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Power, Race, and the Neglect of Science: The HIV Epidemics in Sub-Saharan Africa

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Abstract

This work addresses racial stereotyping and the effect it has in distorting AIDS policy for sub-Saharan Africa.

Keywords

race, HIV/AIDS, sub-Saharan Africa, healthcare, epidemic, racial stereotyping

Disciplines

African Studies | Diseases | Economics | International Public Health | Race and Ethnicity

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The HIV epidemics in sub-Saharan Africa

Eileen Stillwaggon and Larry Sawers

Conventional epidemiology recognizes that epidemics, like individual infections and injuries, arise in a specific ecological context. Epidemics are complex, contingent processes that develop through the interaction of characteristics of the pathogen, the vulnerable population, and environmental factors (Stillwaggon 2009). Whatever the proximate cause of infection or injury, epidemiology examines the multiple factors that influence both individual risk and the vulnerability of populations. In the last half-century, advances in microbiology and medical interventions as well as changes in the political and economic climate have shifted the emphasis of medical research to individual-level rather than population-level factors in theories of disease causation (Rose 1985; Schwartz and Carpenter 1999; Stillwaggon 2006; Susser 1985). Nevertheless, the standard epidemiological approach encompasses the ecological context of both individual and population risk.

Medical researchers who first studied acquired immune deficiency syndrome (AIDS) in sub-Saharan Africa in the 1980s looked at the epidemics through the lens of conventional epidemiology. Although isolated clusters or concentrated epidemics of human immunodeficiency virus (HIV) were found on all continents, primarily among men who have sex with men and needle-sharing drug users, in sub-Saharan Africa the syndrome was seen in men, women, and children. Poor immune status of the population, widespread infectious and parasitic diseases, poorly equipped and poorly staffed medical services, poor nutrition, and chronic exposure to contaminated water, soil, and food made the spread of HIV in Africa if not fully predictable at the start, at least comprehensible in an epidemiological framework. Because of the vulnerable population affected and the diverse presentations of the syndrome, it made sense to search not for one cause, but for the myriad factors that could contribute to the rapid spread of HIV.

By the late 1980s, in a radical departure from standard epidemiology, policymakers and most scholars narrowed their focus to a single proximate cause of HIV infection in sub-Saharan Africa: heterosexual behavior. Since then, the fundamental assumption dominating the AIDS-in-Africa discourse has been that something exceptional about sexual behavior drives the high prevalence of HIV in the region. We call that assumption and the policies that derive from it the behavioral paradigm.

HIV epidemics in sub-Saharan Africa are far larger than anywhere else. By 2009, HIV prevalence in the 9 countries of southern Africa averaged over 17 percent and in sub-Saharan Africa as a whole it was 5 percent. Outside Africa, HIV prevalence averaged only 0.5 percent (UNAIDS 2010). Two-thirds of people living with HIV are in sub-Saharan Africa.

This chapter examines the development of AIDS discourse on sub-Saharan Africa and its implications for policy, the exclusion of much scientific evidence in the prevailing theory of HIV causation, and some of the possible reasons for that departure from conventional epidemiology. We begin by discussing the behavioral paradigm that has dominated HIV policy research on sub-Saharan Africa. Then we examine its latest variant, the concurrency hypothesis. Next is a discussion of ecological explanations for the rapid spread of HIV in sub-Saharan Africa that reflect a standard epidemiological approach. Finally, we explore socio-political factors that have restricted the AIDS-in-Africa discourse to the behavioral paradigm, derailing research and policy from its original epidemiological foundation. Throughout the analysis, we examine the interplay of the ecological determinants of disease and the socio-political obstacles to understanding and controlling its spread.

The behavioral paradigm

In the 1980s in North America and Europe, there were substantial successes in reducing new infections of HIV, especially through promoting sexual behavior change in the gay community, but also by enforcing universal precautions in medical interventions, regulating blood-bank safety, and promoting needle-exchange programs. Transplanting interventions that had succeeded in North America and Europe to sub-Saharan Africa appeared to be a sensible response, but its effect was to displace the broader epidemiological approach of early HIV research in the region.

The ascendancy of the behavioral paradigm that replaced the use of standard epidemiological methods was enabled by long-held Western stereotypes of Africans. By the late 1980s, the presumption that some extraordinary characteristic or characteristics of heterosexual behavior in sub-Saharan Africa explained the high prevalence of HIV in the region was promoted in scholarly and popular literature and became widely accepted among researchers and policy makers (Caldwell and Caldwell 1987; Caldwell *et al.* 1989; Economist 2000; Ford 1994; Rushing 1995; UNFPA 1999). Influential and frequently cited works were characterized by sweeping statements about pan-African sexuality, supported, if at all, by anecdotal evidence dating from the early twentieth century to the 1970s. Through suggestive language and innuendo, they conveyed the impression of Africans bent on self-destruction because of cultural factors that differentiated them from everyone else (*inter alia*, Caldwell and Caldwell 1987; Caldwell *et al.* 1989). We discuss this issue at greater length below.

