, given that AIDS is supposed to be caused by HIV. But the validity of any model can only be established if its outputs can be compared with actual measurements, so this model scenario remains questionable.

On the other hand, those who deny that HIV is the proven cause of AIDS can point out that even this scenario finds actual circumstances difficult to explain. The most “optimistic” extrapolation from actual data for African populations succeeded only in raising the average transmissibility from 1 per 1000 to a postulated 1/250 to 1/50 in the first 3 weeks of an acute infection (Cohen and Pilcher, 2005). How dubious these estimates are is illustrated by the fact that one of the studies on which they are based monitored couples only every 10 months but employed mathematical models to claim that HIV transmission was more than 5 times as probable within 2.5 months after seroconversion as after 6--15 months (Wawer et al., 2005). For gay men a higher rate of 0.1-0.3 per act of anal intercourse has been postulated (Jacquez et al., 1994), but the inevitable assumptions make those numbers, too, guesses more than solid or realistic inferences from the actual data.

The upshot is this: To explain an epidemic spread of HIV, it is necessary to postulate that newly infected people, within a few weeks of infection, have a number of sexual partners who, in turn, also have several other partners within a few weeks of when *they* became infected.

This postulate may not be entirely or obviously absurd in circumstances like those when and where AIDS first appeared, namely, among “fast-lane” gay men in a few cities in the United States in the early 1980s. For descriptions of “fast-lane”, read for example Berkowitz (2003), Callen (1990), or Kramer (1978), or view, for example, the films *The Other Side of AIDS* (Scovill 2004) or *When Ocean Meets Sky* (Robey 2006). Callen estimated that he had 3000 sexual partners in a decade, and cites the Centers for Disease Control and Prevention to the effect that among the first 100 gay men with AIDS, the median number of life-time sexual partners was 1120 (Callen 1990, p. 5). It was not only a matter of promiscuity but also of general behavior, described by observers--themselves gay men--as “burning the candle at both ends and putting a blowtorch to

the middle” (Conlan 2004), with constant passing around of a variety of infectious diseases, frequent treatment with antibiotics, frequent ingestion of antibiotics as prophylactic against the anticipated infections--conditions compared to “third world countries where the water is contaminated by feces” (MacIntyre 2004). Dr. Josef Sonnabend is said to have warned his gay patients that recurrent infections, treatments, and generally dissolute behavior were likely to have dire consequences.

At any rate, the fast-lane lifestyle practiced by some ⁴ gay men following gay liberation may not be obviously inconsistent with the orthodox scenario of a short period of high infectiousness immediately following HIV infection. However, that scenario becomes more than problematic when it turns out that AIDS has become epidemic throughout sub-Saharan Africa; and that scenario surely cannot be invoked to explain why AIDS in the United States has come to affect predominantly African Americans.

RACIST INTERPRETATIONS

Consider a hypothetical situation. A particular malady strikes African American communities to a far greater extent than other American communities; it also strikes people of African ancestry in other countries to a far greater extent than it does people of other ancestries; and globally, the incidence is far higher in sub-Saharan Africa than elsewhere, with the Caribbean region affected less than Africa but more than elsewhere. Would one conclude that this malady reflects *behavior* that is determined by African ancestry, or would one look for a purely genetic cause, by analogy to sickle-cell anemia?

Surely one would consider a genetic cause not only as the more plausible but as the only possible one. Yet concerning HIV/AIDS, behavior is indicted and found guilty--solely because it has become dogma that HIV/AIDS is an infection spread via sex and unclean needles. Various speculations *postulate* behavior that people of African ancestry *must* be practicing, in order to explain why HIV/AIDS is so prevalent among them. That those speculations are even countenanced, let alone accepted without demurrer, may reveal how firmly embedded are racist stereotypes that see Africans as particularly prone to criminality and promiscuity.

AIDS epidemics have hit only two groups: communities of openly gay men in the developed world, and African communities. To give a single explanation for both means asserting that sexual behavior in sub-Saharan Africa is comparable to that of the “fast-lane” lifestyle. That sets a high bar for the behavior attributed to Africans: 20-40% of sub-Saharan Africans must be engaged in multiple concurrent sexual relationships to sustain the supposed epidemic (Chin 2007). . “Although there is ‘no single or satisfactory explanation’ for why HIV prevalence is higher in Southern Africa than the rest of the world, a ‘growing body of evidence points to the predominant role of one aspect of sexual behavior’-- that men and women often have ‘concurrent sexual partners over months or years’” (Kaiser 2007a). The same is postulated by Epstein (2007). Surely the willingness to contemplate that rests on subliminal racist stereotyping.

