
CHAPTER SEVEN

***WHY AFRICA? THE PUZZLE OF INTENSE
HIV TRANSMISSION IN HETEROSEXUALS***

excerpted from

SEEKING THE POSITIVES:

A Life Spent on the
Cutting Edge of Public Health

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SEVEN

WHY AFRICA? THE PUZZLE OF INTENSE HIV TRANSMISSION IN HETEROSEXUALS

This is a lean-forward, not a lean-back, chapter.

It is dedicated to Dr. Wallace Dinsmore,
unsung hero of this odyssey.

“The philosophies of one age have become the
absurdities of the next, and the foolishness of yesterday
has become the wisdom of tomorrow.”

—Sir William Osler

Enter David Gisselquist, PhD a few months before my retirement

It started in the autumn of 2000 with a telephone call from Dr. David Gisselquist, a Yale-trained economist with an interest in the puzzle *du jour*: AIDS transmission in Africa. It seemed improbable to him that “heterosexual” transmission of HIV could be responsible for the turbocharged epidemics being reported from different parts of sub-Saharan Africa, principally from its eastern and southern regions. In perusing the literature in medical and public health journals, it struck him that the official version ascribing these intense epidemics to sexual intercourse was poorly supported by the available evidence. Besides, intense transmission of HIV in heterosexual populations not engaging in injection of street drugs was not

being seen anywhere else in the world. So why Africa, which did not host large populations of injecting drug users? In the literature, he found a few articles by observers who doubted the official view espoused by the experts at the Centers For Disease Control (CDC) in Atlanta, the World Health Organization (WHO) in Geneva, and the recently formed agency in the United Nations (UNAIDS) also in Geneva. He contacted some of these skeptics,^{1- 6} one of whom was me. I told him that I was not very familiar with the HIV literature on sub-Saharan Africa, but suspected that use of unsterile needles in medical and ritualistic settings⁷, as well as local facilitating factors, such as tropical diseases that could damage the integrity of the tissues lining the genitals,³ might be involved. He then asked if I would be interested in working on this problem with him; my reply was that I would be at greater leisure to do so after my retirement at the end of January 2001.

Our collaboration begins

As it was, Dr. Gisselquist had been doing a disciplined review of the literature on AIDS in Africa published during the previous twenty years, compiling and analyzing the evidence. His tack was to assess the quality and completeness of this evidence rather than to construct armchair arguments in speculative space, which was the most common approach to the question: “Why Africa?”

An outsider to the field of STD/HIV epidemiology, Dr. Gisselquist found it difficult to publish his findings in this field’s professional journals. His first full-length article, which re-estimated the transmission efficiency of HIV through unsterilized medical injections, was rejected by three different

journals during 2001.⁸ Rejection slips for this and other manuscripts began to arrive at about the same time as my retirement. By then, I had also persuaded my long-term colleague Dr. Richard Rothenberg to assist in ameliorating Dr. Gisselquist's manuscripts for publication in medical journals. Working together and with other concerned observers, we published brief pieces (letters to the editor) in several medical journals calling attention to evidence inconsistent with the common view that sexual transmission could account for Africa's HIV epidemics.⁹⁻¹³ As a newly (July 2000) minted member of the International Journal of STD & AIDS' editorial board, I knew that this journal would be interested in receiving quality manuscripts that other editors might reject, especially if these submissions did not necessarily echo the received wisdom. Both of Gisselquist's rejected full-length manuscripts, now edited and improved by experienced epidemiologists, were soon published by the International Journal of STD & AIDS.^{8,14} These first letters and articles in 2002 marked the beginning of a long association between Dr. Gisselquist and me, along with several other colleagues who were to periodically contribute to subsequent manuscripts about AIDS in sub-Saharan Africa.

Impact of the early papers in the International Journal of STD & AIDS

The second of Dr. Gisselquist's two full-length articles, co-authored by Drs. Rothenberg, Ernest Drucker and me, presented evidence from the literature showing large numbers of HIV infections in sub-Saharan Africa not explained by either sex or mother-to-child transmission.¹⁴ This controversial paper challenged the official view that 90 percent of adult HIV cases in Africa had been contracted sexually; in Gisselquist's assessment¹⁵ the official

view was an assertion that had been decided without the facts, meaning a consensus was reached without the proper research to control for confound between sexual and non-sexual (skin puncturing) exposures. This 2002 paper was the one that triggered both furor and fury. It also stimulated the coalescing of an informal group calling for a new assessment of HIV transmission in Africa.¹⁶ During the rest of the decade these skeptics, the majority of whom were not trained epidemiologists, were to produce dozens of publications questioning the received wisdom about AIDS transmission in Africa. The furor was the firestorm of commentary in the media and on the Internet; the fury was the angry reactions in the halls hosting the orthodox view: the CDC, the WHO, and UNAIDS. It turns out that this was a relatively mild dress rehearsal for the furor and fury triggered by publication of four additional papers in the same journal five months later.¹⁷⁻²⁰ The Royal Society of Medicine, publishers of the International Journal of STD & AIDS, soon responded to this attention by making this provocative article freely available, if only because the Society aimed to encourage debate.

Our earliest publications about sub-Saharan Africa's HIV epidemics,⁸⁻¹⁴ all published in 2001–2002, provided converging evidence suggesting that much of HIV transmission could not be explained by sexual or mother-to-child transmission. The most glaring observation was that differences in sexual behaviors did not explain the different HIV epidemic trajectories on that continent. Although African hyper-sexuality was a time-honored stereotype in the western mind,²¹ scientific sexual behavior surveys conducted during the late 1980s and early 1990s seriously damaged this stereotype by showing that reported levels of sexual activity (read: rates of partner change) in a dozen African countries¹⁴ were comparable to those reported in European

and North American surveys. These levels were certainly not greater by the order of magnitude difference needed to explain the turbocharged epidemics on that continent. Given that HIV transmission probabilities per penile-vaginal exposure in Africa had been shown to be similar to those observed in Europe and North America,¹⁴ one would have to postulate not only greater rates of sexual partner change than studies were reporting but also phenomenal amounts of sexual activity for sex alone to account for observed levels of HIV infection in African adults.

In addition, studies contrasting the sexual behaviors of African adults living in areas of low- and high-prevalence showed little difference between these regions;²² for example, the variables one would expect to be related with sexual transmission of HIV, such as high rate of partner change, sex with prostitutes, sexually transmitted diseases, concurrent (as opposed to serial) sexual partnerships, dry sex (deliberately drying out the vagina to “tighten” it), and lack of condom use, were NOT more common in the high- versus low-prevalence regions. An intriguing observation was the complete disassociation in epidemic trajectories noted in Zimbabwe between STD and HIV during the 1990s: STD declined by 25% during that decade while HIV prevalence increased from 9% to 25%, implying a stunning 12% annual HIV increase.¹³ Why would sets of sexually transmitted infections behave so differently during the same period in the same place unless modes of transmission other than simply sexual modes were involved? And why would a relatively low efficiency (in its sexual form of transmission) virus like HIV outrun the much more efficiently transmitted garden variety STD? Such data and considerations should have raised a red flag stimulating further epidemiologic investigation.

In addition, high rates of HIV infection were observed in very low-risk people: pregnant and post-partum women, especially women reporting only one lifetime sexual partner, with that partner having tested negative for HIV. Other low-risk people were the 10% or so of HIV-positive pre-pubertal children whose mothers had tested HIV-negative. Lastly, several studies had reported high levels of HIV infection associated with exposures to medical injections. For example: among factory workers in Rwanda in 1985, HIV prevalence in workers reporting a history of STD, but who had not received medical injections, was 9.7%, while those who had received medical injections had the significantly higher prevalence of 27%.²³ Altogether, such observations undermined the assertion that sex was responsible for virtually all HIV infections in African adults. (Officially, only a paltry 2% of infections were thought to be a consequence of non-sexual exposures by HIV-contaminated sharps.)

Defending the decided-without-the-facts consensus view

I was stunned by officialdom's response to this 2001–2002 set of publications critiquing the consensus view: in brief, they dug in their heels. Hard. Nor could I have begun to imagine that this initial response was to be their permanent response in the future, despite a cascade of subsequent publications that provided additional evidence to support this initial critique. The rational response would have been to admit that non-sexual exposures had been under-suspected for too long and had been scientifically under-explored. Programmatically, at the very least, there should have been a call to re-evaluate the evidence that led to the consensus in the first place, to assess

its quality and reliability, and to field empiric studies to resolve dissonances—principally, to tease out the proportional contributions of sexual and non-sexual exposures to local HIV burdens.²⁴ This meant clearly accepting the fundamental fact that HIV was not a sexually transmitted, but a sexually transmissible, infection; there were far more effective ways other than sex to transmit it, and these ways should be properly investigated.

