

# THE ENIGMA OF HIV PROPAGATION IN AFRICA: MAINSTREAM THOUGHT HAS NARROWLY FOCUSED ON “HETEROSEXUAL SEX”

---

By

John J. Potterat

Short running title: Enigma of HIV propagation in Africa

Corresponding author: John J. Potterat

Affiliation: Independent consultant

Telephone: (719) 632-3120; FAX (719) 632-3032

Email: [jjpotterat@earthlink.net](mailto:jjpotterat@earthlink.net)

Website: <http://home.earthlink.net/~jjpotterat/>

#### ABSTRACT

*Introduction:* Three decades after the identification of AIDS, epidemiologists still do not fully understand HIV transmission dynamics in sub-Saharan Africa, nor its differential geographic and demographic spread.

*Discussion:* Despite mounting evidence suggesting a substantial role for nonsexual (puncturing) exposures in HIV transmission, researchers have not systematically investigated its impact on HIV propagation in Africa. Mainstream researchers initially reacted to this idea skeptically, then dismissed it in the short run as apostasy and chose to ignore it in the longer run. This research design flaw has been *the* Achilles Heel of efforts to explain the rapid propagation of HIV in Africa, a flaw that continues to this day - much to the detriment of scientifically trustworthy interventions.

*Conclusion:* A science that ignores potentially important modes of transmission, especially when confronted by challenging and respectable evidence, is inadequate and needs remedial attention.

#### Keywords

HIV epidemiology, HIV transmission, heterosexual transmission, iatrogenic transmission, HIV methodology, Africa

“A biased scientific result is no different from a useless one”  
■ Daniel Sarewitz [1]

## INTRODUCTION

Skepticism disturbs orthodoxy. Predictably, doubting often produces defensive reactions from believers, ranging from irritation to rage. Questioning the orthodoxy that sex is driving the HIV epidemics in sub-Saharan Africa began in a sustained, methodical way in the late 1990s, though early observers had sporadically highlighted the potential contribution of nonsexual transmission about a decade earlier [2-9]. Skeptics grounded their doubts on published studies of HIV in Africa whose results suggested that the reported data did not fit the “heterosexual transmission” view (a vague concept implying penile-vaginal sex), and on assessing the quality of the evidence for that assertion [10-12]. During the first dozen years of the new century, skeptics published more than fifty analyses and critical essays that consistently served to undermine the received wisdom [10, and refs in 13]. These skeptical scientists highlighted anomalies, conflicting evidence, as well as encouraged implementation of appropriately designed studies [10, 14-16]. The principal idea advanced by these skeptics was that resolution of these puzzling findings would follow, as would HIV control programs built on reliable information. This challenge to orthodoxy was greeted as apostasy in the short run and simply ignored in the longer run.

## DISCUSSION

The orthodoxy: weakly supported but pervasive

Nearly all public health workers and researchers focusing on AIDS in Africa believe – despite evidence and arguments presented by skeptics – that unprotected penile-vaginal sex accounts for the vast majority of HIV infections in African adults. This belief persists not only among those professionally charged with stemming the tide of HIV transmissions in sub-Saharan Africa, but also with the press and broadcast media, whose reportage in turn molds the world-wide public’s view of HIV transmission risks. Were this time-honored belief founded on high quality evidence or, at least, not undermined by persistent old [10] and new [17-26] empiric evidence suggesting a substantial role for nonsexual transmission, there would scarcely be a basis for rational dissent. (Nonsexual exposures refer to skin puncturing exposures in cosmetic, ritualistic, medical and dental settings, both formal and informal.) Upon closer examination, it turns out that the view that unprotected penile-vaginal sex drives Africa’s HIV epidemics rests upon both wishful thinking and relatively weak evidence [10-12].