Anomalies in the behavioral paradigm

The hypothetical arguments of ethnographers and sociologists about "African sexuality" generated interest in empirical studies to evaluate their validity. Relatively few of those surveys of sexual behavior, however, were conducted in Latin America and Asia, reflecting the presumption that there was something unique about African sexuality. Surveys sponsored by the World Health Organization's Global Program on AIDS, the United States Agency for International Development's (USAID) Demographic and Health Surveys, and numerous others conducted by individual researchers and government statistical offices from 1989 to the present have produced a substantial body of survey research. They contradict the claim that behavior alone can explain the high prevalence of HIV in sub-Saharan Africa. The surveys demonstrate, on the contrary, that within every country studied there is considerable variation in sexual behavior—some people have many partners but most people have very few—and that prevalence of HIV across the globe does not correlate with patterns of risky behaviors, such as early sexual debut, extra- or premarital sex, number of partners in a year, or number of lifetime partners (for example, Cleland *et al.* 1995; Singh *et al.* 2000; Smith 1991; Turner 1993; UNAIDS 1999; Wellings *et al.* 2006).

Although survey research has found that risky sexual behavior is, if anything, less common in sub-Saharan Africa than elsewhere, HIV prevention policy for the region continues to focus almost exclusively on sexual behavior. While individuals who engage in risky sexual behavior are more likely to become infected with HIV within any population, the behavioral approach cannot explain prevalence of HIV in sub-Saharan African countries that is 10 to 250 times that of affluent countries in North America and Europe. The behavioral paradigm also provides no explanation for the greater vulnerability of individual sub-Saharan Africans compared to North Americans and Europeans.

In the 1990s, a second important empirical finding further undermined the validity of the behavioral paradigm. Researchers found that HIV is not a particularly virulent pathogen and that per-act transmission rates are quite low between otherwise healthy adults in heterosexual exposure (Boily *et al.* 2009; Chan 2005; Gray *et al.* 2001; Pilcher *et al.* 2007; Powers *et al.* 2008; Quinn *et al.* 2000; Wawer *et al.* 2005). Moreover, the infectivity of the person with HIV varies as the disease progresses. After an initial, brief spike in infectivity, transmission risk drops precipitously. From then until the onset of AIDS, the risk of transmission is so low that it calls into question the theoretical possibility of an HIV epidemic driven exclusively by heterosexual activity (Pinkerton *et al.* 2000). For an epidemic to be sustained, each infected individual must pass on the virus to at least one other person before death, but for most of the time that people have HIV, the risk of transmission is extremely low. To continue to characterize African sexuality as exceptionally dangerous, defenders of the behavioral paradigm had to respond to survey data that showed unexceptional sexual behavior in sub-Saharan Africa and evidence of the low infectivity of HIV in heterosexual exposures.

The attempt to rescue the behavioral paradigm

By the mid-2000s, the empirical evidence regarding sexual behavior in sub-Saharan Africa and HIV transmission rates, together with challenges to the behavioral paradigm by its few but persistent critics (inter alia, Packard and Epstein 1991; Stillwaggon 2000, 2001, 2002, 2003) led to a new variant of the behavioral paradigm. Conceding that most forms of risky sexual behavior are no more prevalent in sub-Saharan Africa than elsewhere, the promoters of the updated version of the paradigm argued that long-term overlapping partnerships, referred to as concurrency, are much more common in the region (Epstein 2007; Halperin and Epstein 2004, 2007). Furthermore, they argued that those concurrent partnerships spread HIV much more rapidly than sequential multiple partnering (Morris and Kretzschmar 1997, 2000). In a matter of a few years between 2004 and 2006, what had been occasional musings about concurrency in the 1990s by a handful of researchers (Hudson 1993; Watts and May 1992) were transformed into the new conventional wisdom. Since then, proponents of the concurrency hypothesis have continued to promote their cause (Epstein and Morris 2011; Mah and Halperin 2010; Morris 2010; Morris *et al.* 2009, 2010).

The explanation offered for the special efficiency with which concurrency is hypothesized to spread HIV is as follows: if a person infects his or her partner, but neither of them has another partner, then the infection is “trapped” until the partnership dissolves and new partnerships are formed. In contrast, overlapping partnerships can allow the formation of sexual networks through which infection can spread. For there to be an epidemic of HIV, however, those who become infected must have frequent sex with an uninfected partner during the first few months of infection because only during that brief period are per-sex-act transmission rates high enough to create or sustain a heterosexual epidemic (Epstein and Morris 2011; Halperin and Epstein 2004, 2007; Mah and Halperin 2010). Concurrency proponents argue that only long-term overlapping relationships provide the opportunity for sexual exposures frequent enough to generate the rapid spread of HIV.