But this is not what happened. The mainstream view that sex was driving the HIV epidemics in sub-Saharan Africa was an assertion in dire need of high quality supportive evidence. Instead, as we shall see later, defenders principally relied on inferential reasoning and evidence of modest quality²⁵ to dismiss our arguments.²⁶ Reliance on such evidence to describe reality would have been tolerable had no anomalies or dissonances been observed. It is the breadth and depth of these dissonances that indicated the pressing need for a more rigorous look at HIV transmission dynamics in Africa. Our arguments were not only dismissed but, more importantly, ignored at the highest levels of the health agencies responsible for getting the picture right: CDC, WHO, and, especially, UNAIDS. Sadly, this silence at the highest levels continues to this day, despite more than a decade's worth of respectable evidence challenging the received wisdom.

Impact of the second wave of papers in the International Journal of STD & AIDS

Five months after publication of our first full-length article on the topic¹⁴ both furor and fury continued, but with greater intensity. What amplified the furor and fury was the simultaneous publication of four articles by our

informal group in the March 2003 issue of the *International Journal of STD & AIDS*.¹⁷⁻²⁰ The Royal Society of Medicine orchestrated a press release on 20 February 2003 that received considerably more attention than the one four months prior, which had been based on a single article.¹⁴ It is as if—to borrow a metaphor from the vaccination domain—the October press release primed the public, and the February one fully inoculated (not to be confused with “immunized”) it.

Because Dr. Gisselquist’s analyses tended to be exhaustively detailed and dryly reported, I designed and drafted the first article¹⁷ of the series, focusing on presenting our arguments and evidence as simply and as straightforwardly as possible for both lay and professional readers. It summarized the dozen or so anomalies and dissonances, observing that the mounting toll of HIV infection in Africa echoed the mounting number of puzzling findings; in short, too many stubborn facts did not fit the official interpretation. The second article¹⁸ presented, in ponderous detail, evidence that had been available in the refereed literature prior to promulgation of the consensus of 1988 by the WHO and the CDC. This evidence had either not been considered or had been inadequately interpreted. Not only did forging this official consensus represent a premature closing of debate about “Why Africa?” but it served to discourage further inquiry, not to mention dissent. By the time of the Fifth International Conference on AIDS in Montreal in mid-1989, for example, conversations and presentations about global HIV epidemiology failed to include consideration of non-sexual (blood-borne) HIV transmission in poor countries. That the consensus emerged despite, rather than from, the available evidence did not speak highly for the scientific trustworthiness of the public health bureaucracies charged with the serious

task of getting the epidemiologic picture right. A good part of the reason for this official failure is the well-known tendency of experts to ignore evidence they do not want to see especially, I suspect, if such evidence could jeopardize funding streams. Both considerations can discourage implementation of properly controlled and conducted field studies, for fear that results might not only undermine the experts' cherished views but also their livelihood.

By detailing the evidence that had appeared in the literature prior to the official forging of the consensus, which averred that the vast majority of HIV cases in sub-Saharan Africa were the result of unprotected sex, our informal group managed to trigger the fury of many at the WHO, the CDC, and UNAIDS. I speculate that offended members of these health organizations viewed our analysis as an unkind assessment of their scientific competence or, perhaps, of their political-ideological motives (more on that later). I was very surprised at their reaction because I naively thought that they would be considerate of our suggestion, based on very good evidence, that the problem of anomalies and dissonances could be solved by comprehensively evaluating the contribution of non-sexual modes of transmission to HIV burdens in poor countries. I could not have been more wrong. They reacted with shock, dismay, and anger. And, as we will soon see, their response mainly consisted of rationalizing away the dissonances within the framework of the orthodoxy. In brief, they used their belief framework as a substitute for better evidence.

Just what did this pre-1988 consensus evidence suggest? It could actually best be used to support the interpretation that health care exposures in Africa

caused more HIV infections than sexual transmission! This was a stunning realization. Note that I did not say “revelation”, for this would require data from really well-designed studies. After all, extraordinary claims require extraordinary evidence. In any event, the admittedly crude measures of risk factors obtained from published study samples of the general population were shown to associate more than half of HIV infections in adults with puncturing exposures in health care settings.¹⁸ But that wasn’t all. Gisselquist also used these data to construct a mathematical model designed to estimate the proportion of HIV in Africa that was caused by sex;¹⁹ the model indicated that only about 25%–29% of HIV incidence in African women, and about 30%–35% in men, could be attributed to sexual exposures. True, the substandard quality of the data emanating from Africa and the generally poorly designed research studies prevented us from making definitive statements. As we concluded,¹⁹ “We cannot and do not intend our estimates to be the last word, but rather a step toward better evidence-based estimates...”

Our last article in this controversial issue of the *International Journal of STD & AIDS*²⁰ critiqued the claim that improved management of sexually transmitted diseases in Mwanza, Tanzania during the early 1990s had subsequently caused a nearly 40 percent decline in new HIV infections. This result was suspicious because, while the incidence of HIV infection had dramatically (minus 38%) declined, the garden-variety STD burden had not. Moreover, the researchers had not controlled for the contemporaneous implementation of sterile health care protocols and training in Mwanza clinics by a different public health team.²⁷ Our article called for re-analysis of

the Mwanza trial data in light of these considerations. This was not done or, if done, it was not reported.

Analyses and reflections in these four simultaneously published papers should have injected enough doubt in the minds of experts that research agendas should have been re-conceptualized and modified accordingly. This was not to be. Shockingly, refusal to properly investigate non-sexual HIV transmission modes remains true as of the date of this writing (Spring 2015): reported studies controlling for these kinds of confounding factors are exceedingly rare. And I certainly did not endear myself to the CDC, WHO, and UNAIDS experts when I told them, behind closed doors on 14 March 2003, that HIV epidemiology in sub-Saharan Africa consisted of “First World researchers doing second rate science in the Third World countries.” But it was and remains true. When all is said and done, what kind of science is it that ignores a potentially major mode of transmission by obstinately refusing to properly measure it?

“I and my public understand each other very well; it doesn’t hear what I say, and I don’t say what it wants to hear.”

—Karl Kraus

From here to enmity: the March 14, 2003 meeting in Geneva

The international health agencies’ formal response to our controversial papers was to convene a meeting of experts at WHO headquarters in Geneva shortly after these articles’ appearance in print. The idea, as stated in the

memorandum to participants, was to “bring together the leading epidemiological and modeling experts with Gisselquist and Potterat” and to “prepare the ground for a strong well founded statement from WHO and UNAIDS on the role of unsafe injections in HIV transmission. This statement will have to be published in a leading scientific journal, and also has to be disseminated widely in the press. There will not be a report from the meeting itself.”²⁸ Regrettably, this meeting was neither tape- nor video-recorded. There were approximately thirty participants, including at least five from WHO, two each from UNAIDS and CDC, five from European schools of tropical medicine, one each from the National Institute of Allergy and Infectious Diseases, the US Agency for International Development, and Immunization, and three of us (Drs. Stuart Brody, David Gisselquist, and me).

It turns out that this “consultation” was to set the tone for all subsequent discourse about what was driving HIV transmission in poor countries. First, no one from the highest echelons of the three international health agencies was present at this consultation, foreshadowing their future and continuing absence from public commentary on this discussion, in either the media or in the scientific literature, other than the occasional *pro forma* denial that anything but sex could be responsible for HIV epidemics in Africa. Second, their official response was highly choreographed, bureaucratic (read: science by committee), and dismissive of the evidence and arguments presented. The orthodoxy was staunchly reaffirmed and continued to be buttressed by weak empiric evidence—although the conclusion was capped by a (soon to prove hollow) promise to obtain “improved data”²⁶ to strengthen confidence in the mainstream view. Third, the free-floating anger that was part of the

background noise at the meeting, and which several times flared into ill-tempered remarks, was to remain part of the subsequent discourse.