Assessing quality of evidence

As shown below, the strongest evidence for sexually transmitted disease (STD)/HIV transmission patterns comes from bench science and from contact tracing [27], not from the sky (ecologic observations and inferences, or mathematical modeling, or armchair speculation). Contact tracing refers to seeking and testing the partners of HIV-infected cases to establish presence or absence of infection and to determine whether HIV infection is likely linked to sexual or/and other, nonsexual, exposures. Among case-partner

pairs, infection status may be unrelated (viz., HIV is absent or bears a different genetic “signature”). Risk factor assessment for incident infections can also provide good information, especially if researchers ask about and report all relevant risks, although it is not as revealing as is direct tracing and linking of infections, because studies of risk factors are usually population-, rather than person-to-person, specific. The table below provides a framework for gauging the relative strength and reliability of epidemiologic evidence in discerning STD/HIV transmission patterns.

**Table 1** Epidemiologic evidence for person-to-person spread of infection

<u>Viewing level</u>	<u>Distance from transmission events</u>	<u>Strength of evidence</u>
Ecologic	Far	Low
Risk factors	Far to intermediate	Low to medium
Contact tracing (With controls & DNA sequencing)	Very close	High

Preconceptions, misrepresentations, and incentives

When AIDS was first identified in the early 1980s, it was viewed as a principally sexually transmissible disease (STD) of hypersexual populations, especially sexually adventurous gay men. As for Africa, although several early studies implicated contaminated medical sharps as a possibly significant source of HIV spread [2-9], the framing of AIDS in the West as a disease of hypersexual populations fitted the time-honored Western view of Africans as promiscuous people [28]. Even though during the mid-1980s several reports recommended continued investigation of puncturing exposures in sub-Saharan African health care [2-9], this potentially important means of HIV transmission was gradually overlooked so that, by the time of the International AIDS Conference in Montreal in 1989 (which I attended), assessment of nonsexual modes of transmission virtually disappeared from formal presentations and, indeed, research agendas [10-11]. Henceforth, for all intents and purposes, HIV epidemics in Africa were assessed using only “heterosexual transmission” as the frame of reference, as were prevention initiatives. It was perhaps not a coincidence that the “heterosexual scare” of the West during the mid- to late 1980s was largely responsible for the arrival of substantial funding; this had not been forthcoming as long as only stigmatized populations (such as gay men and injecting drug users) represented the faces and future of the epidemic. That sub-Saharan Africa’s “heterosexual” HIV epidemics could be presented as the best example of the future of the epidemic elsewhere was a godsend for the allocation of adequate funding for research and prevention. Not only did the proclaimed heterosexualization of Africa’s HIV epidemics in published reports and at conference presentations close the door to conscientious investigation of modes of transmission other than penile-vaginal sex, but the generous funding responses to the threat of heterosexual HIV transmission elsewhere provided a powerful incentive to maintain the research status quo [29].

#### Asserting “heterosexual transmission” as principal mode

The evidence suggesting sex as principal mode of transmission in Africa’s adult populations was the nearly equal male-to-female ratio in diagnosed cases of HIV/AIDS, which contrasted starkly with the approximately 14 males to each female ratio in developed countries. This level of evidence was ecologic and hence distant from events on the ground (Table 1). At no point was there any effort to do contact tracing or to link infections on the ground - the gold standard for determining modes of transmission with confidence in person-to-person spread – as was done very early in the United States to demonstrate that a contagious agent was being sexually transmitted among gay men diagnosed with AIDS [30]. This view was also strengthened by the rarity of HIV infection in some but not all of the (alas, few) tested populations of African children aged 2-12 years [13, 31]. Moreover, the political pressure to maintain the idea of efficient penile-vaginal (implied in the word “heterosexual”) transmission may have played a key role in investigators’ decision to ignore nonsexual modes of transmission. This research flaw is *the* Achilles Heel of efforts to explain the rapid propagation of HIV in sub-Saharan African adults, a flaw that continues to this day [32]. This was probably not viewed as a flaw by investigators, who tended to discount the efficiency of HIV transmission in medical procedures [33] (other than blood transfusions). In addition, they believed that although medical injections might occasionally transmit HIV, such findings would be confounded by reverse causation (patients already infected with HIV frequently seeking treatment by injections) [34]. In brief, investigators prejudged the data: they put the cart before the horse without benefit of proper studies to demonstrate that reverse causation did explain any reported association between medical injections and HIV prevalence. Inadequately supported beliefs, coupled with incentives to maintain the impression that “heterosexual sex” was the dominant form of HIV transmission served to discourage both inquiry and skepticism [29].

