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REVIEW

Let it be sexual: how health care transmission of AIDS in Africa was ignored

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Summary: The consensus among influential AIDS experts that heterosexual transmission accounts for 90% of HIV infections in African adults emerged no later than 1988. We examine evidence available through 1988, including risk measures associating HIV with sexual behaviour, health care, and socioeconomic variables, HIV in children, and risks for HIV in prostitutes and STD patients. Evidence permits the interpretation that health care exposures caused more HIV than sexual transmission. In general population studies, crude risk measures associate more than half of HIV infections in adults with health care exposures. Early studies did not resolve questions about direction of causation (between injections and HIV) and confound (between injections and STD). Preconceptions about African sexuality and a desire to maintain public trust in health care may have encouraged discounting of evidence. We urge renewed, evidence-based, investigations into the proportion of African HIV from non-sexual exposures.

Keywords: HIV, Africa, nosocomial, iatrogenic, risk factors

Introduction

The conventional wisdom that heterosexual transmission accounts for most adult HIV infections in Africa emerged as a consensus among influential HIV/AIDS experts no later than 1988. In that year, the World Health Organization's (WHO) Global Program on AIDS circulated estimates that 80% of HIV infections in Africa was due to heterosexual transmission, 10.8% was from mother-to-child transmission, 6% from blood transfusions, 1.6% from contaminated medical injections and other health care procedures, and 1.6% from men who have sex with men (MSM) and injection drug use (IDU)[1]. In the same year, experts from Zaire's National AIDS Control Program and the United States (US) Centers for Disease Control published comparable estimates[2]. By mid-1989, an overview of global HIV epidemiology by leading AIDS experts at the Fifth International Conference on AIDS, did not even mention medical injections as a risk for HIV[3].

If experts had treated the consensus as an hypothesis—which it was and still is—and had used it to guide research to test competing hypotheses, it could have played a constructive role. Unfortunately, many experts have accepted the consensus as fact, and have not seen any need for further research to test its estimates. The result has been that the consensus has suppressed inquiry and dissent. Hence, from 1988 the consensus has been self-reinforcing, as researchers in Africa—and in Asia and the Caribbean—have often assumed sexual transmission without testing partners, without asking about health care exposures, and when conflicting evidence nevertheless emerges—such as infected adults who deny sexual exposures to HIV—routinely rejecting it.

From the beginning, AIDS in Africa has been a puzzle. In 1983, physicians in France and Belgium reported AIDS diagnoses in African men and women seeking health care in Europe[4–6]. Initial hospital-based surveys in Kinshasa, Zaire (now Democratic Republic of the Congo [DRC]), and Kigali, Rwanda, in late 1983 reported an AIDS incidence comparable to large US cities, with cases evenly distributed between men and women, and without notable representation by MSM or IDU[7,8]. In contrast, in the US through late 1983 less than 1% of 2800 AIDS cases were diagnosed in heterosexual partners of persons in high risk groups (MSM, IDU, and recipients of blood products), and there were 14 men for every woman with AIDS[9].

During 1983–1988 experts debated the relative importance of health care versus heterosexual transmission in Africa[10–14]. A meeting convened by WHO in late 1983 identified unsafe injections and transfusion of untested blood as risks in tropical countries and was undecided about

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Table 1. Risk factors for HIV from studies 1984–1988 among general population groups, inpatients and outpatients, and high-risk men