Modeling concurrency

To show that concurrency spreads HIV more rapidly than other forms of multiple partnering, one must use mathematical models of epidemic dynamics. A pioneering model described in a series of articles published between 1996 and 2000 showed that concurrency could, under very unrealistic assumptions, spread HIV more effectively than serial monogamy (Kretzschmar and Morris 1996; Morris and Kretzschmar 1997, 2000). That model played a pivotal role in turning the concurrency hypothesis into conventional wisdom. The model was mathematically impressive, but also intimidating to those who had neither the temperament nor skills to unravel it. One version of the model found that “when one-half of the partnerships in a population are concurrent, the size of the epidemic after 5 years is 10 times as large as under sequential monogamy” (Morris and Kretzschmar 1997: 641). Astronomical rates of increase in HIV

generated by dazzling but impenetrable mathematics convinced many that the concurrency hypothesis was correct and muted the criticism of others.

To generate that tenfold difference, however, the model assumes a per-sex-act risk of transmitting HIV that is approximately 100 times the generally accepted estimate (Boily *et al.* 2009; Chan 2005; Hollingsworth *et al.* 2008), rates of concurrency higher than ever observed for any country, and equal rates of concurrency for men and women (although every survey finds women reporting far less concurrency than men). The model assumed that everyone had sex with every partner every day (up to four times a day, every day). Common sense and a substantial body of research show that the assumption is fantastical. One study, for example, reports on national surveys in five sub-Saharan African countries and shows that between 32 and 59 percent of adults with regular partners report no sex with their regular partner in the previous month (Caraël 1995). Another study of nine countries in the region finds that the frequency of sex for women in their first year of marriage ranged from 2 to 4.4 times per month and was much less frequent in later years of marriage (Brewis and Meyer 2005). Numerous other studies show the same pattern of infrequent sex in long-term partnerships (for citations, see Sawers and Stillwaggon 2010a: 3–4).

With the help of Alan Isaac, we have created a model that is identical to Morris and Kretzschmar's with two exceptions. It replaces their unrealistic rates of transmission with rates provided by widely respected authorities on the subject (Hollingsworth *et al.* 2008). In addition, our model, unlike Morris and Kretzschmar's, incorporates vital dynamics, that is, births and deaths. Simulating our model with the more realistic transmission rate produces HIV prevalence that is the same at every level of concurrency including serial monogamy, not the tenfold difference that Morris and Kretzschmar found. When half of partnerships are concurrent, HIV prevalence rises from 0.05 percent to 45 percent in Morris and Kretzschmar's model. Our model if anything overstates the increase in HIV prevalence, since the inclusion of vital dynamics accelerates the spread of HIV. Nevertheless, with realistic transmission rates, HIV prevalence in our model increases from 0.05 percent to only 0.06 percent.

Morris and Kretzschmar describe their article as a "proof of concept," but the promoters of the concurrency hypothesis used the results of the model to demand an immediate reorientation of HIV prevention policy to address concurrency (Epstein 2007; Morris 2010). The hypothesis is now dictating HIV prevention policy in many countries of sub-Saharan Africa. As early as 2006, the official position adopted by SADC (the consortium of the 15 countries of southern Africa) included concurrency as one of two key drivers (along with lack of circumcision) of the HIV epidemics in the region (SADC 2006; Shelton 2009).

Recent efforts to incorporate realistic assumptions about parameter values into the Morris and Kretzschmar model show that concurrency cannot be an important driver of HIV epidemics in sub-Saharan Africa. Eaton *et al.* adapted the original Morris and Kretzschmar model using transmission rates that reflect the current consensus among researchers and incorporating the variation in infectivity at different stages of infection. That modeling shows that HIV epidemics move to extinction if the prevalence of concurrency (the average of men and

women's rates) is 8 percent or less (point prevalence, that is, measured at a point in time; Eaton *et al.* 2010). Their results are a serious blow to the concurrency hypothesis since no survey using currently accepted questionnaire designs (UNAIDS Reference Group on Estimates 2009) has found any country-level point prevalence of concurrency in sub-Saharan Africa or anywhere higher than 8 percent. For example, recent surveys that were the first to use the methodology for measuring concurrency recommended by a group of experts convened by UNAIDS (UNAIDS Reference Group on Estimates 2009) found point prevalence of concurrency to be 5.1 percent in Lesotho (based on our calculations from the survey dataset provided by Measure DHS, www.measuredhs.com) and 3.7 percent in Malawi (National Statistical Office and ICF Macro 2011).

We have made a modification of Eaton *et al.*'s model to correct a problem that is found in most if not all other models of sexual network dynamics and HIV. Previous models simplified their analysis of sexual networks by assuming the same frequency of sex in every partnership. Models have assumed that people with two, three, or four partners have double, triple, or quadruple the frequency of sex as someone who has a single partner. Not only is that intuitively implausible, it contradicts all of the available evidence on coital frequency in multiple partnerships (Sawers *et al.* 2011). We incorporated empirically based assumptions about coital dilution—the lower frequency of sex in secondary partnerships—into Eaton *et al.*'s model. Doing so generates simulated HIV epidemics that move rapidly to extinction at any level of concurrency, including serial monogamy. Thus, properly constructed mathematical models show that the concurrency hypothesis cannot be correct because concurrency does not spread HIV more effectively than other forms of multiple partnering.