#### Emergence of data that did not fit

Skepticism and further inquiry should have been stimulated by reports of important differences between the generally accepted view and information that did not fit. Serious anomalies surfaced by the 1990s, the principal one being that HIV did not consistently behave like STD. For example, STD are usually concentrated epidemics (viz., in core groups), while HIV in Africa was broadly distributed. Studies frequently showed little association between sexual risk factors (e.g., high rate of partner change, lack of condom use, sex with prostitutes, sexual concurrency) and HIV epidemics in communities [35-38]; in addition, some key sexual variables that *were* associated with common STD were *not* associated with HIV [39]. Indeed, opposite epidemic trajectories were reported for HIV and STD during the same periods in several African countries [40-41], that is to say, for example, that while HIV was rapidly increasing, STD were declining. Importantly, silent assumptions of African hyper-sexuality were challenged by results from multiple national surveys in the late 1980s and early 1990s showing that indicators of sexual activity in Africa were comparable to those reported in North America and Europe [42], where population-level heterosexual spread of HIV was not occurring. Remarkably, HIV was not infrequently diagnosed in persons denying sexual intercourse, as well as in persons reporting monogamous sex with an HIV-negative spouse [10-11]. Researchers generally discounted these findings by pointing out that there was something special about the

way black Africans had sex, or that HIV diagnoses were not sufficiently accurate, or that respondents lied about their sex lives [43-44]. Crucially, researchers failed to validate their speculations with proper scientific inquiry.

#### Mainstream response to skeptics

Mainstream researchers on the African HIV epidemics responded to skeptics viscerally, with shock, dismay, and anger – sometimes accompanied by ad hominem attacks. The World Health Organization in Geneva organized an influential response in *The Lancet* [34], where the authors dismissed the concerns of skeptics, using ecologic and risk factor evidence derived from the few and often inadequate (read: uncontrolled for puncturing exposures) scientific studies available, while also relying on inferential reasoning and appeals to logic, rather than data. Ironically, though the *Lancet* article’s authors conclude with a call for “*improved* (emphasis mine) data...to identify these risks...” in both formal and informal health care settings, the article served to discourage both doubt and inquiry, as many researchers and supporting agencies subsequently and characteristically dismissed or ignored contrary evidence, even their own [10, 45].

Thus, a decade of mounting evidence that did not fit the official framing of HIV transmissions in Africa regrettably failed to stimulate more rigorous studies to answer the fundamental question with confidence: Why Africa? Unsurprisingly, this question remains unanswered. It is stunning that, 3 decades into Africa’s HIV epidemics, no one can state with confidence why the continent has been so exceptionally burdened by HIV [32]. For 30 years, researchers and other observers have littered the epidemiologic landscape with armchair speculations about what might explain these epidemics: African sexuality, non-condom use, high genital morbidity and high STD burdens, lack of circumcision, HIV transmission-permissive tropical infections, polygamy, population migration and attendant prostitution, sexual concurrency, and age disparities between sex partners (older men coupling with very young women) [46-47]. In short: the dominant, if not exclusive, discourse in both professional and public arenas has been about “heterosexual sex”. Hence experts still don’t know [32].