The press release summarizing the proceedings not only misrepresented the sense and outcome of the consultation but it was finalized before the end of the deliberations. It therefore became clear to us that the international agencies' minds were made up before the meeting. What was reported was that although "no consensus emerged from the conference", the "prevailing view was that sexual transmission was responsible for the large majority of HIV infections in sub-Saharan Africa; Gisselquist and colleagues demurred (Emphasis mine)." This was "prevailing view" by plebiscite: there were many more persons present at that meeting supporting the orthodoxy than those questioning it or willing to voice skepticism. What the press release should have emphasized is that the studies cited during the discussions by participants committed to the orthodox view at best provided only weak support for that view. Indeed, lack of evidence supporting the orthodox view was why the press release concluded with a call for better data to more definitively determine the role of puncturing exposures in HIV transmission.

And yet "better data" were already available but conveniently ignored. One of the experts at this March 2003 meeting presented unpublished data from Uganda showing incident (new cases) HIV to be more common among people reporting medical injections than among those who didn't. These data were eventually published, but 4 years later.²⁹ Moreover, the CDC had recently reviewed a UNAIDS-commissioned research study by one of its own which showed that there was a strong "association between HIV infection and health care injections. Incidence studies, all conducted in sub-

Saharan Africa, indicated that contaminated injections may cause between 12% and 33% of new infections in the region.”³⁰ The paper, reviewed in Atlanta in the early autumn of 2002, was not cleared for publication by the international health agencies (CDC, WHO, and UNAIDS).

For me, the singular memory I retain from this long ago meeting was the comment made to us by a ranking and seasoned representative of an internationally respected health agency that “maybe you’re right [about puncturing exposures] but don’t tell the African people”. I was stunned. This was spontaneously (?) uttered, presumably because this kind of information could jeopardize public health initiatives such as vaccination campaigns—as if Africans were not smart enough to pay attention to two different risks at once. That person also said “I cannot wrap my mind around this”. I now certainly knew who wasn’t smart enough to understand, and it wasn’t African adults.

A distillation of these proceedings, which omitted the evidence presented at the meeting that was unsupportive of the orthodoxy, appeared in the high-impact medical journal, *The Lancet*, one year later.²⁶ This influential article, spearheaded by the WHO, concluded that “there is no compelling evidence that unsafe injections are a predominant mode of HIV-1 transmission in sub-Saharan Africa”. Crucially, it failed to address the key anomalies that indicated that sex was not the main driver of the epidemics in Africa. Yet its anemic rebuttal of our analyses effectively closed the door to more rigorous inspection of transmission dynamics in Africa. Indeed, it continues to be the article customarily cited in support of the orthodoxy and invoked to dismiss dissenting views such as ours.

Here is not the place to detail the arguments and evidence the international agencies presented to rebut us, but to emphasize that they focused narrowly on medical injections in formal health care settings, rather than also considering other forms of puncturing exposures, such as phlebotomy (blood testing) and exposures in informal health care and ritualistic (for example, tattooing) settings. Moreover, they ignored their own research when it supported our position.²⁹⁻³⁰ As for manner, they relied principally on ecologic evidence, which is the most distant from the actual transmission events and settings,³¹ on risk factor evidence derived from inadequate (read: uncontrolled for puncturing exposures and proper measures of anal intercourse) studies; on inferential reasoning; on speculation; and what they consider to be logical rather than relying on quality data.

Their counter arguments also relied on wholesale rejection of the evidence we presented, claiming that many findings were not true because patients had lied about their sex lives; because mistakes had been made in HIV testing procedures; and because the data were too old and, by insinuation, untrustworthy. In brief: they selectively denied the validity of the evidence they did not like. When in doubt, blame the data. In my opinion, they chose not to believe the evidence because they did not want to. In any event, their position reminded me of the famous line uttered by the professor in Alfred Hitchcock's movie *The Lady Vanishes*: "Nonsense, my theory is perfectly correct. It is the facts that are misleading." And that, in a nutshell, summarizes the official countering of our analyses.

Exiting the consultation meeting at WHO headquarters late that Friday afternoon, Dr. Brody and I entered the elevator. By (what we thought might be) good luck, its occupant was the WHO Secretary General, accompanied by an armed bodyguard. Noticing our meeting identification badges, she mentioned that she had been informed that a consensus had been reached at this consultation. Dr. Brody assured her that not only was this not the case, but that the “consensus” statement had been decided, and even disseminated, before the meeting was over. At that point, the elevator reached the bottom floor and we all exited with polite “good evenings”. Two days later, after our return home, Dr. Brody, Dr. Gisselquist and I emailed the Secretary General, reminding her of our recent encounter in the elevator, and reiterating that the WHO press release “misrepresented both the sense and the outcome of the consultation”; we requested that the inaccurate press release be rescinded and offered our assistance “in the drafting of an accurate consensus statement”. She never replied.

The U.S. Congress reacts

Our papers’ conclusions also reached the ears of Congress. Under the leadership of Senator Jeff Sessions (R-Alabama), the US Senate Committee on Health, Education, Labor, and Pensions held hearings shortly after publication of our four articles, to determine whether HIV/AIDS funds should be earmarked for programs targeting unsafe puncturing exposures. (Sessions’s staff had contacted Dr. Gisselquist and me almost immediately, on 10 March 2003.) Senator Sessions, via the Department of Health and Human Services, also commissioned RTI International, an independent and non-profit research institution, to assess our claims that contaminated

needles might well be driving the HIV epidemics in sub-Saharan Africa. The RTI report,³² released in early January 2004, concluded that “Arguments used to inform the debate about the etiologic significance of unsafe medical injections for HIV infection in sub-Saharan Africa or the Caribbean are based on meager evidence at best.” (Ironically, the same criticism could much more fairly be applied to the assertion that heterosexual sex was driving the epidemics!) Importantly, the report noted the inadequacies in the published literature, principally the lack of quality and relevant studies to confidently settle the issue. The report (properly) recommended that unpublished data that could shed light on the issue should be solicited and published, and that new, well-designed studies be implemented as indicated. Neither recommendation, to my knowledge, was ever followed. The report also concluded that, until shown to be misdirected, HIV control efforts in Africa should stay the course. This was, to me, the equivalent of temporarily settling the issue using an epidemiologic penalty shoot-out. In any event, at least Senator Sessions was able through two hearings to generate support to target \$300 million out of the President’s Emergency Program for AIDS Relief (PEPFAR), primarily in Africa, to promote safe injections and safe blood transfusions.

“I believe in getting into hot water. I think it keeps you clean.”

—G. K. Chesterton

Explaining the resistance to our critique and reflections

Skepticism disturbs orthodoxy. Predictably, doubting often produces defensive reactions from believers, ranging from irritation to rage.³³

Defensiveness on the part of the international health agencies and mainstream HIV researchers was strong, far stronger than any of us had anticipated. It was closer to the rage, than irritation, end of the spectrum. Nor had we anticipated that part of their reaction would include (unflattering) *ad hominem* comments. Among other, less printable, things I was called “Africa’s Newest Plague”; “Core Stigmatizer”; “Linus Pauling—in his later years” (when Pauling was thought to be advancing crackpot ideas); and [a reward being offered] “for his head on a platter”. There may come a time in one’s professional life when one loses respect for colleagues once admired. These adrenal cortex-derived epithets were my baptism under fire, because each of them emanated from esteemed colleagues in these agencies; they were uncalled for, considering that the only thing I stigmatized was shoddy science, that predictable impostor of truth.

By their own admission, the international agencies feared that our work would cause Africans to lose trust in modern health care, especially childhood immunizations, as well as undermine safer sex initiatives. (Recall that their condom campaigns were also aimed at curtailing rapid population growth in sub-Saharan Africa.) We speculate that disbelief on the part of HIV researchers that medical care in Africa could be harming patients may have been a significant factor in their defensive posture. We were also impugning the quality of their scientific research and potentially threatening their livelihoods. In addition, our analyses also directly threatened the politically correct view that AIDS was not just a disease of gay men and injecting drug users, but also of heterosexuals. Lastly, our data were undermining the time-honored belief about African promiscuity, a notion

that may well have initially contributed to the (pre)conception that AIDS was thriving in Africa because of it.

“God was satisfied with his own work, and that is fatal.”