Country, year, reference	Sample: cases/total	Exposure	Reporting period	PAF			APAF
				ρ (%)	RR	PAF (%)	(%)
General population studies							
Congo, 1987–88[18]	W at mother and child clinics: 71/1,833	Transfusion	8 yrs	2.8	3.24	6	
		Induced abortion	Ever	16	1.66	10	
		>1 sex partner‖	1 yr	11	1.79	8	
DRC, 1984[19]	Hospital workers: 152/2,384	Injections	3 yrs	81	1.83	40	
		Transfusions	10 yrs	5.1	1.90	4	
		Scarification	3 yrs	5.0	1.12	1	
		Foreign travel	10 yrs	16	1.24	4	
DRC, 1984–86[20]	Hospital workers: 62/1,905	Injections	INC	73	1.54	28	21†
		Transfusion	INC	2.0	4.54	6	
		Scarification	INC	4.0	0	<0	
DRC, 1987–88[21]	Workers and wives: 411/11,616	Transfusions	5 yrs	3.3	2.33	4	
	Workers: 236/7,068	Sex w/prostitute	2 yrs	23	1.36	8	
		>0 nonmarital sex partners¶	1 yr	29	1.63	15	
		GU	5 yrs	7.6	2.72	12	
		Urethritis	5 yrs	13	1.83	10	
	Workers' wives: 175/4,548	Induced abortion	5 yrs	4.1	2.34	5	
		>1 sex partner	1 yr	0.7	4.29	2	
		GU	5 yrs	14	2.41	16	
Rwanda, 1985[22]	Hospital and urban workers: 80/452	Injections (all)	2 yrs	77	2.42	52	
		Injections (for STD only)	2 yrs	30	2.23	27	20†
		Transfusions	5 yrs	4.0	3.44	9	
		STD	2 yrs	44	2.1	33	13‡
	Urban working men: 52/302	Scarification	2 yrs	15	0.62	<0	
		Not circumcised	Current	90	0.89	<0	
Rwanda, 1985[23]	Rural A: 7/206	Transfusions	5 yrs	0	Und	0	
		STD	2 yrs	11	11.2	52	
		Travel to an urban centre	5 yrs	12	18.1	67	
Rwanda, 1986*[24]	M in HIV+/+ vs HIV-/- couples: 124/150	Transfusions	2 yrs	1	1.21	0	
		Sex w/prostitute	2 yrs	21	12.6	71	
		STD	2 yrs	15	10.1	58	
		Not circumcised	Current	70	1.04	3	
		Travel in Rwanda	2 yrs	30	1.47	12	
	W in HIV+/+ vs HIV-/- couples: 124/150	Transfusions	2 yrs	4	2.34	5	
		STD	2 yrs	11	7.10	41	
Rwanda, 1988[25]	ANC and paediatric clinic W: 32%/1,428	Transfusions	Ever	6.8	1.43	3	3§
		>1 sex partner	Ever	32	1.89	22	16§
		STD	5 yrs	17	2.06	16	14§
		Partner not circumcised	Current	66	1.07	4	
		Good income**	Current	55	1.45	20	24§
		Own education >4 yrs	Current	62	1.11	7	
		Partner's education >4 yrs	Current	81	1.82	40	
Tanzania, 1987[26]	Urban GP A: 134/533	Injections	8 yrs	90	3.0	64	
		Transfusions	8 yrs	5.6	1.1	1	
		>1 sex partner	8 yrs	77	1.7	34	
		Travel out of region	8 yrs	61	1.0	0	
	Rural GP A: 86/1,744	Injections	8 yrs	79	2.6	56	
		Transfusions	8 yrs	3.7	3.8	9	
		>1 sex partner	8 yrs	55	2.3	41	
		Travel out of region	8 yrs	26	1.7	16	
Uganda, 1987[27]	GP A: 417/3,879	Injections	1 yr	66	1.68	31	30‡
		STD	5 yrs	20	1.64	11	7‡
	GP M: 158/1,799	>1 sex partner	6 mos	41	1.22	8	
	GP W: 262/2,091	>1 sex partner	6 mos	13	1.67	8	
Zambia, 1987[28]	W at childbirth: 227/1,954	Transfusions	Ever	8.2	1.84	6	
		> 1 sex partner	Ever	54	1.27	13	
		GU	Ever	1.0	1.73	1	
		Travel abroad	Ever	11	1.65	6	