#### Possible reasons for failure to investigate

It is disconcerting that – with so much at stake for successful HIV prevention in Africa – there has been sustained reluctance on the part of researchers and public health authorities to resolve incongruities in the orthodox view using comprehensive research designs. It is especially surprising given the high frequency of reports about health care transmission of blood-borne agents in both rich and poor countries [48]. Why have researchers not clamored for rigorous studies to verify their assertions and to rebuke the skeptics’ doubts? One can only speculate about which comfort zones – ideological, political, programmatic, financial, academic – were threatened or could account for their failure to voice doubt, at least publicly. Was it due to inertia? To time-honored assumptions about African promiscuity? To not wanting to discourage Africans from seeking modern health care (e.g., immunizations, prenatal care)? To wishing it to be so? To hoping that condom use would enhance population control initiatives? To fears of losing comfortable funding streams? To constructing a sense of shared coping with Western homosexual men and injecting drug users? To

fearing damage to academic or organizational reputation? To fears of public rebuke, scapegoating, or legal action? Painful as this process may turn out to be, answers to these questions must be sought. As previously and succinctly stated: “Unasked questions yield no answers” [49].

## CONCLUSION

A science that ignores potentially important modes of transmission, especially when confronted by challenging and respectable evidence, is inadequate. Had defenders of the received wisdom attempted to falsify their assertion – that unprotected “heterosexual sex” accounts for virtually all HIV infections among sub-Saharan African adults – by implementing studies which simultaneously and comprehensively assessed sexual (including heterosexual and homosexual anal intercourse) and nonsexual exposures (skin puncturing in injecting drug use and in cosmetic, ritualistic, medical and dental settings, both formal and informal [50]), and which traced infections on the ground [10, 50-51], prevention efforts based on rigorous designs might well have had greater impact on efforts to stem rapid propagation of HIV infection. It is encouraging to note that other independent researchers in Africa are presently calling for renewed and comprehensive investigation of “...factors [which] may contribute to the differential spread of the HIV pandemic in the region...” because “...sexual transmission alone may not explain HIV infections in Sub-Saharan Africa” [52]. Until scientifically trustworthy studies are implemented in various sub-Saharan regions, caution demands that skepticism about “Why Africa” regrettably remain the order of the day.

## References

1. Sarewitz D. Beware the creeping cracks of bias. *Nature* 2012; 485: 149.
2. Vachon F, Coulaud JP, Katlama C. Epidemiologie actuelle du syndrome d'immunodeficit acquis en dehors des groupes a risque. *Presse Medicale* 1985; 14: 1949-50. French.
3. Wycoff RF. Female-to-male transmission of AIDS agent. *Lancet* 1985;ii: 1017-8.
4. Wyatt HV. Injections and AIDS. *Trop Doc* 1986; 16: 97-8.
5. Imperato PJ. The epidemiology of the acquired immunodeficiency syndrome in Africa. *New York State J Med* 1986; 86: 118-21.
6. Potterat JJ. The AIDS epidemic and media coverage: a critical review. *Critique* 1987; 26: 36-8.
7. Piot P, Plummer FA, Mhalu FS, Lamboray J-L, Chin J, Mann JM. AIDS: an international perspective. *Science* 1988; 239: 573-9.
8. Packard RM, Epstein P. Epidemiologists, social scientists, and the structure of medical research on AIDS in Africa. *Soc Sci Med* 1991; 33: 771-83.
9. Minkin SF. Iatrogenic AIDS: unsafe medical practices and the HIV epidemic. *Soc Sci Med* 1991; 33: 786-90.
10. Gisselquist D. Points to consider: responses to HIV/AIDS in Africa, Asia and the Caribbean. London: Adonis & Abbey 2007. Updated version freely available at: [http://davidgisselquist.googlepages/points to consider](http://davidgisselquist.googlepages/points%20to%20consider). Accessed 10 Apr 2013.
11. Gisselquist D, Rothenberg R, Potterat J, Drucker E. HIV infections in sub-Saharan Africa not explained by sexual or vertical transmission. *Int J STD AIDS* 2002; 13: 657-66.
12. Gisselquist D, Potterat J, Brody S, Vachon F. Let it be sexual: how health care transmission of AIDS in Africa was ignored. *Int J STD AIDS* 2003; 14: 148-61.
13. Potterat JJ. HIV epidemiology in Africa: a changing of the guard. *Int J STD AIDS* 2009; 20: 812-5.
14. Brewer DD, Brody S, Drucker E, et al. Mounting anomalies in the epidemiology of AIDS in Africa: cry the beloved paradigm. *Int J STD AIDS* 2003; 14: 144-7.
15. Gisselquist D, Potterat JJ. Review of evidence from risk factor analyses associating HIV infection in African adults with medical injections and multiple partners. *Int J STD AIDS* 2004; 15: 222-33.
16. Potterat JJ. Randomized controlled trials for HIV/AIDS prevention in Africa: learning from unexpected results. *Future Virol* 2010; 5: 21-4.
17. St Lawrence JS, Klaskala W, Kankasa C, West JT, Mitchell CD, Wood C. Factors associated with HIV prevalence in a pre-partum cohort of Zambian women. *Int J STD AIDS* 2006; 17: 607-13.
18. Peters EJ, Brewer DD, Udonwa NE, et al. Diverse blood exposures associated with incident HIV infection in Calabar, Nigeria. *Int J STD AIDS* 2009; 20: 846-51.
19. Deuchert E, Brody S. The role of health care in the spread of HIV/AIDS in Africa: evidence from Kenya. *Int J STD AIDS* 2006; 17: 749-52.
20. Deuchert E. Maternal health care and the spread of AIDS in Burkina Faso and Cameroon. *World Health Pop* 2007; 9: 55-72.