Although the books by the Chirimuutas and by Konotey-Ahulu do not question the connection between HIV and AIDS, they document wide-ranging incompetence on the part of early Western researchers (for example, Chirimuuta & Chirimuuta 1987/1989, pp. 38-9). That several of those researchers remain prominent in the field (for instance, Kevin de Cock, Peter Piot, Thomas Quinn) suggests a basis for questioning the competence of their later work as well. Both books point out that all the actual evidence suggests a non-African origin for AIDS. It was first recognized in the United States; had it come out of Africa, surely it would have migrated first to European countries with long colonial histories of African connections--Belgium, Britain, France, Germany. Konotey-Ahulu visited several African countries in the early 1980s, interviewing medical personnel in particular, and concluded that AIDS was an imported phenomenon. (Konotey-Ahulu himself, and Rosalind Chirimuuta, have distinguished records in medical practice and research.)

So obstinate is the presumption that AIDS must have originated outside the United States that the Food and Drug Administration reiterated in 1990 a policy that barred Haitians and sub-Saharan Africans from donating blood, even though at the time Haiti had an AIDS rate of 5.4 per 100,000, much less than New York State at 36, Puerto Rico at 43, and Washington DC at 82 (Antoine, Pierre and Page, 1990).

To make plausible an African origin of AIDS, a popular theory has it that HIV arrived in humans after jumping the species barrier and mutating from a similar simian virus. At first the jump was supposed to have been from African green monkeys, now chimpanzees are the favored species⁵. This could happen, it is alleged, because of such practices and beliefs as the following, ascribed to Africans with little if any evidential support:

- injecting monkey blood as a sexual stimulant (Chirimuuta and Chirimuuta 1987/1989, p. 161; Konotey-Ahulu 1989/1996, pp. 73-4, 83);
- dead monkeys used as toys by African children (Chirimuuta and Chirimuuta 1987/1989, p. 72);
- black children becoming sexually active at 12 or 13 (Chirimuuta and Chirimuuta 1987/1989, p. 161);
- interchange of blood in “blood brotherhood” rituals (Chirimuuta and Chirimuuta 1987/1989, pp. 31-2);
- having sex with a virgin as the only cure for AIDS (Chirimuuta and Chirimuuta 1987/1989, p. 148);
- “dry sex” (Baleta 1998);
- “widow cleansing”: widows must have sex with a relative of the deceased husband (LaFraniere 2005).

Such allegations have persisted (Kaiser 2006):

a “host of traditional ceremonies and practices” in rural parts of Africa are creating routes of HIV transmission that are unique to the continent

Some practices -- such as birthing ceremonies, scarification for ethnic identification, cutting for ritual healing, communal breast-feeding, injection of herbal medicines and group circumcisions -- might lead to a “wildfire spread” of HIV

So much for Africa. But how to cope with the fact that HIV/AIDS in the United States has come to be associated disproportionately with African Americans?

Just as in Africa, official explanations indict behavioral norms and practices, including a propensity for multiple concurrent sexual relationships, just because the orthodox view that HIV is sexually transmitted permits of no other explanation.

Such contemporary social circumstances as the disproportionate incarceration rate in the United States of black males, as compared to non-black males, may be attributable to cultural factors stemming from centuries of slavery and Jim Crow conditions as well as less overt discrimination. However, this type of explanation works only when racial disparities are sensitive to social circumstances. Incarceration rates are disproportionately high among young black males from inner-city locales where unemployment rates are high, and the sheer numbers involved ensure that national averages also show incarceration rates disproportionately high among young black males overall; but if one were to collect a sample of socially well-established, financially well-off families, and within that group were to compare properly matched black and white pairs, one would not find the same disproportion. Among prominent Hollywood celebrities, after all, the publicized instances of committing murder, dying of AIDS, driving under the influence, and shop-lifting have featured white Americans disproportionately.