Concurrency in sub-Saharan Africa

For the concurrency hypothesis to be valid, concurrency not only has to spread HIV more effectively than other forms of partnering, it must also be substantially more common in sub-Saharan Africa than elsewhere. Our systematic review of studies published between 2004 and 2010 by the most prominent proponents of the concurrency hypothesis, finds that none of the studies they cite provides credible support for the proposition that concurrency is unusually high in sub-Saharan Africa (Sawers and Stillwaggon 2010a).

The proponents of the concurrency hypothesis whose works we examined repeatedly confuse data on concurrent and non-concurrent partnerships. They incorrectly report data from more than a third of the studies they cite. They compare data with different numerators (from different age brackets, for example) or different denominators (all adults, or those who are sexually experienced, or those who are sexually active). Most of the studies they cite cover cities or regions that are not representative of the country of which they are a part or are based on very small and/or non-random samples. Almost all of the studies they cite use definitions of concurrency that UNAIDS and the proponents of the hypothesis themselves argue are poor measures of concurrency and lead to overestimation (UNAIDS Reference Group on Estimates 2009). All of the errors,

inaccuracies, imprecise statements, and obfuscation exaggerate the difference between concurrency in Africa and concurrency elsewhere.

In short, after 20 years of trying, the proponents of the concurrency hypothesis have failed to demonstrate that concurrency exposes people to an especially high risk of HIV infection or that concurrency is especially common in sub-Saharan Africa. The notion that exceptional sexual behavior in sub-Saharan Africa explains the HIV epidemics there is without empirical support. Acceptance of the behavioral paradigm, however, has blocked consideration of other factors that contribute to high rates of HIV transmission in sub-Saharan Africa.

Other factors influencing the spread of HIV

The exclusive focus on African sexual behavior abstracts from the many other factors that influence health and vulnerability to any disease for people in the region. The majority of children born to HIV-infected mothers do not acquire the virus at birth. The difference in vertical transmission risks between sub-Saharan Africa and high-income countries was an early clue to the importance of ecological factors in HIV transmission. In the mid-1990s, before anti-retroviral therapy was introduced to prevent mother-to-child transmission of HIV, about 40 percent of infants born to HIV-infected mothers in sub-Saharan Africa were infected at birth, about 25 percent in the US, and about 14 percent in Europe (Fowler and Rogers 1996). That was evidence that differences in immune status of both infected (the mother) and uninfected (the child) individuals had an important effect on HIV transmission risk.

Furthermore, the overwhelming majority of sex acts with an HIV-infected person do not lead to transmission. For most of the time that a person is infected with HIV, his or her risk of infecting a partner is less than 1 in 1000 sex acts if both partners are otherwise healthy. Even in the 1990s, it was known that sexual intercourse was a proximate, but not necessarily sufficient, cause of HIV transmission. Thus, focusing almost all research and policy resources on a sexual explanation for high HIV prevalence in sub-Saharan Africa was a radical departure from conventional evidence-based epidemiology that considers contributing factors. That diversion of research and policy funding continues to the present day.

While the behavioral paradigm has dominated HIV prevention policy and social science HIV research of the past 20 years, some medical researchers continued to employ the conventional epidemiological methodology, exploring the ecological context in which the AIDS epidemic was spreading in sub-Saharan Africa. As we demonstrate, a substantial body of scientific literature indicates that host and ecological factors play an important role in determining an individual's vulnerability to HIV infection and the contagiousness of HIV-infected partners (and mothers). Poor nutrition and parasitic and infectious diseases weaken the immune system and make people more vulnerable to infection with HIV as they would for any disease, however transmitted (Bentwich *et al.* 1995). Moreover, certain diseases, discussed below, are highly prevalent in Africa and provide more efficient transmission routes for HIV during heterosexual and vertical (mother-to-child) exposures.

Nutrition

From 1988 to 1998, when nascent or concentrated HIV epidemics developed into generalized epidemics in sub-Saharan Africa, 30 percent of the population of the region was malnourished (World Bank 1998). Malnutrition increases vulnerability to infectious and parasitic diseases generally, increases HIV viral load and viral shedding, and undermines the integrity of the skin and mucosa, thereby increasing sexual and vertical transmission of HIV (Beisel 1996; Chandra 1997; Fawzi and Hunter 1998; Friis and Michaelsen 1998; John *et al.* 1997; Landers 1996; Nimmagadda *et al.* 1998; Pelletier *et al.* 1995; Semba *et al.* 1994; Stillwaggon 2006). While poor nutrition is a serious problem in parts of Asia and much of Latin America, sub-Saharan Africa was the only world region in which protein and calorie consumption declined from 1970 to 1990 (UNDP 2000).

Malaria

More than 90 percent of all acute malaria infections occur in sub-Saharan Africa. Malaria increases viral load up to ten times for as much as seven weeks after an episode of fever, and that can double heterosexual transmission (Abu-Raddad *et al.* 2006; Hoffman *et al.* 1999; Whitworth *et al.* 2000). The World Health Organization estimates that there are over 200 million cases of malaria in Africa every year and 750,000 deaths (WHO 2008b: viii). An HIV-infected person could have elevated viral load for more than half of every year if repeatedly reinfected.