continued

Table 1: *Continued*

Country, year, reference	Sample: cases/total	Exposure	Reporting period	PAF			APAF
				ρ (%)	RR	PAF (%)	(%)
Zambia, 1987–88[29]	Postpartum W: 16/634	Transfusions	INC	1.4	9.79	11	
		GU	INC	2.4	13.6	23	
Zimbabwe, 1987*[30]	Blood donor M: 69/119	Injections	Ever	95	3.61	71	
		Transfusions	Ever	6.7	1.08	1	
		Scarification	Ever	32	1.95	23	
		Sex w/prostitute	Ever	55	1.72	28	
		STD	Ever	73	3.86	68	
		Travel outside Zimbabwe	Ever	20	0.83	<0	
In and outpatient studies (except STD, pregnancy, and childbirth)							
DRC, 1984*[31]	Inpatients all ages: 17/236	Injections	3 yrs	90	Und	100	
		Transfusions	3 yrs	23	6.00	54	
		Scarification	3 yrs	17	6.34	48	
DRC, 1984–85*[32]	Inpatients 2–14 yrs: 40/328	Injections	1 yr	82	4.08	72	
		Transfusions	Ever	33	3.10	41	
		Scarification	1 yr	20	1.32	6	
DRC, 1985*[33]	Inpatients 1–24 mos w/HIV-mothers: 16/222	Transfusions	Ever	6.8	6.27	26	
		Scarification	Ever	15	0.38	<0	
DRC, 1986*[34]	In and outpatients 1–13 yrs 31/812	Transfusions	Ever	14	15.6	66	
DRC, 1988*[35]	Outpatients 1–13 yrs: 29/695	Transfusions	Ever	17	5.40	42	
Rwanda, 1984–86*[36,37]	Inpatients 1–48 mos w/HIV-mothers: 18/61	Transfusions	Ever	6.6	9.07	35	
Uganda, 1987*[38]	In and outpatients, mostly adults: 559/745	Injection in the market	5 yrs	38	1.24	8	
		Injections in medical facilities	5 yrs	84	0.86	<0	
		Traditional skin piercing	5 yrs	23	0.75	<0	
		Travel w/in Uganda	5 yrs	58	1.33	16	
	In and outpatient M, mostly adults: 252/342	Sex w/prostitute	5 yrs	25	1.25	6	
Uganda 1986*[39]	In and outpatient men: 10/76	Sex w/prostitute	5 yrs	41	13.1	83	
High risk men							
Ethiopia, 1988[40]	Men in an Ethiopian prison: 27/450	Injections	5 yrs	62	1.24	13	
		Sex w/prostitute	Ever?	64	2.45	48	
		Syphilis (VDRL)	Current	31	3.73	46	
Uganda, 1986[41]	Drivers and turnboys: 24/68	Syphilis (TPHA)	Ever	43	1.88	27	
		GU	Ever?	29	2.84	35	
		GD	Ever?	51	2.29	40	
Sudan, 1987–88[42]	Soldiers: 13/773	Sex w/prostitute	Ever	52	3.12	52	
		STD	Ever	31	2.64	33	
		Any HBV marker	Current	78	Und	100	

ρ = percent of total sample or cases exposed; RR = rate ratio; PAF = population attributable fraction; INC = observation interval in studies of HIV incidence; APAF = adjusted PAF; ANC = antenatal clinic; HBV = heptatitis B virus; STD = sexually transmitted disease; VDRL = veneral disease research lab test; TPHA = *Treponema pallidum* haemagglutination test; GU = genital ulcer; GD = genital discharge; GP = general population; M = men; W = women; A = adults; DRC = Democratic Republic of the Congo; Und = undefined

*Case–control study or equivalent (e.g., studies where all or many cases are inpatients), for which the table shows numbers of cases/controls, exposures among controls, OR instead of RR, and the PAF is approximated as ρ(OR−1)/(1+ρ[OR−1])

†Adjusted for direction of causation by stratifying across symptoms. For hospital workers in DRC, the adjusted PAF is calculated after excluding six seroconverters with HIV-related symptoms. For hospital and urban workers in Rwanda, the adjusted RR for injections for STD is calculated among those reporting STD, and the table shows HIV infections associated with injections for STD as a per cent of all infections