On the other hand and by contrast, people of African ancestry *always* test HIV-positive significantly more often than others; all over the world, as well as *in all social milieus* in the United States: among blood donors, applicants for military service, members of the Armed Forces and Reserve components, members of the Job Corps, new mothers, newborns, college students, gay men, injecting drug users. Social circumstances are doubtless responsible for the higher incarceration rates of black males; but they can hardly be responsible for the fact that among those imprisoned, those of African ancestry test HIV-positive more often than others; or that among injecting drug users, those of African ancestry test HIV-positive more often than others. Disparities found in every economic and social group stem from something that is a direct expression of genetic characteristics just as are sickle-cell anemia, skin color, hair texture, and the like.

In spite of that, official and unofficial comments about HIV/AIDS attribute the higher rates of HIV-positive among black people to behavioral and cultural factors; because they have no other clue: “AIDS researchers don’t have a solid explanation for why black women in America have such a shockingly high prevalence of HIV infection” (Cohen 2004).

Absent solid evidence, explanations tend to be based on prejudices. The Centers for Disease Control and Prevention have this to say: “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” (CDC 1992, p. 37). In simpler language: multiple concurrent sexual relationships, moreover carried on carelessly without condoms, are the norm in black communities, and there is much mixing of those in low-risk groups with those in the high-risk groups of injecting drug abusers and men who have sex with men. To say that plainly would be unacceptable, of course, so we have these euphemisms couched in jargon; but the meaning is no different.

In similar fashion, the down-low has been accepted without apparent question, including within much of the black community: whenever a black woman turns out to be HIV-positive, her partner is automatically judged to have been on the down-low. Thus “Bill Duke, actor and director of ‘Sister Act 2: Back in the Habit,’ explained how his goddaughter became HIV-positive. She was infected by her boyfriend, who had slept with another man” (Samuels 2006). A *Newsweek* (2006) review of 25 years of AIDS included the following quotes from black women: “My fault was that I slept with my husband” (now her ex-husband); “I let my guard down with the wrong person”; a 20-year-old was “the victim of unprotected sex with a guy she thought was her soulmate”.

“Wilbert C. Jordan, MD, MPH recognized as the nation’s leading expert and researcher on HIV/AIDS among African-Americans doesn’t mince words when he warns Black men who have sex with men (BMSM) are destroying the fabric of Black America” (Levister 2006).

It has become quite routine to emphasize that HIV/AIDS in the United States is of particular concern to African Americans. There is a National Black HIV/AIDS Awareness Day, and

frightening statistics are promulgated: “In the U.S., Blacks account for half of new AIDS cases, while representing just 12% of the population. Among Black women aged 25-34, HIV is the number one cause of death, and a leading cause of death among the Black population overall, higher compared to any other racial and ethnic groups” (BET 2007). Described by its well-intentioned promoters as a message that “educates our viewers and evokes social change”, this is in point of fact propaganda that succumbs to racist stereotypes in accepting that something spread by promiscuous, carelessly unsafe sex and injecting of illegal drugs could be so much more rife among African Americans than among others. To the contrary: Whenever actual studies were carried out, they revealed the sexual behavior of Africans and African Americans to be if anything more restrained than that of Europeans.

EVIDENCE AS TO SEXUAL BEHAVIOR

A range of studies have reported that sexual behavior and practices among Africans and among African Americans are no riskier than among Caucasians or white Americans (Brewer et al., 2003; Gisselquist et al., 2002). As to Africans, Epstein (2007) debunks suggestions about such bizarre practices as “dry sex” and “widow cleansing”. In London (UK), “There were no significant differences between white gay men and those from other ethnic background in terms of sexual behaviour” (Dawson 2007). Bausell et al. (1986) found white Americans *less* likely than black Americans to take protective measures during sex.

The claim (Chin 2007; Epstein 2007) that heterosexual sub-Saharan Africans are as promiscuous and careless as high-risk fast-lane gay men, thus explaining the AIDS epidemic in Africa, runs counter to actual studies that find “HIV-positive gay men are more likely than HIV-positive black African heterosexual men and women to engage in sexual behaviour that presents a risk of HIV transmission” (Dawson 2007). Historical data from Zimbabwe record a higher incidence of venereal disease among the white South Africa Police and the British Armed Services than among the Native Police or among Africans in general (McCulloch 1999, pp. 205, 207).