Schistosomiasis

Virtually all cases of urogenital schistosomiasis occur in Africa, and it afflicts more than 100 million people in the region (WHO 2008a). Schistosome worms (*Schistosoma hematobium*) and their eggs colonize the reproductive tract in men and women, causing inflammation, viral shedding, and genital ulcers that increase transmission of HIV (Attili *et al.* 1983; Feldmeier *et al.* 1995; Leutscher *et al.* 1998; Marble and Key 1995). Women with genital ulcers of schistosomiasis have three times the risk of being infected with HIV as women in the same village without genital ulcers of schistosomiasis (Kjetland *et al.* 2006). According to the World Health Organization (WHO), schistosomiasis is highly prevalent in every country in sub-Saharan Africa, including high HIV prevalence countries such as Botswana, Swaziland, Lesotho, South Africa, and Namibia, and in no other country except Algeria (WHO 2004). A recent double-blind, controlled trial found that treating ascariasis (caused by a soil-transmitted intestinal worm) in HIV-infected persons results in a statistically significant increase in CD4 counts (Walson *et al.* 2008). That suggests that a simple, inexpensive (2 US cents) and effective deworming medication could allow HIV-infected people to be healthier, reducing the risk of infecting their partner, and allowing postponement of antiretroviral therapy.

Non-sexual transmission

In addition to disease co-factors that can increase per-act transmission rates during heterosexual and vertical exposure, we must also recognize that non-sexual modes of transmission could play an especially important role in sub-Saharan Africa and among other poor populations. There are numerous, common medical blood exposures (for example, injections with unsterilized syringes, blood transfusions, catheter and intravenous placements, and internal obstetrical examinations) and non-medical blood exposures (for example, barbering and hairdressing, tattooing, scarification, injections given by non-medical personnel, and intravenous recreational drug use) that can potentially transmit HIV (Brewer *et al.* 2003; Deuchert and Brody 2006; Gisselquist 2008). Some have argued that civil war, high levels of violence, and associated sexual assaults on women accelerate the spread of HIV (Mworozi 1993; Serwadda *et al.* 1985). Nevertheless, they also disrupt the healthcare delivery system and thereby reduce iatrogenic and nosocomial transmission (infection spread by medical treatment or in a healthcare facility), so the net effect is unclear (Gisselquist 2004). Even if each one of those possible non-sexual routes of transmission produces only a small share of new infections, together they would play an important role in the epidemics of sub-Saharan Africa and provide an explanation for why at the present time more women than men are infected.

By 2004, most of the information about the connection between HIV and malaria, schistosomiasis, helminthes, and malnutrition was available in the medical literature. There was also ample evidence that HIV transmission through the many kinds of blood exposures were common. Nevertheless, that evidence was ignored by most of the HIV/AIDS community. In light of the conventional epidemiological understanding of disease synergies and the evidence that interactions of specific parasites and infections increase vulnerability to and contagiousness of HIV, the exclusively behavioral focus of AIDS policy reveals a very simplistic notion of disease causation. Moreover, transmission of HIV between men is much more efficient than heterosexual transmission (Chan 2005), but the role of men having sex with men in sub-Saharan HIV epidemics has been almost completely ignored. The single-minded focus not just on sexual behavior but also on heterosexual behavior, and the refusal to consider the ecological environment of HIV in Africa (which increases the transmission efficiency of a virus that is normally not very infectious), have been a costly detour. That has caused a deadly delay in addressing the true drivers of HIV in sub-Saharan Africa and other impoverished populations.

AIDS policy became derailed, but why is it still off track?

We have argued that there is, at best, feeble evidence supporting the behavioral paradigm and that there are viable alternatives for which substantial scientific support is at hand. Why then does the HIV/AIDS research and policy community cling to the behavioral paradigm while ignoring more plausible explanations for sub-Saharan Africa's extraordinarily high HIV prevalence? We argue that, in the

decades-long project of addressing AIDS in sub-Saharan Africa, politics won out over science. One of the most important aspects of political dominance is the ability to define problems and to define groups, both the Self and the Other. Westerners (that is, high-income countries) dominate international organizations and bilateral donor organizations in research and policy, and Western views hold sway even among elites in poor countries. Politically dominant organizations, including academia, have defined AIDS discourse.

Racial stereotyping

In the past, racism played an important role in providing the justification for slavery and then colonialism, and old ideas can linger in the public psyche long after they are directly useful. Racial stereotypes continue to pervade Western culture, casting their shadow over scholarship and public policy, even among persons who, on a conscious level, vigorously and sincerely oppose racial discrimination.

Gunnar Myrdal, a Swedish economist writing about race relations in the United States, observed that cultural influences “pose the questions we ask; influence the facts we seek; [and] determine the interpretation we give these facts” (Myrdal 1944: 92). He continued: “Biases in research ... are not valuations attached to research but rather they permeate research ... [and] insinuate themselves into research in all stages, from its planning to its final presentation” (Myrdal 1944: 1043). The influence of notions of “race” in both the popular mind and in the imagery of science is insidious and difficult to counter because so much of racial stereotyping is in the “unstated assumptions and unthinking responses” (Dubow 1995: 7), rather than in explicit postulates. That is aggravated by the tendency for both academic and journalistic writing about sub-Saharan Africa to consist of a “repertoire of amazing facts” (Coetzee 1988: 13). Writing about sub-Saharan Africans, popular and scholarly, almost always emphasizes how they are different from others, not their commonality with people everywhere. Given the presumption of difference with which authors, readers, reviewers, and editors begin, there is a much lower bar for what constitutes evidence regarding Africans, as long as what is written fits those preconceived notions.