—Samuel Butler

The aftermath

The international health agencies and mainstream HIV-in-Africa researchers essentially stonewalled our views by denigrating, dismissing, or ignoring the evidence we were presenting—all the while asserting, *ex cathedra*, that their catechism was right. The few who (semi-)publicly reserved judgment until better evidence arrived soon became silent, not only because better evidence in the form of well-designed studies had not been forthcoming but because, gauging the ferocity of the resistance, they probably succumbed to peer pressure and chose to remain on the sidelines, whatever their private views might have been.

Two attributes color the study of HIV transmission in Africa: lack of tolerance for dissent and, above all, virtual absence of publicly expressed skepticism for the orthodoxy in the halls of mainstream public health organizations and of academia. Received wisdom, uncritically accepted, usually ossifies into dogma. This is precisely what happened. Regrettably, lack of tolerance soon reached the editorial offices of the major medical and public health journals. We speculate that the international health agencies and mainstream reviewers persuaded the editorial staffs at these high-impact journals not to publish our papers, probably by asserting that our views

would be detrimental to HIV interventions in Africa and would contribute to increases in HIV transmissions and mortality. In this regard it was truly unfortunate that AIDS deniers like the Duesbergians³⁴ had previously given dissent a bad name. Whereas Duesbergians denied that HIV caused AIDS, we knew that the evidence for HIV causing AIDS was incontrovertible and said so. Yet somehow we seemingly were lumped with the Duesbergians and other deniers.³⁴ As I specifically pointed out elsewhere, there is a world of difference between dissenters, which we are, and deniers, which we are not.³⁵

Post Geneva meeting publications: manner

During the ten years following the Geneva “consultation” we publicly³⁶ encouraged both debate of available evidence and implementation of well-designed studies, and this was a frequent part of the concluding remarks of our publications. Our informal group, whose membership fluctuated during this period, eventually published dozens of full-length articles, commentaries, and letters to the editor. After a series of rejections from top journals, we lowered our expectations and generally submitted manuscripts to lower-impact journals. Of the 55 original articles (and 6 commentaries) eventually published, 34 (56%) appeared in the *International Journal of STD & AIDS*. (This apparent favoritism was probably the major reason for sacking the editor-in-chief, Dr. Wallace Dinsmore—a founding editor of the journal—in early 2010 after 20 years at the helm.) We were also inveterate scientific correspondents, sending 82 letters about AIDS in Africa to editors of medical and public health journals, 55 (67%) of which were published (See Appendix 2). Of the unpublished letters, almost all were rejected by high-impact American journals such as *Science* and the *Journal of the American*

Medical Association. It is a tribute to editors of several British medical journals that they were much more willing to publish our articles, editorials, and letters than journals elsewhere. Indeed it is astonishing that virtually no scholarly debate on the controversy surrounding HIV transmissions in Africa took place within the pages of American medical or public health journals.

Post Geneva meeting publications: matter

Our informal group's post Geneva meeting publications focused on 1) providing more evidence suggestive of the importance of non-sexual HIV transmission in sub-Saharan Africa (full-length articles) and 2) critiquing newly published studies, pointing out errors of commission and omission, and suggesting ways to achieve adequate data analysis and/or offering more logic- and evidence-driven interpretations of their data (letters to the editor) and 3) providing commentary (editorials).

For example, one of the objections by the international agencies to our suggestions that non-sexual (puncturing) exposures might be driving the HIV epidemics was the apparent absence of HIV in children ages 5 to 12. They argued that were such exposures common, children would be as much at risk as adults. Actually this was a largely data-free inference. Children were unlikely to be medically attended at (say) prenatal or STD clinics, where HIV prevalence was known to be very high; hence their exposure risk would be different from that of adults attending such clinics. Secondly, surveys of HIV prevalence in pediatric populations had rarely been conducted. In several articles we discussed the implications of a then recent national probability sample of the South African population showing an astonishing HIV

positivity rate of 5.6% for 5–12 year olds.³⁷ Their reply was that the testing had been faulty, again blaming the data. In a series of later publications, we showed that this result was not an anomaly.³⁸⁻⁴⁴ We summarized dozens of prior studies of pediatric HIV infection in clinical settings; these reported young children becoming infected from sources other than their (uninfected) mothers.³⁹ We published analyses of alarmingly high rates of HIV in young children in Mozambique,⁴³ Swaziland,⁴¹ Kenya,⁴¹ and Uganda⁴⁴ based (mostly) on independently conducted nationally representative surveys in these countries. In light of such findings, the proper response would have been to conduct surveys in several different countries with high and low HIV prevalence, making sure that testing protocols and specimen processing were scientifically unimpeachable—using cases and controls, contact tracing, comprehensive assessment of risks, and sequencing (“fingerprinting”) of HIV. None of this was done.

Another objection on the part of the international health agencies was that we had grossly overestimated the efficiency of HIV transmission under various skin puncturing circumstances. This putative “overestimate” had allowed them to reject our claims out of hand. We therefore conducted an extensive review of available data in the medical literature⁴⁵ concluding that their assessment was probably wrong by an order of magnitude. Yet, aware that our data were incomplete, we admitted that our conclusion was in need of improved data via new studies. To my knowledge, such studies have not been fielded.

A particularly damaging article appeared in the prestigious journal *Nature* in the form of a brief communication in 2003. It concluded that, because HIV

and Hepatitis-C prevalence patterns in sub-Saharan Africa do not often coincide, puncturing exposures were “not the dominant contributor to the African epidemic”.⁴⁶ Actually, this ecologic inference was faulty, as we detailed in a rebuttal shortly afterward, but in a different venue.⁴⁷ (Nature declined to publish our rebuttal.) The principal weakness of their argument was the assumption that these two different blood-borne viruses were transmitted the same way. In fact, Hepatitis-C was known to be efficiently transmitted intravenously (into the veins) but not intramuscularly (usual route for medical injections)—therefore, a different epidemic pattern could be expected. In addition, the prevalence data they used to plot Hepatitis-C patterns were based on old and unrepresentative surveys, not to mention that tests for Hepatitis-C were often insensitive to detecting African strains, as a recently (2015) reported study confirmed.⁴⁸ In the intervening decade, however, no studies had been fielded to resolve these uncertainties.

We also published articles indicating that anal intercourse in heterosexual African populations was likely to be more common than currently believed, and we suggested ways to obtain valid research results.⁴⁹⁻⁵² Regrettably, this did not result in modification of prevention messages to specifically address the dangers of anal intercourse. In addition, we published results of analyses showing that HIV infection was much more common in circumcised than in uncircumcised virgins of either sex and in adolescents in Kenya, Lesotho, and South Africa, implicating non-sexual transmission.⁵³ Moreover, our collaborators published several analyses highly suggestive of HIV transmission in medical settings⁵⁴⁻⁵⁷ and what could be done to attenuate such risks.⁵⁸ In this regard, we proposed that several policies could be immediately and inexpensively implemented: 1) broad public education about

non-sexual risks; 2) transparent (patient-observed) sterile medical care procedures; and 3) zero tolerance for health care associated infections.⁵⁸ We recommended implementation of quality control in health care settings and, crucially, investigation of unexplained HIV infections; both of these aspects of health care delivery were routinely conducted in rich countries and should be supported in poor countries as a priority.⁵⁹

Only when people have accurate knowledge of HIV modes of transmission can they make good decisions to protect themselves and their families from inadvertent infection. Hence the importance of public education. Dr. Devon Brewer tested this view and attempted to measure its impact by examining Demographic and Health Survey data from 16 sub-Saharan African countries.⁶⁰ In a cleverly thought out analysis, he concentrated on examining data that could illuminate his question: is knowledge of blood-borne (not sexual, which everybody in Africa knows about) HIV risk associated with a country's HIV prevalence? It turned out that countries in which many people were aware of blood-borne risk indeed had lower HIV prevalence than countries in which few people were aware of such risk. This observation suggests that explicit public education campaigns about blood-borne (read: puncturing exposures) HIV risk may be very important in protecting the population from such exposures.