21. Brewer DD, Potterat JJ, Roberts JM Jr, Brody S. Male and female circumcision associated with prevalent HIV infection in virgins and adolescents in Kenya, Lesotho, and Tanzania. *Ann Epidemiol* 2007; 17: 217-26.
22. Okinyi M, Brewer DD, Potterat JJ. Horizontally acquired HIV infection in Kenyan and Swazi children. *Int J STD AIDS* 2009; 20: 852-7.
23. Vaz P, Pedro A, Le Bozec S, et al. Nonvertical, nonsexual transmission of human immunodeficiency virus in children. *Pediatr Infect Dis* 2010; 29: 271-4.
24. Brewer DD. Scarification and male circumcision associated with HIV infection in Mozambican children and youth. *WebmedCentral EPIDEMIOLOGY* 2011; 2 (9): WMC002206.
25. Okwen MP, Ngem BY, Alomba FA, Capo MV, Reid SR, Ewang EC. Uncovering high rates of unsafe injections equipment reuse in rural Cameroon: validation of a survey instrument that probes for specific misconceptions. *Harm Red J* 2011; 8: 4. Available from <http://www.harmreductionjournal.com/content/8/1/4>. Accessed 10 Apr 2013.
26. Reid SR. Injection drug use, unsafe medical injections, and HIV in Africa: a systematic review. *Harm Red J* 2009; 6: 24.
27. Potterat JJ. Contact tracing's price is not its value. *Sex Transm Dis* 1997; 24: 519-21.
28. Epprecht M. *Heterosexual Africa: the history of an idea from the age of exploration to the age of AIDS*. Ohio/Scoville: Ohio University/University KwaZulu-Natal: 2008.
29. Hausmann M. Political determinants of variable aetiology resonance: explaining the African AIDS epidemics. *Int J STD AIDS* 2009; 20: 834-8.
30. Auerbach DM, Darrow WW, Jaffee HW, Curran JW. Cluster of cases of the acquired immune deficiency syndrome: patients linked by sexual contact. *Am J Med* 1984; 76: 487-92.
31. Gisselquist D, Potterat JJ, Brody S. HIV transmission during paediatric health care in sub-Saharan Africa – risks and evidence. *S Afr Med J* 2004; 94: 109-16.
32. Buve A, Laga M. Epidemiological research in the HIV field: towards understanding what we do not know. *AIDS* 2012; 26: 1203-4.
33. Gisselquist D, Upham G, Potterat JJ. Efficiency of human immunodeficiency virus transmission through injections and other medical procedures: evidence, estimates, and unfinished business. *Infect Control Hosp Epidemiol* 2006; 27: 944-52.
34. Schmid GP, Buve A, Mugenyi P, et al. Transmission of HIV-1 infection in sub-Saharan Africa and effect of elimination of unsafe injections. *Lancet* 2004; 363: 482-8.
35. Buve A, Carael M, Hayes R, et al. The multicenter study of factors determining the differential spread of HIV in four African cities: summary and conclusions. *AIDS* 2001; 15 (Suppl. B): S589-98.
36. Mapingure MP, Msuya S, Kurewa NE, et al. Sexual behavior does not reflect HIV-1 prevalence differences: a comparison study of Zimbabwe and Tanzania. *J Int AIDS Soc* 2010; 13: 45 (doi: 10.1186/1758-2652-13-45).
37. Lurie MN, Rosenthal S. Concurrent partnerships as a driver of the HIV epidemic in sub-Saharan Africa? The evidence is limited. *AIDS Behav* 2009; 14: 17-24. Doi: 10.1007/s10461-009-9583-5.
38. Sawers L, Stillwaggon E. Concurrent sexual partnerships do not explain the HIV epidemics in Africa: a systematic review of the evidence. *J Int AIDS Soc* 2010 (13 Sept); 13: 34. Doi: 10.1186/1758-2652-13-34.