Notions of racial difference pervade the social science literature on AIDS in Africa and were especially explicit during the first 15 years of the epidemic. No one uses the word race, but the notion enters into the discourse as “culture.” Two of the most frequently cited anthropological works of the first two decades of the AIDS pandemic employ a foundational metaphor to convey the idea that modern-day sub-Saharan African fertility and sexual behavior choices derive from a religious world view that harks back to the dawn of humankind. The authors propose “a focus on Africa as the domain of *Homo ancestralis* ... [to] explain many African anomalies” (Caldwell and Caldwell 1987: 410; see also Caldwell *et al.* 1989). Those articles never say that there are primordial genetic differences that set sub-Saharan Africans apart from everyone else and thus explain “African anomalies,” but the use of species terminology and italicized Latin words in the metaphor “*Homo ancestralis*” inescapably makes that point.

The metaphor is especially effective in a discussion of sexual behavior because so much of the racial difference literature of the nineteenth and early twentieth centuries focused on the sexuality of sub-Saharan Africans (Dubow 1995; Gould 1981; Stepan 1982). Racial science in an earlier epoch and popular racial stereotypes that persist to the present day stress sexual differences between the races, and portray sub-Saharan Africans as exotic, strange, and even disturbing (Gilman 1985, 1990, 1992).

In 1989, Caldwell *et al.* used the *Homo ancestralis* metaphor to explain sub-Saharan African AIDS in a social context they had already characterized as primeval. The premise of their interpretation of both fertility preference (Caldwell and Caldwell 1987) and HIV prevalence (Caldwell *et al.* 1989) is that sub-Saharan Africans are so different and their belief system so ancient that they are inscrutable to the Western (read "modern") mind. Their articles were cited in hundreds of scholarly works and policy documents as though they had established their argument empirically. Many other works in the 1980s and 1990s made similar or even less subtle arguments purporting to explain the prevalence of HIV in sub-Saharan Africa by asserting unusual cultural practices or cultural idiosyncrasies for which there was no empirical evidence (Delius and Glaser 2001; Ford 1994; Forster 2001; Rushing 1995). In spite of the lack of evidence, the theme in much AIDS scholarship and policy literature remains that "Africans are not like everyone else."

In the early years of the HIV epidemics, the fear that a heterosexual epidemic could engulf North America and Europe reinforced the desire to posit a sub-Saharan African "Other." High-income countries had very serious epidemics of other sexually transmitted diseases, so the potential for a heterosexual epidemic of HIV seemed quite probable. As the dimensions of the sub-Saharan African epidemics grew increasingly clear, racial stereotypes provided assurance that HIV was still restricted to specific types of people, that is, to the "Other." Westerners could find security in the belief that the African HIV epidemics were in a faraway place and among people with very different sexual behavior. As Roger Chapman describes it, HIV was framed "in terms of the types of people who were getting it, rather than the ways they were becoming infected" (Chapman 2009).

Blatantly racial arguments in the scholarly literature on sub-Saharan African HIV epidemics are rare now in contrast to the 1990s. More common are works that begin from an assumption about sub-Saharan African sexual behavior that is essentially comparative without providing comparative data. The literature abounds with broad, unsupported assertions about presumed differences in behavior that resonate with Western stereotypes of sub-Saharan Africans. We are not suggesting that the proponents of the concurrency hypothesis are attempting to make a racial argument. But the question they pose, as do all proponents of the behavioral explanation of HIV in sub-Saharan Africa, is this: how are Africans different from everyone else?

One example of the characterization of Africans as fundamentally different from everyone else is the repeated emphasis on transactional sex, or the exchange of sex for material gain. Proponents of the concurrency hypothesis frequently use the notion of transactional sex to bolster their arguments about concurrent

partnerships in sub-Saharan Africa (Epstein 2007; Halperin and Epstein 2007; Mah and Halperin 2010). Transactional sex, it is even claimed, is the “norm” in sub-Saharan Africa (Shelton 2009). Young women in sub-Saharan Africa are portrayed as poor, materialistic, and thus eager to trade sexual favors for gifts such as clothes, cosmetics, or a cell phone. It is never stated explicitly, but the necessary presumption for the argument to work is that sub-Saharan Africans (a billion people from 2000 ethnic groups) have attitudes condoning sex-for-gifts transactions that are different than elsewhere. The language used to make the argument, on its face, appears to emphasize the distinction between transactional sex and commercial sex work. But the only difference ever mentioned is that transactional sex sometimes involves gifts, not cash. In this way, the “norm” for sex in sub-Saharan Africa is collectively branded as prostitution. None of these discussions of transactional sex in sub-Saharan Africa is placed in a comparative context. Those who talk about transactional sex seem unaware that women receive gifts from lovers in every country on earth, including, for example, the practice of Valentine’s Day in Western countries. The transactional sex discourse is one more way to make sub-Saharan Africans “the Other.” It is about exceptionalizing their sexual behavior.