The narrow focus on sexual transmission of HIV can lead to deeply harmful and unwarranted stigma, such as for an infected woman whose sole lifetime sexual partner is her HIV-negative husband. We specifically addressed this danger in a commentary.⁶¹ It is the default assumption of sexual transmission that puts such infected women in the position of being unfairly accused of

infidelity or promiscuity. Silence about non-sexual exposures destroys reputations and lives, with women in traditional African societies being particularly vulnerable to abuse and abandonment.⁶¹

Countering UNAIDS/WHO/CDC inertia and inaction

By mid-decade, it was clear that the dozens of publications presenting evidence undermining the official view that heterosexual transmission was driving sub-Saharan Africa's epidemics had failed to have the intended impact of stimulating scientifically sound research. As we had previously pointed out, what was needed was evidence that one could have confidence in. Because we were unaware of any research being done to do the required studies and because we despaired that these would ever be done in our lifetime, Dr. Brewer suggested we do our own. This was a bold suggestion, considering that we not only lived a long way from Africa, but also had no source of funding. We also knew that grant proposal reviewers, being affiliated with the international health agencies or with the preponderantly scientifically conservative academia, would be unlikely to approve our proposals should we apply. What is viewed as maverick science has a predictably tough time being funded.

Luckily, some members of our informal group had contacts on the ground in Africa. For example, Dr. Gisselquist's other field of expertise, agricultural policy, frequently engaged him as a consultant in both South Asia and sub-Saharan Africa, while Dr. Brewer's interest in assessing blood exposures in non-formal health settings (viz., traditional medical practitioners) led him to contact the author of an article on such risks in *Tropical Doctor*⁶²—a

Nigerian physician practicing in a teaching hospital in Calabar, located in the southeastern coastal region of the country. Dr. Gisselquist's connection, an enterprising part-time journalist from Kenya named Moses Okinyi and Dr. Brewer's connection, Dr. Etete Peters, were both willing to participate in our efforts to implement two empiric studies, one involving HIV-infected children in Kenya and the other, involving newly infected adults in Nigeria.

2007: A turning point year—or so we hoped.

After at least five years of providing evidence that the mainstream view ought to be reconsidered, a recommendation that fell on unwilling if not deaf ears, Dr. Gisselquist decided to publish a book summarizing our findings and make it freely available on the internet,⁵⁹ bearing the felicitous double-entendre title of "Points to Consider...". Contemporaneously, Dr. Brewer and I decided to field 2 studies on the ground in Africa, despite lack of outside funding. It was hoped that Gisselquist's book would attract the attention of thoughtful persons outside the entrenched AIDS industry and thus maybe bring pressure on the AIDS-in-Africa establishment to do what was scientifically correct. This apparently did not happen.

The first field study was carried out in Calabar, Nigeria between August 2007 and February 2008, under the aegis of Dr. Etete Peters and his medical students. Because we had long thought about proper study design to assess the contribution of sexual and non-sexual exposures, we took the lead in proposing a survey instrument that would prospectively and comprehensively assess both sexual and non-sexual (puncturing) exposures in newly-infected patients in Dr. Peters's clinics. This survey instrument's architect was Dr.

Brewer who collegially solicited suggestions from his American and African collaborators. Dr. Brewer was especially interested in controlling for “reverse causation” because this had been one of the main objections by mainstream researchers to our evidence that medical procedures were driving HIV epidemics in Africa. “Reverse causation” refers to misinterpreting the sometimes strong association between, say, medical injections and HIV prevalence by concluding that HIV-infected patients had become infected while attending a clinic for medical care when, in point of fact, it could be that these HIV-infected patients had been previously infected (by sex, naturally!) and were attending a clinic because they were now sick with symptoms of advanced HIV infection. Hence an important aim of the study was “to assess the relationship between a broad array of blood exposures, especially those not received in response to HIV-related symptoms or complications, and incident HIV infection in sub-Saharan Africa”.⁶³

The study’s participants were recruited from the voluntary HIV testing clinics at the University of Calabar’s Teaching Hospital. About three-quarters of clients approached were successfully enrolled; remarkably, most declined the approximately \$5 compensation offered to cover transportation costs. During the six-month study interval, 321 clients participated. Because our interest was HIV incidence, analyses focused on serial testers: with them, one could observe the change from last negative test to first positive test. Forty-five clients were serial testers, of whom 10 became HIV infected during the study period, for a (high) 10% annual incidence rate. In brief, although we found that many types of blood exposures were commonly reported, persons who became HIV-infected were more likely to report both specific and aggregate blood exposures during the study period than persons who

remained uninfected. Crucially, newly HIV-infected clients reported blood exposures that could not be explained to be a consequence of seeking medical care for symptoms of HIV, thereby undermining mainstream researchers' conviction that reverse causation accounted for the association between medical care and HIV prevalence. Nor could newly-acquired HIV infection be accounted for by unprotected sexual exposures: no sexual variable was associated with newly-acquired HIV infection. HIV incident cases were associated with use of someone else's razor, surgery, blood transfusions, enemas, vaccinations, and infusions. The principal shortcoming of this study was the small number of serial testers and of incident cases (ten). Yet this pilot study supported our contentions and should have provoked the conducting of larger studies to confirm our findings.⁶³ Regrettably, this did not happen.

The second field study was also initiated in 2007 and lasted one year, starting in March.⁴¹ Like the first, it was a case-control study, but took place in Kenya, and focused on HIV-infected children rather than adults. These children had HIV-negative mothers and, therefore, were not likely to have acquired infection at birth or while their mother breast-fed them. As in the Nigerian case-control study, these HIV-infected patients, whose controls were their own HIV-negative siblings, were shown to have had more kinds of blood exposures than their uninfected siblings. In particular, punctures related to health care for malaria (blood testing, injections, infusions), and dental surgery by informal providers, were more commonly reported in HIV-infected children, confirming previous findings in a South African pediatric survey³⁷—the same one that the WHO had denigrated by blaming the data's quality. We also combined these Kenyan findings with those of a

representative national health survey in Swaziland conducted between 2006 and 2007 that included 1665 children aged 2 to 12. Fifty Swazi children (3%) in the sample were HIV-positive, with 11 (22%) of the 50 having HIV-negative mothers.⁴¹ From this observation, we inferred that 1808 children aged 2 to 12 in Swaziland had HIV and HIV-negative mothers, implying infection by some non-sexual means, possibly contaminated sharps (because infection via the sexual route, including sexual abuse, was deemed very unlikely). Although the Swazi press rapidly disseminated our findings⁶⁴ and although this publicity prompted a public call for investigation by a Swazi Crown Prince, interest quickly fizzled and no follow-up was, to our knowledge, ever done by Swazi authorities. And, unsurprisingly, no follow-up studies or investigations were done by mainstream researchers or by international health agencies to challenge or confirm these findings, or to protect other children via finding the source of these unexplained infections. And it's not as if these would have been expensive studies to implement: I personally funded the two studies^{41,63} for less than 10,000 US dollars. All things considered, 2007 was not a turning point year.

Sigh.

Other voices

Between 2006 and 2014, several studies appeared in the literature whose empiric results were supportive of our view that HIV transmission dynamics in sub-Saharan Africa seriously needed revisiting. Among them, one of the most persuasive was done by the CDC's own Dr. Janet St. Lawrence and her colleagues,⁶⁵ who had historical (1989–2001) risk factor data on more than

3000 pregnant women at University Teaching Hospital in Lusaka, Zambia. These women were about 24 years old, married (90%) and monogamous (90% with no other partner in the previous three years), yet an astonishing thirty percent were HIV-infected. Not only were injections, both intramuscular and intravenous, overwhelmingly associated with HIV infection in these women—far exceeding the contribution of sexual behaviors—but HIV infection could clearly not be blamed on reverse causation. Only 4 of the 11 sexual behaviors measured were associated with HIV infection, yet, counter-intuitively, they were inversely associated, implying that sexual behaviors were protective of HIV infection! Clearly such findings should have triggered flashing red lights. On the contrary, this retrospective study so upset the CDC that Dr. St Lawrence was asked not to publish it. She refused her superiors' request. Turned down by several high-impact journals, the manuscript was eventually submitted, at my recommendation, to the International Journal of STD & AIDS. Once peer-reviewed and accepted for publication, the CDC pressured its editor-in-chief, Dr. Dinsmore, and publisher (the Royal Society of Medicine) to withdraw acceptance. Dr. Dinsmore's courageous decision to publish it anyway saved this important analysis from disappearing into medical oblivion, as happened with a previous CDC study mentioned earlier.³⁰ Dr. St. Lawrence was due to retire from the CDC shortly afterward and she (understandably) opted to do so.