The specific claim that sub-Saharan Africans engage in multiple concurrent sexual partnerships whereas people in developed countries practice serial polygamy is contradicted by

the London School of Hygiene and Tropical Medicine, whose survey in 59 countries found that “People in western countries tend to have more sexual partners than those in the developing world”. Moreover, as to the relation between promiscuity and venereal disease, “social factors such as poverty, mobility and gender equality may be a stronger factor in sexual ill-health than promiscuity” (BBC 2006).

Among young gay men in several urban centers in the United States, “potentially risky sex and drug-using behaviors were generally reported most frequently by whites and least frequently by blacks”, yet the black men were 9 times as likely as the whites to be HIV positive; evidently, “Understanding racial/ethnic disparities in HIV risk requires information beyond the traditional risk behavior and partnership type distinctions” (Harawa et al., 2004). The San Francisco Department of Health (1986) found no differences between races as to practicing anal intercourse. Samuel and Winkelstein (1987), too, had found no significant racial differences in behavior among gay men in San Francisco, and concluded that the black-to-white ratio of HIV-positivity could not be explained by differences in major risk factors. A decade-long study of gay men in New York reached the same conclusion: “wide racial disparities in seroprevalence were observed that were not attributable to disparities in risk factors such as STD, bisexuality, or acceptance of HIV testing” (Torian et al., 2002).

So too among injecting drug users: the racial disparities in HIV-testing could not be explained on the basis of behavior, for there were no significant differences by race in numbers of sexual partners, frequency of intercourse, numbers of sexual partners who were drug-injectors (IDUs), numbers of non-IDU sexual partners, prostitution, or intercourse with people then or later diagnosed with AIDS (Friedman et al., 1987).

Comparing young black and white women, “Although characteristics of sexual partners and relationships often differed by race, this did not explain racial disparities in STDs” (Harawa et al., 2003).

Also pertinent here is a study reporting that white Americans were at risk of becoming HIV-positive if they engaged in high-risk behavior, whereas African Americans were at risk irrespective of their behavior (Hallfors et al., 2007), supporting the view that HIV-positive

reflects something genetic and not something behavioral. Consistent with that is the study by Adimora et al. (2006) which found that one quarter of the black HIV-positives in their sample had no history of the supposed high-risk factors of drug use, drug-using partner, or gay sex.

Altogether, then, when racial stereotypes as to black promiscuity have been subjected to actual examination in the context of HIV/AIDS, the evidence has contradicted the stereotypes. That official explanations for the distribution of HIV nevertheless draw on those stereotypes indicates that racist notions remain strongly embedded, albeit unacknowledged and perhaps unconscious, among many medical researchers as well as others.

IMPLAUSIBLE OR UNWARRANTED INFERENCES

Because HIV/AIDS is unquestioningly accepted to be infectious and sexually transmitted, discourse concerning racial disparities has also accepted unquestioningly certain stereotypes as to the behavior of people of African ancestry--stereotypes that in other connections have been thoroughly discredited. On the other hand, if one considers HIV/AIDS in the context of the hypothetical situation suggested earlier, one might be led to ask, "What is the actual evidence that HIV/AIDS is a sexually transmitted infection?" That question has been taboo since the mid-1980s in leading journals and forums, in company with its cousin, "What is the actual evidence that HIV causes AIDS?" Those who persist in raising these questions have not only been dismissed as discredited but even branded as immoral, on a par with Holocaust denialists (for example, Cameron 2005; Gallo et al., 2006). Yet the accumulated body of publications in the accredited medical scientific literature provides ample grounds for believing it more plausible that HIV is not a sexually transmitted infection than that people of African ancestry are genetically doomed to behavioral practices that make them particularly vulnerable to venereal diseases.

So firmly entrenched is the belief that HIV is spread via sex that plain evidence to the contrary is ignored. Thus, "The whole issue of black men in prison is one we also have to talk about if we're serious about this," one pundit said (Jackson 2006), referring to a report on many years of observation in Georgia prisons. That report finds that 88 out of 45,000 prisoners became HIV-positive while incarcerated, about 6 per year, about 1 in every 7500 per year (Brown 2006). That rate is 5 or 10 times *lower* than the rate typically observed among monogamous heterosexual

couples, which contradicts the conventional wisdom that HIV transmission in prisons is enhanced because gay sex is rampant in prisons. There are other studies as well that found no increased HIV transmission among prisoners (see references cited by Bauer 2007, p. 47), and further studies that found the apparent rate of HIV transmission among gay men no greater than the apparent rate of heterosexual transmission (see references cited by Bauer 2007, pp. 46-7).