Western researchers, editors of academic journals, bilateral donor agencies, and international organizations have the power to define and delimit AIDS discourse in sub-Saharan Africa to the behavioral paradigm. That paradigm has abstracted from every other factor in the ecology of HIV dynamics, and all that remains in the explanation of high rates of HIV in sub-Saharan Africa is sexual behavior. The power to define AIDS discourse in behavioral terms is the most important part of determining research questions and policy responses. Because the question—how are Africans different from everyone else—seems so reasonable to most people, the behavioral paradigm and its latest variant, the concurrency hypothesis, became the conventional wisdom without a demand for credible empirical support. The answer to the question is not nearly as important as the question itself.

Institutional inertia

An ideology of racial superiority provided the justification for carving up Africa, Asia, and Latin America into colonies and spheres of influence. Moreover, in the late nineteenth and early twentieth centuries, the slowing birth rates of Europeans stoked fears in Europe and North America of being overwhelmed by people of color. Western stereotypes of people of color as strange, dangerous, and numerous were recast in the mid-twentieth century as the “population bomb.” That emphasis on “too many births” encouraged Western aid to developing countries to focus on sex and reproduction rather than on the need for food, housing, clean water, health care, personal freedom and safety, economic stability, and education, all of which influence the demographic transition, morbidity, mortality, and fertility preference.

The political environment of the 1980s and 1990s further narrowed the development agenda in the United States to little more than population control,

so organizations whose mission was to slow population growth played an increasingly prominent role in external assistance to developing countries. By the time that HIV prevention became an objective of US foreign aid policy, the behavioral paradigm was already firmly rooted in AIDS discourse for Europe and North America. Consequently, official and unofficial external assistance to HIV prevention efforts in sub-Saharan Africa focused almost exclusively on changing sexual behavior. Donors in the United States and elsewhere naturally turned to population-control organizations since they had decades of experience in talking about sex and condoms to sub-Saharan Africans. Except for those accustomed to promoting population control, discussing HIV was not within most people's comfort zone. Thus, population control organizations took the lead and ultimately were given wide latitude in shaping HIV prevention in sub-Saharan Africa.

Some sexual behaviors, such as having many partners, clearly raise one's risk of HIV infection. To the extent that the safe-sex message has been effective in encouraging behavior change (the experts do not agree), then the work of population-control organizations has played a role in slowing the spread of HIV in sub-Saharan Africa. But organizations whose institutional mission derives from their origins in population control necessarily view the people and the problem of HIV in sub-Saharan Africa in sexual and reproductive terms, rather than in epidemiological terms, or even more broadly in the context of social and economic determinants of health. That lent institutional momentum to the choice of sexual behavior change as the almost exclusive focus of HIV prevention policy.

An example is instructive: prevalence data suggested that international border crossings in sub-Saharan Africa were hot spots for HIV transmission because truckers were compelled to wait for as much as 10 days to clear customs. They found either that there were no hotels or that it was cheaper to visit prostitutes than pay for a room during their long stays at the border. A number of these border posts are, not surprisingly, in ecological zones with numerous disease vectors, since rivers form the borders in much of the region. Looking at the economic and ecological context, the obvious solution is to reduce costly and wasteful customs regulations that strangle trade and slow economic growth, as well as impeding cross-border trucking in ecological hotspots.

To resolve this source of epidemic spread of HIV at border posts, USAID engaged the resources of three population-control organizations that implement behavior change communication and condom distribution projects. Their solution naturally was behavior change and condom distribution. As stop-gap measures, neither behavior change nor condom distribution was a bad idea. Identifying the problem in such a limited way, however, merely created a permanent need for such programs. They ignored a critical opportunity for addressing a serious economic, political, and ecological problem in a sustainable and creative way. The choice of contract partners (and the power or latitude of USAID to choose those partners) reinforced the dominance of the sexual behavior paradigm that viewed sexual behavior as the principal driver of sub-Saharan Africa's epidemics to the exclusion of broader epidemiological and ecological considerations.

Path dependence and institutional interests

Institutional inertia is not limited to bilateral aid organizations and international population-control organizations that are beneficiaries of billions of dollars spent on HIV prevention programs in sub-Saharan Africa. There are also thousands of sub-Saharan African non-governmental organizations whose *raison d'être*, funding rationale, and daily activities, such as running training programs and focus groups, are based on the behavioral paradigm. In addition, national and subnational governments in sub-Saharan Africa have HIV control agencies, other governmental bureaus involved in HIV prevention, and whole divisions of ministries of health that channel the flow of dollars coming from government budgets and foreign donors. It is difficult for these organizations to consider programs outside the behavioral paradigm because of the institutional momentum that commonly characterizes all private and public bureaucracies. As one researcher who has interviewed most of the donor representatives and agency directors working on HIV prevention in Tanzania put it:

An organization specialized in abstinence workshops or information, education and communication campaigning, for instance, is unlikely to be a suitable implementing agency for non-behavioural structural prevention measures [such as working on co-factor infections]. A shift in today's prevention strategy would thus entail at least partial reallocation of existing resources. Given the organizations' incomplete convertibility from one activity to another, this reallocation would imply taking funding away from some organizations and giving it to others. As a result, there is a political constituency for keeping preventive priorities unchanged, while, as of today, no clearly circumscribed constituency exists that would push for the adoption of structurally oriented prevention measures.