Another investigator whose analyses made it foolish to ignore or dismiss findings suggestive of a substantial role for puncturing exposures in sub-Saharan Africa's HIV epidemics was Savanna Reid. She and her colleague van Niekerk used information from official South African surveys to

conclude that omission of non-sexual transmission from consensus HIV epidemiology in Africa was not only a serious oversight, but had profound ethical and operational implications for interventions.⁶⁶ Among other findings, they pointed out that more than a quarter of recent HIV infections detected in the 2005 South African national survey occurred in adults who denied sex during the previous twelve months; they presented evidence showing that only a very high rate of misreporting could produce this stunning observation—this particular comment aimed at readers inclined to dismiss this finding by saying that participants had lied about their sex lives. They also reported on recent surveys that indicated widespread lapses in infection control in health and dental care facilities,⁶⁷ subverting the commonly held view that medical care in South Africa was of high quality and safe.²⁶

Moreover, Reid undermined the assertion by mainstream researchers that if injections were driving the HIV epidemics, one would see many more HIV-infected kids aged 2 to 12 years.⁴⁰ She challenged it with a meta-analysis of published literature and with mathematical modeling to indicate that HIV prevalence in children would not likely increase with age, as mainstream researchers had insisted. Here, then, was yet another petard under a mainstream assumption that had not, in the first place, been supported by data, only by inference and speculation. In addition, Reid and another colleague (Juma) independently estimated the risk of HIV transmission during blood exposures taking both viral and host characteristics into account, concluding that three separate avenues of evidence (infective dose, viral load, and injection volume) support Gisselquist's estimates that HIV transmission efficiency is likely to be considerably higher than that believed

by mainstream HIV researchers.⁶⁸ Finally, Reid pointed out a serious error in the WHO estimates⁶⁹ that contaminated injections accounted for only 2.5% of HIV infections in sub-Saharan Africa. The error consisted of the WHO model having used the general population's rate of HIV prevalence rather than the more relevant HIV prevalence in clinics to calculate frequency of exposure to contaminated injections; this would be especially important in clinics seeing patients experiencing advanced disease, which is known to be more infectious than latent infection. Reid's model adjustments raised the estimated contribution by medical injections from 2.5% to somewhere between 12% and 47%, a considerable difference.⁶⁹

By mid-decade, my long-time colleague Stuart Brody had been contacted by a German graduate student, Eva Deuchert, to help assess available data on health care in Africa that made little sense to her. In several papers, she and Brody provided additional evidence suggestive of an important role for health care related HIV transmission.⁵⁴⁻⁵⁷ The first paper showed that Kenyan women who received tetanus shots during pregnancy were about twice as likely to have HIV infection compared to women who had not received this vaccination.⁵⁴ Importantly, these findings were not confounded by reverse causality; not only were tetanus shots prophylactic rather than given for treatment for disease symptoms, but none of the infected women was even aware that she had HIV infection. The second paper showed that specific health care indicators (for example, failure to use disposable syringes designed to only be used once, and broader implementation of tetanus vaccination) were strongly associated with HIV prevalence in African countries with available data.⁵⁷ The third, and highly technical, paper⁵⁵ showed that mathematical models generally used to simulate heterosexual

HIV epidemics suffer from use of model parameters that are distant from evidence on the ground. Hence simulations have not accurately portrayed the epidemics in sub-Saharan Africa. The authors detail how many models have used grossly inflated per-contact HIV transmission probabilities, grossly overestimate numbers of sexual partners African adults have, and grossly overestimate frequency of their sexual activity.

Starting mid-decade, several other investigators outside our informal group began reporting findings that undermined the mainstream orthodoxy that sex was driving HIV epidemics in Africa. First, a little detour. In the early days, it was widely accepted in both policy and scholarly circles that sexual promiscuity explained “Why Africa?” When population-based surveys, conducted during the late 1980s/early 1990s, failed to support the idea of African hyper-sexuality,¹⁴ researchers speculated that sexual concurrency (overlapping partners instead of serial partnering) accounted for the extraordinarily rapid rates of HIV infection, particularly in eastern and southern Africa. Although the renowned Four-Cities study²² at the beginning of the new millennium, and our own observations a year later,¹¹ provided persuasive evidence that sexual concurrency was not likely to explain “Why Africa?”, defenders of this notion were undaunted and shrilly continued to advocate its importance. Helen Epstein, for example, wrote a widely publicized book half a decade later claiming that discouraging long term concurrency could be Africa’s “invisible cure”.⁷⁰ As I stated elsewhere, this was clearly a case of “invisible evidence”.⁷¹ Two years later, Lurie and Rosenthal published a landmark analysis⁷² showing that there was not any conclusive evidence in Africa that concurrency was associated with HIV prevalence or with increases in the size of the HIV epidemics or with

increases in the speed of HIV transmission or with HIV persistence in populations. Shortly thereafter, Sawers and Stillwaggon meticulously examined the concurrency mathematical model—warts and all—and its unrealistic (their word) assumptions and came to a similar conclusion: there is no correlation between sexual concurrency and HIV prevalence in Africa.⁷³ The warts? In their own words: “...quantitative evidence cited by proponents of the concurrency hypothesis is unconvincing since they exclude Demographic and Health Surveys and other data showing that concurrency in Africa is low, make broad statements about non-African concurrency based on very few surveys, report data incorrectly, report data from studies that have no information about concurrency as though they supported the hypothesis, report incomparable data and cite unpublished or unavailable studies.”⁷³ This damning scholarly assessment could easily be the template for most of the mainstream HIV epidemiology research in Africa.

Sawers and Stillwaggon, along with Hertz, also provided evidence for HIV transmission-boosting factors that have been neglected by mainstream researchers: “endemic parasitic and infectious diseases... [that] increase the likelihood of HIV infection and alter the dynamics of epidemic spread.”⁷⁴ They present data from many developing countries that diseases such as schistosomiasis, lymphatic filariasis, gonorrhoea, chlamydia and malaria, for example, may well be the transmission-boosting co-factors that could explain “Why Africa?” In any event, these authors state that business-as-usual-sex-behavior-driven epidemiology needs to be given a permanent rest, since it has so consistently and so long failed to solve this puzzle.

Akeke and colleagues⁷⁵ reported on how frequently inmates in a Lesotho prison, 90% of whom are in the high-risk years for HIV, received tattoos while in detention: two-thirds of the nearly half who sported tattoos. They note that tattooing instruments in prisons were seldom sterilized and, half of the time, were used serially on several inmates. Although no HIV results were reported, this African report echoed one from the previous year in Georgia (USA) that noted a strong association between receiving a tattoo in prison and HIV incidence in inmates.⁷⁶ These observations clearly suggested the need for a closer look at tattooing as a probable contributor to HIV transmission in Africa.

Apetrei and colleagues investigated the risk of HIV transmission through unsafe injections.⁷⁷ Not only had this transmission route been asserted by mainstream epidemiologists to be, in the absence of convincing evidence, too inefficient to contribute significantly to Africa's HIV epidemics,²⁶ but two papers published the following year had buttressed that assertion. An investigation in Ethiopia, using suboptimal laboratory techniques, had failed to find HIV in needles that had been used on clinic patients in areas the authors claimed—without providing data—to have high HIV prevalence.⁷⁸ Another study, conducted in Zimbabwe, specifically excluded unsafe injections as a major source of HIV in that country.⁷⁹ Apetrei's group, using rigorous laboratory techniques, demonstrated that HIV was present in 33% of syringes used for intravenous injections and in 2.3% of syringes used for intramuscular injections.⁷⁷ As the authors conclude: "...we provided proof of concept that injection practices could account for a significant proportion of new HIV infections." The different findings from these three African

countries should have prompted the launching of other studies to settle this important issue. This did not happen.

Lastly, two of the most seasoned epidemiologists working in sub-Saharan Africa recently admitted: “We still do not fully understand why the spread of HIV has been (and still is) so different in sub-Saharan Africa compared to heterosexual populations in other parts of the world and why the incidence of HIV infection in young women in southern Africa is so high”.⁸⁰ This stunning confession of ignorance, made in 2012—three decades into the African HIV epidemics—indicates that the original question “Why Africa?” is still very much with us and, therefore, a crucial and urgent challenge. Any bets that comprehensive risk factor assessment, rather than the monochromatic focus on heterosexual sex, might help solve this puzzle?