‡Adjusted for confound by stratified analysis. Adjusted RRs for STD are based on the difference in HIV prevalence between persons with and without STD and without injections (or without injections for STD); this RR is used to estimate the number of HIV infections associated with STD and the adjusted PAF for STD. Adjusted PAFs for injections are based on differences in HIV prevalence between persons with and without injections and STD. See further explanation in the text (Discussion). For Uganda, Konde-Lule *et al.* report numbers with and without STD, and of those figures, numbers without injections; our calculations assume that all others had 1 or more injections

§Adjusted for marriage, children, adjusted variables in the table, other variables. The APAF is estimated as APAF ≈ PAF([AOR−1]/AOR)/([OR−1]/OR), which assumes that the ratio PAF/APAF is equivalent to the ratio of PAF (≈[OR−1]/OR) to APAF (≈[AOR−1]/AOR)

‖During the year before pregnancy

¶Roughly 10% of the men were unmarried

** Partner's income > 10,000RWF/month (roughly $100/month)

heterosexual promiscuity as a risk[9]. On the other hand, some influential early studies associated AIDS in Africans with large numbers of heterosexual partners: for example, three of seven Rwandan women with AIDS in a 1983 study were prostitutes[8] and in another early study, 58 African men with AIDS symptoms reported a median of 32 sex partners per year[15].

Early findings on risk factors for AIDS in Africans may have been influenced by preconceptions about where to look. Through mid-1984, unavailability of serological tests for HIV and an unexpected mix of opportunistic infections in Africans with AIDS impeded epidemiological research. Discovery of the lymphadenopathy associated virus (later renamed human immunodeficiency virus [HIV]) in 1983 led to reliable laboratory markers of infection and—despite some early problems with false positive results[16]—much better information on risk factors for HIV in Africa.

In this communication we document premature closure of the debate about the relative importance of heterosexual and health care exposures to Africa's HIV/AIDS epidemic. We show that epidemiological evidence from field studies completed through 1988 allowed that health care transmission was not only significant, but might well have been responsible for more HIV than heterosexual (hereafter sexual) transmission, and we discuss how this evidence might have been ignored. (Since Africa has many formal and informal health care providers and settings[17], we use the terms iatrogenic and nosocomial to refer to infections from all providers and all settings—not only doctors and hospitals.)

Methods

Using Medline and other bibliographic resources, we searched for articles in refereed medical journals on HIV epidemiology in Africa based on field research completed through 1988. We include all identified studies conducted among general population groups, patients, and high risk men (eg, truck drivers) that provide data to calculate population attributable fractions (PAFs) of HIV-associated risks. For these studies, we calculate and report all available PAFs for HIV associated with medical injections, blood transfusions, induced abortion, scarification, more than one sexual partner, prostitute contact, sexually transmitted disease (STD), lack of circumcision, and selected socioeconomic variables. From these papers, we summarize all information on distribution of HIV in general population adults according to sexual activity. From these studies and others, we present selected information on HIV in children. We also include all identified studies of prostitute women and STD patients, for which we report all available information on injections and selected information on sexual and other risk variables. Although we have undoubtedly missed some studies, and although we exclude conference abstracts, the internal consistency of the evidence we present suggests that further research into pre-1988 knowledge about HIV epidemiology in Africa will not change any of the major elements of our analysis.

Results

PAFs from studies of general population, hospital, and high risks samples and cohorts

Eleven prevalent and two incident studies of risk factors for HIV in samples and cohorts from the general population (eg, general population, blood donors, postpartum women) with field research completed through 1988 provide data to calculate PAFs for one or more risks (Table 1). In summary, seven PAFs for HIV associated with injections (two of which are adjusted; see notes to Table 1) average 48%; these and other studies[43] show high rates of exposure to medical injections. The 14 available PAFs for risk by blood transfusion average 5%. Four PAFs for scarification range from $<0\%$ to 23% and average 6%. The nine PAFs reflecting risk for reporting more than one sexual partner average 16%. The three PAFs for contact with prostitute women average 36% (range: 8–71%). Lastly, the 12 PAFs for reported or current STD (2 of which are adjusted; see notes to Table 1) average 27%. Thus the largest average PAF, 48%, is for medical injections.