Another assertion contrary to fact as well as logic came from “Robert Janssen, director of CDC’s Division of HIV/AIDS Prevention, [who said that] blacks do not engage in riskier sexual behavior compared with other groups, but the population’s HIV/AIDS infection rates mean that blacks who have sex with other blacks are more likely to get HIV than people in other ethnic groups” (Kaiser 2007b). How then did the infection rate become so high in the black population in the first place, if behavior there is no riskier? Especially since AIDS appeared first in largely white communities of gay men? One factor adduced to explain the higher incidence of sexually transmitted diseases in black communities is that “sexually transmitted infections stay within the African American population because their partner choices are more segregated (assortative mating) than other groups” (Laumann and Youm, 1999)--which makes the initial transition of HIV into black communities from predominantly white communities of gay men yet more implausible.

Uganda has been held exemplary for its success in curbing the spread of HIV: In the course of a dozen years, the rate of infection is said to have declined from about 18% to about 6 or 7% (Austin 2006). But this is beyond belief. Since HIV infection is supposed to be permanent and irreversible, such success would require either that an additional 1% of the population died each year while no new infections occurred, or that the population trebled, or some combination of those two inconceivable circumstances. In point of fact, the population grew at about 2.7%⁶ or about 3.1%⁷ per year. The death rate was about 1.85% in 2000⁶; if the normal rate had been augmented by an extra 1%, that could not fail to have been noticed. Perhaps even odder is the report that the number of couples in which only one partner was HIV-positive was equal to the number in which both were HIV-positive, bespeaking an extraordinarily low rate of transmission of HIV between partners. Also strange is how different are the reported HIV rates among

different ethnic groups: 15% for the Batoro (of Bantu affiliation) but only 1.7% among the Karimojong (of Nilotic affiliation).

The claimed decrease of HIV in Uganda remains something of a mystery. Since the spread of HIV is said to be powered by behavior, the only available explanation is changed behavior (see, for example, Epstein 2007). But it is incredible that any Government propaganda campaign, in Uganda or anywhere else, could change a population's sexual practices so drastically in the space of a few years. If that were possible, we should easily and promptly fix what is so widely seen as undesirable, the mounting number of children in the United States who are born out of wedlock. Consider, too, that people of African ancestry everywhere and anywhere test HIV-positive far more often than do others. If that is owing to behavior, then that behavior must be so firmly implanted as to be virtually genetic or hereditary--and thus surely impervious to a few years of official propaganda.

FACTS, RACIST STEREOTYPES, EVIDENCE, IMPLAUSIBILITIES

To summarize the foregoing:

1. All data comparing rates of HIV-positive by racial classification show people of African ancestry testing positive far more often than others--in all social groups, in all countries.
2. Because it is believed that one becomes HIV-positive only as a result of risky behavior, explanations for the racial disparities have been based on traditional racist notions as to the sexual habits of people of African ancestry.
3. However, studies aimed at elucidating this in more detail have invariably failed to find racial differences in behavior that could explain the disparities in HIV testing.
4. Nevertheless, highly implausible scenarios have been concocted in attempts to show that, all evidence notwithstanding, racial differences in behavior suffice to explain the HIV data.

Apparently the dogma is so entrenched, that HIV is sexually transmitted, that hard evidence is ignored about the failure to observe sexual transmission adequate to explain the spread of HIV/AIDS epidemics. Instead, official explanations entail racist notions that would not be countenanced if proposed in other connections than HIV/AIDS.

As mentioned at the outset, recent analyses of official data by Bauer (2005, 2006a, b, 2007) serve to demonstrate that what HIV tests detect is not a sexually transmitted agent. Further, those analyses indicate that testing HIV-positive reflects a quite unspecific reaction of the immune system. The question then becomes, is there any reason why people of African ancestry should respond particularly strongly to a range of challenges to the immune system?