39. Rothenberg R, Potterat J, Gisselquist D. Concurrency and sexual transmission. *AIDS* 2002; 16: 678-80.
40. Potterat JJ, Brody S. HIV epidemicity in context of STI declines: a telling discordance. *Sex Transm Infect* 2002; 78: 487.
41. Mhlongo S, Fiala C, deHaven E, Rasnick D, Stewart GT. HIV a sexually transmitted disease? An analysis of the latest antenatal screening for HIV and syphilis from South Africa. *Int J STD AIDS* 2003; 14: 574-6.
42. Wellings K, Collumbien M, Slaymaker E, et al. Sexual behavior in context: a global perspective. *Lancet* 2006; 368: 1706-28. Erratum in: *Lancet* 2007; 369: 274.
43. Allen S, Meinzen-Derv J, Katzmann M, et al. Sexual behavior of HIV discordant couples after HIV counseling and testing. *AIDS* 2003; 17: 733-40.
44. Gavin LG, St Louis M, Galavotti C. Converging evidence suggests nonsexual HIV transmission among adolescents in sub-Saharan Africa (The authors reply). *J Adolesc Health* 2007; 40: 290-3.
45. Whitworth JA, Biraro S, Shafer LA, et al. HIV incidence and recent injections among adults in rural southwestern Uganda. *AIDS* 2007; 21: 1056-8.
46. Epstein H. *The invisible cure: Africa, the West, and the fight against AIDS*. New York: Farran, Strauss & Giroux: 2007.
47. Chin J. *The AIDS epidemic: the collision of epidemiology and political correctness*. Abington, United Kingdom: Radcliffe: 2007.
48. Alter MJ. HCV routes of transmission: what goes around comes around. *Semin Liver Dis* 2011; 31: 340-6.
49. Brewer D, Rothenberg R, Potterat JJ, Gisselquist D, Brody S. HIV epidemiology in Africa: rich in conjecture, poor in data (reply to letter by Boily et al). *Int J STD AIDS* 2004; 15: 63-5.
50. Brody S, Potterat JJ. Establishing valid AIDS monitoring and research in countries with generalized epidemics. *Int J STD AIDS* 2004; 15: 1-6.
51. Brewer DD, Hagen H, Sullivan DG, et al. Social, structural and behavioral underpinnings of hyperendemic hepatitis C virus transmission in drug injectors. *J Infect Dis* 2006; 194: 764-72.
52. Duri K, Stray-Pedersen B. HIV/AIDS in Africa: trends, missing links and the way forward. *J Virol Antivir Res* 2013; 2:1  
<http://dx.doi.org/10.4172/2324-8955.1000107>