Nine studies of risk factors for HIV among inpatients and outpatients (except parturient women included in general population studies and STD patients considered below) present sufficient data to calculate PAFs for prevalent HIV for one or more variables (Table 1). Again, the average of four PAFs for medical injections is high, 45% (setting the one negative PAF to 0), while the average of 6 PAFs for blood transfusions is also high, 42%. Four PAFs for scarification range from $<0\%$ to 48%. Two PAFs for contact with prostitutes are 6% and 83%. Finally, data from three studies of high risk men (soldiers, prisoners, and truck drivers and helpers) provide two PAFs for prostitute contact of 48% and 52%; and five PAFs for reported or diagnosed STD average 36%.

Overall, crude PAFs from general population studies through 1988 suggest that medical exposures were responsible for more African HIV than sexual exposures. Importantly, PAFs point to injections—not blood transfusions—as the main health care risk. In addition, early studies also suggest high risk associated with prostitute contact and STD. Considering that many of the PAFs for HIV prevalence recorded in Table 1 were calculated from exposures spanning the previous one to five years, they fail to reflect risks for HIV

Table 2: Distribution of HIV according to sexual activity in general population studies

Country, year, reference	Sample, sex	% HIV+	Sexually least active			Sexually most active		
			Partners per unit time	% of sample	% of HIV	Partners per unit time	% of sample	% of HIV
Congo, 1987–88[18]	ANC W	4.0	1 partner last year	89	82	>1 partner last year	11	18
DRC, 1987–88[21]	Workers M	3.3	0 non-marital	72	61	>0 non-marital	2.3	5.4
	Wives W	3.8	partners last year	99	97	partners last year	0.7	2.7
Rwanda, 1986[24]	M in HIV+/+ *vs* HIV−/− couples	~20*	0 prostitute contact per month	68*	23*	⩾2 prostitute contacts per month	9.9*	29*
Rwanda, 1988[25]	ANC W	32	1 lifetime partner	68	53	>2 lifetime partners	13	24
Tanzania, 1987[26]	Urban GP M and W	24	0–1 partner last	~31†	~16†	>1 partner last	~69†	~84†
	Rural GP M and W	5.0	8 years	~52†	~28†	8 years	~48†	~72†
Uganda, 1987[27]	GP M	8.8	0–1 partner last	59	54	⩾6 partners last	4.7	7.6
	GP W	13	6 months	87	80	6 months	0.7	0.8
Zambia, 1987[28]	W at delivery	12	1 lifetime partner	46	41	⩾6 lifetime partners	4.3	8.8
Zimbabwe, 1987[30]	Workers M	6	⩽15 lifetime partners	78‡	68‡	>15 lifetime partners	22‡	32‡

* Calculated from reported sexual behaviours for cases and controls, assuming 20% prevalence in the sample (cf: 18% urban prevalence reported in Rwanda's 1986 national survey[46])
†Percents estimated from reported risk ratios and percents exposed for any sexual experience and partner change in the last eight years
‡Calculated from reported sexual behaviours for cases and controls and 6% reported prevalence in the sample from which cases and controls were chosen

contracted before this reporting period; hence, some PAFs may be misleadingly low. On the other hand, as discussed in a later section, PAFs for injections and STD may be inflated by reverse causation (ie, people with HIV symptoms seeking injections, and people with weakened immune systems having more STD), and PAFs for STD and prostitute contact may be inflated by confound with injections to treat STD.

HIV distribution according to sexual behaviour in general population studies

STD transmission is associated with core groups, ie, a small proportion of individuals that account for most of the STD burden[44,45]. One would expect that, insofar as HIV is sexually transmitted, the distribution of HIV would show the same pattern. Yet, most studies that reported the distribution of HIV among adults in the general population showed little concentration according to sexual activity (Table 2). Most studies showed a majority—often a large majority—of prevalent HIV infections in both men and/or women with 0–1 partners in the reporting interval (from six months to lifetime). Notably, data in Table 2 show no obvious trend for greater concentration according to sexual activity in communities with lower prevalence and hence supposedly earlier epidemics (when core groups were supposed to account for more infections).