CONCLUSION: A NON-RACIST INTERPRETATION
OF RACIAL DISPARITIES IN TESTING HIV-POSITIVE

Bauer (2006b, 2007) suggests that since humans originated in Africa, where challenges to health from bacterial, fungal, parasitic and viral agents are omnipresent, evolutionary pressures are likely to have selected for strong immune systems. Humans who migrated to other regions no longer needed such powerful immune systems. So people of relatively recent African ancestry command stronger immune reactions than do Asians, Caucasians, non-black Hispanics, or Native Americans, whose African ancestry is between 100,00 and 200,000 years in the past. The situation is analogous to sickle-cell anemia: carriers of one (but not two) sickle-cell genes are better able to survive malaria than those who have none, and people whose ancestors came relatively recently from regions where malaria has long been epidemic--Africa or the Mediterranean--continue to harbor sickle-cell anemia genes at a disproportionately high frequency. Also analogous is the dark skin that is the most obvious sign of recent tropical ancestry: that opacity of the skin serves to shield against the production of too high a level of vitamin D; but humans who migrated away from the tropics needed to absorb an increasing proportion of sunlight in order to generate sufficient vitamin D, and therefore experienced a lightening of the skin.

Bauer (2007, pp. 100-1) cites as support for this view that, in temperate regions, black women are nowadays at higher risk for vitamin D deficiency than are white women (Semba et al., 2000); that people of African ancestry are at increased risk of rejecting kidney transplants (Creemers and Khan, 1998); that black people generate a stronger HIV-antibody response (Chaisson et al., 1991); and that various studies have found certain sets of immune-system genes to be strongly linked to racial classifications.

Highly suggestive is the fact that the San tribes of sub-Saharan Africa have a low rate of HIV-positive even though they live in Botswana and Namibia, where the incidence of HIV/AIDS is extremely high. The San are physically and genetically distinct from the sub-Saharan Bantu or Negroid type (Bauer 2007, p. 171), and--also known as Bushmen of the Kalahari Desert--they had adapted to desert life where bacterial, fungal, parasitic and viral agents are far less prevalent than in the non-desert tropics. The report noted above from Uganda, that the rates for HIV-positive were “15% for the Batoro (of Bantu affiliation) but only 1.7% among the Karimojong (Nilotic affiliation)” is similarly consistent with the view that the prime racial differentiator for testing HIV-positive resides in sets of inherited immune-system genes strongly correlated with the commonly used racial categories of human beings: the Nilotic Karimojong are genetically distinct from the sub-Saharan residents of Angola, Equatorial Guinea, Mozambique, and Rwanda (Gusmãoa et al., 2006).

In sum: The weight of the evidence is that the behavior, in particular sexual behavior, of people of African ancestry is not markedly different from that of other human beings. Disparities in rates of testing HIV-positive stem from ancestral lineages of immune-system genes. To insist that HIV is transmitted sexually entails giving credence to discredited racial stereotypes.

ENDNOTES

- ¹ At that time the virus was referred to as HTLV-III or HTLV-IIIB by one of those who claimed its discovery (Robert Gallo) and as LAV by the other (Luc Montagnier). The declaration was made at a press conference, by the Secretary for Health and Human Services.
- ² The same behavior was traditionally described as “being in the closet”, and that continues to be the usual term where gay or bisexual white men are concerned.
- ³ Fauci is director of the National Institute of Allergy and Infectious Diseases and the government’s top administrator concerned with HIV/AIDS.
- ⁴ A most important caveat: it is not being implied that gay men as such are extraordinarily promiscuous or drug-abusing. The AIDS epidemics of the early 1980s affected only a small minority of gay men (for example, Brown 1995), namely, those who were most publicly

visible because they had taken gay liberation as an invitation to incessant partying and public demonstrating. If promiscuity seems more prevalent among young gay men than among young heterosexual men, it is because their prospective partners are similarly inclined--the urge to indiscriminate sex is a characteristic of *male* animals by contrast to females (Adams 1989, p. 123 ff.).

⁵ Definite proof has been claimed that chimpanzees are “a natural reservoir of HIV-1” (Keele et al. 2006). Lay people might wonder why, if this is so, determined efforts to establish a model of HIV/AIDS in chimps have always failed (Donnelly 2006).

⁶ http://www.photius.com/wfb2000/countries/uganda/uganda_people.html

⁷ <http://devdata.worldbank.org/external/CPProfile.asp?PTYPE=CP&CCODE=UGA>

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