Official negligence

In a thought-provoking essay, the German social scientist Moritz Hunsmann explored the political incentives of international health agencies and of African governments to dismiss or ignore evidence of non-sexual transmission in Africa’s HIV epidemic.⁸¹ In brief, he proposed that continued sexualization of the epidemic makes it easy to blame individual victims, whose personal behaviors (implied to be immoral) caused their disease. On the other hand, acknowledging an important role for non-sexual (puncturing) exposures in health, dental, cosmetic, and ritualistic settings would be politically threatening, because assuring healthcare and workplace safety would be viewed as the responsibility of governments and public health authorities. Implicit in his view is official negligence: deliberate

underestimation, “if not outright denial”, of non-sexual transmission, which exculpates governments and health agencies. To him, epidemiologists and researchers have been complicit, because they uncritically supported sexualization of Africa’s HIV epidemics; furthermore, this turn of mind had a deleterious effect on rational evaluation of dissonances and anomalies (“conformist analysis inhibits reasonable debate”) which “has done a lot of harm to both science and prevention policies”.⁸¹

In a small series of papers⁸²⁻⁸⁴ Gisselquist and colleagues⁸⁵ detailed errors of commission and omission by the public health establishment, donor agencies, and African governments. He states that public health authorities should be ashamed of themselves for the inadequate science and, therefore, missed prevention opportunities, not to mention the avoidable suffering⁶¹ that resulted from both. What particularly irks him is what he views as the double standard, one for rich and one for poor countries, in HIV research ethics, epidemiologic science, and health care safety matters.⁸² Double standards have enabled: 1) implementation of studies in which participants were not notified of their HIV-positive test, with all its implications for downstream transmission; 2) neglect for health care safety and for providing explicit warning about the dangers of unsanitary medical procedures; 3) failure to investigate unexplained infections by tracing and testing persons exposed at about the same time in the same medical setting; 4) failure to conduct scientifically defensible studies to resolve dissonances; 5) and failure to publish empiric evidence suggestive of non-sexual transmission.^{20,30,86} As he points out, such public health shortcomings would not have been tolerated in rich countries.

Emerging African voices

It remains remarkable that, three decades into the African HIV epidemics and more than a dozen years since our controversial papers suggested that the epidemiologic picture was not right, comprehensive investigations have still not been conducted by the international agencies responsible for effectively and confidently intervening in the sub-Saharan spread of HIV. One is unlikely to intervene effectively if the respective contributions of different modes of transmission are not solidly documented. In this regard, several groups of native investigators have recently voiced concern about the orthodox view stubbornly clung to by mainstream researchers.

First out of the gate in the new decade was Mapingure and his colleagues who published their puzzling findings in the *Journal of the International AIDS Society*.⁸⁷ The authors examined sexual risk factors for two groups of pregnant women, one from Zimbabwe and the other from Tanzania. Their aim was to elucidate risk factors that could explain the enormous difference in HIV prevalence: 26% in Zimbabwean, and 7% in Tanzanian, women. Counter-intuitively, risky sexual behaviors were more common among Tanzanian than Zimbabwean women. Differences in frequency of sexually transmitted infections among these women differed only moderately, while HIV infection differed by a factor of four. Intriguingly, a history of schistosomiasis was four times greater among Zimbabwean women, providing support for Sawers, Stillwaggon, and Hertz's view that such tropical diseases may influence HIV transmission, directly or indirectly.⁷⁴ Finally, the authors suggest that non-sexual routes of transmission might have played an important role in these substantial differences in HIV

prevalence between the two countries, although such routes were regrettably not measured.

In an exceptionally well-done recent review of currently available information, Duri and Stray-Pedersen list observations that undermine the assertion that sexual transmission is mostly responsible for the observed high HIV prevalence in many regions of Africa.⁸⁸ They detail the multifarious dissonances, paradoxes, and shortcomings in the mainstream orthodox view and, importantly, offer suggestions for obtaining evidence on multiple fronts to solve this puzzle. Of particular concern to these two researchers, based at the University of Zimbabwe, is the real possibility that the pronounced differences noted in different regions of Africa could be explained by unsafe medical practices, such as the re-use of needles and other sharps. In any event, they recommend comprehensive study designs to examine the contributions of sexual behaviors, unsafe puncturing exposures, ethnic variation in HIV restriction genes, nutritional status in susceptible populations, viral characteristics, and co-infection with other pathogens that are common in Africa.

A 2014 article by a group of Kenyan researchers, analyzed data from the population-based representative national AIDS Indicator Survey to assess the magnitude of medical injection use and its relationship with HIV status.⁸⁹ In brief, the authors report that of nearly 14,000 participants, a little more than a third reported receiving one or more injections in the previous twelve months; of these, both men and women were about three times more likely to be HIV-positive than participants who did not report receiving injections during that interval.

Unlike HIV researchers associated with international health agencies and principally European and North American universities, these African researchers may see things clearly not only because their friends and family are subject to the risks they consider, but also because they may not be subject to professional or financial pressures to conform to the consensus view. Perhaps, for them, questioning the orthodoxy would not be accompanied by threats of punishment or banishment. And yet, frankly, I am currently unconvinced that there will be sustained efforts on the part of skeptical Africans to solve the puzzle of rapid HIV transmission in their countries. Not only do I sense a certain passivity towards getting this thing done, but more disquieting is my guess that such a venture may not bring sufficient rewards. Dissenting or skeptical African researchers, it seems to me, are unlikely to be invited into international collaborations or granted funding. In my view, they would also be unlikely to be accorded status from their scientific peers, or accolades from their brethren who are working in government and in medicine. Indeed they could even earn their scorn. Am I misinterpreting the meager cues I've been exposed to in my work with AIDS in Africa, and thus being unduly pessimistic? I hope so.

As for non-African researchers the situation, based on this chapter's content, warrants pessimism. The depressing fact is that research on this topic ("Why Africa?") is dead. Here's this vibrant domain of research and controversy that I and my colleagues have been embroiled in for so many years and, with the exception of some offshoots and a few independent inquiries, as described above, all of the traditionally limited interest has seemingly fizzled.

“We do not believe any group of men adequate enough or wise enough to operate without scrutiny or without criticism. We know that the only way to avoid error is to detect it, that the only way to detect it is to be free to enquire. We know that the wages of secrecy are corruption. We know that in secrecy error, undetected, will flourish and subvert.”
—Robert Oppenheimer

Twelve years before the mast: what I learned from this odyssey

Disappointment. Were I asked to confine myself to a single word to characterize the dozen or so years I spent thinking and writing about AIDS in Africa, this word gets the job done.

1. Disappointment with the failure of the international health agencies to commission scientifically rigorous studies after dissonances and anomalies were (relentlessly) pointed out.^{90 - 93} A decade-and-a-half after these weaknesses in the official view were detailed, they continue to ignore or dismiss pertinent evidence. It is difficult to blame this failure on ignorance. For example, Dr. Peter Piot, director of the world’s leading AIDS agency (UNAIDS) from its creation in 1995 until the end of 2008, was one of the earliest and savviest researchers on the ground in Africa with Project SIDA (French acronym for AIDS) in Kinshasa, Zaire. Here are the recommendations he published in the African Journal of Sexually Transmitted Diseases in 1986: “Other possible routes of transmission that should be studied include scarification rituals, tattooing, male and female

circumcision and inadequate sterilization of needles re-used for medical treatment.” And: “Further research is needed to accurately determine all risk factors for AIDS transmission in Africa, to determine the actual extent of AIDS, to work out control strategies, and determine the impact on other health facilities.”⁹⁴ (Emphasis mine, in both sentences.) No one in our informal group could have articulated it better. Similar conclusions were published in the prestigious journal *Science* that same year.⁹⁵

What happened? What made such researchers ignore their own considered, prescient advice? Or ours—and that of other skeptics—which was along similar lines? Finally, how many AIDS cases could have been prevented by conscientious implementation of these early researchers’ advice? Or of other similarly minded researchers? 10,000? 100,000? 1,000,000? More? Finally, it does not engender trust in the official view to know that our informal group has solid evidence of several instances by international health agencies actively working to suppress findings supportive of non-sexual transmission and to discourage research into non-sexual transmission.^{28,30,65,86,96}

2. Disappointment with the generally inadequate studies conducted by academic researchers from European, British, and North American universities. None implemented field studies that comprehensively took account of non-sexual exposures in sub-Saharan Africa or in other Third World countries. (If done, they were not published.) As Daniel Sarewitz pointed out: “A biased scientific result is no different from a useless one”.⁹⁷ Their studies were almost always designed with “heterosexual transmission” as the frame of reference and, more often than not, relied on ecologic evidence,³¹ anemic (read: missing non-sexual) risk factor assessment,

inferential reasoning, and logic, rather than scientifically-relevant data. Worse, they were mired in group-think. What, to me, truly distorted epidemiological research was deference to unsound yet established theories of HIV transmission in Africa. (In fact this reminded me of the Western intelligentsia's misguided belief, during the 1930s and 1940s, that Marxism/Utopian Communism would save humanity, and how this fervently-held belief blinded them to the brutalities of Soviet totalitarianism. True Believers saw what they wanted to see. Did AIDS academic researchers suffer from a similar intellectual straightjacket?)