For example, in a 1987–88 study of factory and bank workers and their wives in Kinshasa, the 99% of wives who reported no non-marital sex in the previous 12 months had 97% of HIV in wives. Among men, the 71% who claimed no non-marital partners in the last year had 61% of HIV in men, while the 2.3% reporting five or more non-marital partners had only 5.4% of HIV in men (10% of men who were not married may have accounted for some non-marital partners)[21]. Although some men probably contracted HIV from non-marital partners and subsequently transmitted the infection to their wives, crude PAFs for HIV in men associated with any non-marital sex and prostitute contact in the previous two years—15% and 8%, respectively—were too low for this to explain more than a fraction of infections in men or their wives; furthermore, in nearly half (90/204) of serodiscordant couples, the wife was the HIV-infected partner.

Finally, in a study of HIV prevalence in couples in Rwanda, 15 of 25 women with HIV and HIV-negative husbands reported one lifetime sex partner only[47]. Although some adults may have under-reported numbers of sexual partners, the consistency of the evidence suggests a large majority of HIV infections in non-promiscuous adults, and little concentration in the general population according to sexual activity.

HIV associated with socioeconomic variables

Field studies completed through 1988 indicated that HIV prevalence was often associated with

urban residence, education, travel, and other socioeconomic variables reflecting higher status. For example, Rwanda's 1986 national survey reported an HIV prevalence of 18% in urban areas compared to 1.3% in the countryside[46]. A 1987 study in Kagera District, Tanzania, reported 24% adult HIV prevalence in Bukoba town, 10% in adjacent rural, and 0.4% in remote rural, areas[26].

A 1988 study in Rwandan women showed PAFs for prevalent HIV of 7% for educational attainment exceeding four years and 40% for husband's education exceeding four years[25]. The average of seven PAFs for HIV associated with travel in general population studies was 15% (Table 1). A 1984 seroprevalence survey among employees at Mama Yemo Hospital in Kinshasa found 9.2% HIV prevalence among high-level administrators compared to 6.4% for all employees[19]. In two businesses in Kinshasa in 1987, HIV prevalence was greater in higher paid employees than in manual labourers (6.8% compared to 4.2% in one business; 4.6% compared to 2.8% in the other)[21].

Presumably, associations between variables measuring socioeconomic status and HIV are due to correlations between status and sexual and/or medical exposures. Without empiric determination of such correlations, experts in the late 1980s speculated that differences in sexual behaviour according to status explained the associations. Since STD have long been associated with lower socioeconomic and educational attainment[48], it was at least equally plausible that associations between high status and HIV pointed to differences in health care rather than sexual behaviour.

HIV in children

Through 1988, a number of hospital- and community-based studies reported HIV infections in children that could not reasonably be attributed to vertical transmission. In 1985, Mann and colleagues in Kinshasa found 17 (39%) of 44 HIV-positive inpatient and outpatient children 1–24 months old to have HIV-negative mothers (C+M- children)[33]. In a case–control study of risk factors for HIV—with C+M- cases and C-M- controls (ie, uninfected children and mothers)—the PAF for transfusions was 26% (Table 1), and C+M- children averaged 44 lifetime injections compared to 23 for C-M- children. During 1984–86, Lepage and colleagues found that 18 (24%) of 76 children 1–48 months old with symptomatic HIV infections at a hospital in Rwanda had HIV-negative mothers[36]. In a similar case–control study, the PAF for blood transfusions was 35%, while C+M- children averaged 23 lifetime injections compared to 16 for C-M- children. (The authors argued that injections were not a risk, proposing that mothers who tested negative may have been infected. On re-testing, three of 12 initially HIV-negative mothers were HIV-positive, leaving 15 [20%] of 76 HIV-positive children with HIV-negative mothers[36,37].) Since these studies took place in hospitals with ongoing HIV research, health care providers may have taken more care to ensure sterile practices than providers in other health care settings with less attention to HIV. On the other hand, risks for children in these studies may have been higher than for rural children not subject to so many invasive medical procedures.