(Hunsmann 2010)

Those bureaucratic fiefdoms with their thousands of jobs and millions of dollars in salaries then channel funds down to implementing partners. The creation of the Global Fund in 2002 and PEPFAR in 2003 produced a substantial acceleration of this avalanche of money, further cementing the power of vested interests in the behavioral paradigm. To accept that the paradigm is bankrupt would mean the loss of jobs, perks, and salaries of the many people whose careers and skills are linked to that paradigm (Hunsmann 2010).

That path dependence characterizes academic research as well. Researchers in universities in both high- and low-income countries have built their careers around elaborating and refining the behavioral paradigm. A social scientist who spends 20 years studying sexual behavior in sub-Saharan Africa cannot suddenly work in a laboratory studying malaria parasites and HIV replication. The behavioral paradigm is not just an intellectual concept; it is a multibillion dollar project. None of this is intended to say that people working in governmental and non-governmental organizations and academia are not sincerely, perhaps with great passion and energy, trying to turn back the HIV pandemic.

But institutional inertia allows the continued dominance of a faulty paradigm that ultimately undermines hope for success.

Conclusion

In summary, for a quarter of a century, HIV epidemics in sub-Saharan African have been defined almost entirely through the lens of heterosexual behavior despite the lack of evidence that it is a useful approach. There are other, more plausible explanations for the extraordinarily high prevalence of HIV in the region that derive from epidemiological methods and are supported by evidence. Although ecological explanations of HIV epidemics have a more solid basis in scientific evidence, they lack the political momentum of the behavior-in-Africa paradigm and thus they are for the most part either politely ignored or scornfully dismissed. The behavioral paradigm, which is organized around the portrayal of sub-Saharan Africans as different from everyone else, is starkly devoid of nuance. It comprehends neither the politics nor the ecology of HIV spread. It draws attention away from the differences between rich and poor countries in nutrition, clean water, waste disposal, and access to safe healthcare that are important factors in disease transmission but are less entertaining and lack the culturally confirming ring of racial and sexual stereotypes. The HIV/AIDS industry grew up around the behavioral paradigm and cannot easily change course. The dominance of the behavioral paradigm also shields the medical establishment from charges that it has unwittingly spread the infection through poor infection control procedures.

The focus of this chapter is on sub-Saharan Africa, where most people with HIV live. We argue that the region's nutritional and disease burdens are part of the ecological setting for sub-Saharan Africa's extraordinary HIV epidemics. Two of the diseases we point to as critical cofactor infections helping to spread HIV have unusually high burdens in sub-Saharan Africa. Although malaria is found in scores of countries, 85 percent of malarial morbidity and mortality occurs in sub-Saharan Africa where it infects more than a fifth of the population every year (WHO 2008a).

Almost everyone infected with *Schistosoma haematobium* lives in sub-Saharan Africa, and it afflicts more than 10 percent of the population of the region. Furthermore, HIV apparently originated in Africa; the epidemic was building in the region long before HIV was recognized by medical professionals, and long before it arrived in other developing countries. While those factors help to explain why sub-Saharan Africa is more affected than other regions, they do not explain the divergence in incidence between, for example, west Africa and eastern and southern Africa. HIV epidemics are complex, contingent processes with multiple interacting causes (Sawers and Stillwaggon 2010b; Stillwaggon 2009). No one has yet untangled completely the combination of factors that tipped the balance in sub-Saharan Africa, in particular in eastern and southern Africa, but the issues we raise in this chapter at least move the discussion in the appropriate direction.

This chapter has examined the socio-political reasons for the resilience of the behavioral paradigm despite its lack of empirical support, but there is reason

for optimism that change is possible. Most people accept the behavioral paradigm because everyone else does. The end of any paradigm is brought about by unexplained anomalies: questions that cannot be answered within the existing intellectual framework (Kuhn 1962). There is a growing realization that HIV prevention policy has largely failed. In some countries in sub-Saharan Africa, HIV prevalence appears to have fallen, but there is no clear agreement among the experts that HIV prevention programs have actually played an important role in that decline. Furthermore, HIV prevalence has increased or remained essentially the same in more than half the countries in the region. The obvious failures and, at best, modest successes of HIV prevention measures are motivating many to search for a new understanding of what drives HIV epidemics in sub-Saharan Africa. Basing inquiry on sound epidemiological principles and asking the right questions about the ecology of risk are the essential first steps for turning back HIV epidemics in sub-Saharan Africa.

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