3. Disappointment with the seemingly partisan leanings of scholarly journal editors, especially by editorial staff in high-impact journals. Not only did they generally reject our manuscripts, but they also often declined to publish our rebuttals to articles they published. This was our own, palpable introduction to publication bias.

“In the end, we will remember not the words of our enemies,
but the silence of our friends.”

—Attributed to Martin Luther King, Jr.

4. Disappointment with so many (non-Colorado Springs) colleagues in the STD/HIV fields who remained silent on the sidelines, both officially and personally. Their reticence was difficult to understand not only because I had expected that, being scientifically trained, they would have greater respect for skepticism than for the received wisdom but, especially, because they knew of the high quality epidemiologic research I had done for decades. They

certainly knew that I was—even if I personally lacked gravitas and had a reputation as a gadfly—an expert in STD/HIV epidemiology and control, having contributed cutting-edge empiric studies for decades. And they knew that I certainly was not some sort of ideologue, whether Duesbergian or Mbekian denier, Anti-condomer, Anti-circumcisioner, or Pro-Abstinencer. True to my Swiss roots, I was neutral and pragmatic. I speculate that part of the reason for their silence may have been the same as it may have been for the international health agencies: the so-called Adverse Consequences Fallacy:⁹⁸ the error of evaluating the validity of an argument by considering its potential negative consequences. Whatever the cause of their silence, it was deafening and deeply disappointing.

5. Disappointment with the lack of truly scientific, as opposed to politically- or ideologically-motivated, debate. As I've said elsewhere: "I 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."³³

Two centuries ago, a German philosopher articulated this (probably relevant) insight: “How unwillingly we think of things which powerfully injure our interests, wound our pride, or interfere with our wishes, with what difficulty do we determine to lay such things before our intellects for careful and serious investigations...in that resistance of the will to allowing what is contrary to it to come under the examination of the intellect lies the place at which madness can break open the mind.” (Schopenhauer, 1818)

6. Disappointment with misguided *ad hominem* comments. Other than the inappropriate name-calling referred to earlier, there were frequent and irritating instances of (irrelevant) deprecating comments. For example: “The American authors are not linked to a university.”⁹⁹ (The authors referred to were Gisselquist and me.) The comment’s intent was clearly to devalue our view, since it insinuates that valid work or critical thinking is not possible outside academia. I’ve often wondered how many readers of our papers dismissed their content based on similar considerations. Another not infrequently leveled accusation was that among our informal group were “scientists, some of whom have an insufficient understanding of basic epidemiological principles”.¹⁰⁰ A truly gratuitous assessment, for it was unencumbered by evidence other than what the authors wanted to believe. Certainly the totality of our (the informal group) published work seriously challenged this belief. Along similar lines was the dismissive comment that our papers were: “...a propagandist message based on distinctly flimsy analysis and inference.” This comment was emailed to the Royal Society of Medicine on 19 April 2003 by a person who had been editor-in-chief of the prestigious American Journal of Public Health. It was certainly not lost on any of us that his assessment perfectly fit our view of the official version of

HIV dynamics in Africa promulgated by the international health agencies! Many similar comments, made by people who should know better, were based on the arrogance of belief rather than the humility of doubt. Voltaire once said: "Doubt is not a very pleasant state, but certainty is a ridiculous one". This is a turn of mind regrettably missing from many in the international agencies and in academia who defend the consensus view.

Glimpsing into the future

It is entirely possible that we may never know what truly drove, and is currently driving, the HIV epidemics in sub-Saharan Africa. By dismissing or ignoring evidence that undermines the consensus view, mainstream agencies and researchers have effectively discouraged new research. The easiest way this is done is to refuse to fund proposals that challenge the consensus view or/and refuse to reward independent-minded researchers with advancement in academia.

No less a brilliant observer of enforced orthodoxy than George Orwell said it far better than I ever could:

At any given moment there is an orthodoxy, a body of ideas of which it is assumed that all right-thinking people will accept without question. It is not exactly forbidden to say this, that or the other, but it is 'not done' to say it...Anyone who challenges the prevailing orthodoxy finds himself silenced with surprising effectiveness. A genuinely unfashionable opinion is almost never given a fair hearing, either in the popular press or in high-brow periodicals.

Amen.

Again from Orwell:

The point is that we are all capable of believing things which we know to be untrue, and then, when we are finally proved wrong, impudently twisting the facts so as to show that we were right. Intellectually, it is possible to carry on this process for an indefinite time: the only check on it is that sooner or later a false belief bumps up against solid reality, usually on a battlefield.

The shocking recent report of hundreds of HIV infections in Roka village, western Cambodia, apparently due to skin puncturing medical procedures administered by a village practitioner may be such a battlefield.¹⁰¹ This tragic outbreak can certainly serve as proof of concept that turbocharged HIV transmission can be generated by contaminated injections and other invasive medical procedures.

“Cultivate a taste for distasteful truths. And...
most important of all, endeavor to see things
as they are, not as they ought to be.”

— Ambrose Pierce

The paradigm that failed: Phoenix should rise from its ashes

This was a difficult and, at times, painful chapter to write. And it is even more painful for an irrepressible optimist like me to end on a negative note. And so I won't.

We've come full circle and again ask: Why Africa? What is it about conditions in many parts of sub-Saharan Africa that HIV is so efficiently

transmitted in its heterosexual populations, a phenomenon experienced nowhere else on earth? The short answer still is: we don't know. I certainly don't know. But neither do they—"they" being the international health agencies and the preponderance of the academic researchers who study HIV transmission in Africa. And certainly, shortcomings in our arguments do not, *ipso facto*, provide support for the consensus view. The quality of the evidence they rely on for asserting that unprotected penile-vaginal intercourse accounts for the vast majority of infections in African adults is not high enough to be scientifically trustworthy. Were their assertion not undermined by persistent evidence suggesting a substantial role for non-sexual HIV transmission, there would be little reason to worry about quality of evidence. Indeed, it is the multifarious facts that don't fit which demand a higher standard of evidence. Neither weak evidence nor wishful thinking can get the job done of persuading thinking people that the present consensus view is correct. It is especially disconcerting to note that, with so much at stake for implementing correctly targeted interventions in Africa, there has been such stubborn and sustained reluctance on the part of researchers and international health agencies to resolve incongruities and get the picture right, using comprehensive research designs. It is even more disconcerting to realize that they had twice recommended³² or promised²⁶ to look into non-sexual modes of transmission and twice failed to follow through.

What does one say about a paradigm, promulgated as the consensus view more than a quarter of a century ago, that has not been modified since, in light of respectable evidence, old and new, clearly indicating the need to revisit this view? The answer can only be: ossified dogma—dogma maintained by the weight of authority and tradition, not quality evidence.

And one can only speculate, as I did above, about the vested interests maintaining what Dr. Gisselquist calls the epiganda (a contraction of “epidemiologic propaganda”) which discourages taking a fresh look. At the very least, readers should ask the establishment researchers and health agencies charged with monitoring and intervening in HIV epidemics why they have settled for evidence from a lesser god when the stakes for getting the picture right are so high.³⁵ Africans need a picture based on rigorous epidemiologic science. What they have received so far, regrettably, is more political, than rigorous, science. Africans deserve better than the inadequately supported views of the heterosexual transmission fundamentalists.

There is a way forward. Its starting point is to recruit the best possible epidemiologic study designs and conscientiously implement them in several different regions of sub-Saharan Africa. Above all there is ample reason to care, on the deepest level, because taking a fresh look is far more about humanitarian considerations than about rigorous science.

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