Many other studies reported HIV in children without, unfortunately, testing mothers. Even so, HIV in African children between the ages of five and 14 probably indicates some non-vertical transmission because low adult prevalence prior to the late 1980s and high mortality in HIV-infected children argues against vertical aetiology. In a study at Mama Yemo Hospital in Kinshasa in 1984–85, 11% of paediatric inpatients 2–14 years old were HIV-positive[32]. Other studies during 1984–88 reported HIV in children in the general population (Table 3). If we consider that children 0–15 years old comprise roughly half of African populations, even low rates of HIV prevalence in children can point to significant numbers of infections. For example, data from a 1984–85 survey in Kinshasa suggest that infected children 2–14 years old had one-eighth of all HIV infections in persons aged two years and older (see Table 3; calculating that 40% of DRC's population was 2–14 years old compared to 52% aged 15 years and older[50,51]).

Table 3. HIV in children and adults in selected population-based studies, 1976–1988

	Children		Adults	
Country, location, year	Ages	% HIV+ (HIV+/tested)	Ages	% HIV+ (HIV+/tested)
Rwanda, urban, 1986[46]	6–15	4.2 (10/238)	>15	20.8 (308/1,484)
Rwanda, rural, 1986[46]	6–15	1.7 (2/115)	>15	1.5 (8/518)
DRC, rural, 1976[49]	5–14	0.6 (1/160)	>14	1.1 (5/447)
DRC, Kinshasa, 1984–85[50]	2–14	1.3 (3/230)*	>14	6.5 (287/4,449)*
Tanzania, urban Kagera, 1987[26]	5–14	1.9 (4/215)	>14	24.2 (132/553)
Tanzania, rural Kagera, 1987[26]	5–14	0.2 (2/985)	>14	5.0 (87/1,744)

*Sample of 'healthy persons'

Similarly, data from a population-based survey in Rwanda suggest that urban children aged 6–15 years had 8% of infections in urban persons six years and older (see Table 3; calculating 27% of the population aged 6–15 years and 50% aged 16 years and older[46,52]). Limited data from rural Rwanda in 1986[46] and rural DRC in 1976[49] suggest even higher proportions of HIV in children (Table 3).

On the other hand, HIV prevalence in children 0–14 years old was much lower in Côte d'Ivoire in a population-based survey in 1987[53] and was zero in a number of studies in Gabon, Cameroon, Central Africa, Equatorial Guinea, and Uganda in 1985–87[54,55]. Although some studies tested too few children to derive sound estimates, and findings vary from country to country, studies through 1988 suggest non-trivial numbers of non-vertically acquired HIV infections in African children. Without explicit evidence, it is not reasonable to attribute more than a small number of these infections to early sexual activity and child sexual abuse.

Prostitute women and STD patients

During 1984–88, researchers working principally through STD clinics conducted many studies of HIV in prostitutes (Table 4). A 1985 study in Nairobi reported health care exposures during the previous five years among 64 low- and 26 high-class prostitutes: 97% reported medical injections; 97% immunizations; 56% scarification; 29% surgical procedures; 21% dental extractions; 10% induced abortions; and 10% reported blood transfusions[62]. Since almost all prostitutes reported medical injections, assessing risks from injections required detail on numbers of injections, which was regrettably not collected. Virtually all other studies of prostitute women and STD clinic visitors in Africa during 1984–88 either failed to collect information on injections, collected information only on injections received elsewhere (outside the study clinic), or failed to report collected information. Several studies even reported no association without providing the data, leaving readers clueless about trend.

Much of the evidence on sexual risks for HIV in studies of prostitute women is confusing or ambiguous. Many studies found little or no association between years in prostitution or numbers of partners per unit time and HIV prevalence (Table 4). Many studies reported one or more STD as a major risk, which could implicate either sexual transmission or the injections used to treat STD. Condom users had higher prevalence in one study[57]. On the other hand, several studies reported that HIV infection was associated with lack of condom use and/or with hormonal birth control methods; these could implicate sexual transmission without condoms, injections to treat additional STD, hormonal enhancement of sexual transmission, or other risks[59,63,64].

Comparison of HIV prevalence and incidence in STD clinics with prevalence in general population studies suggests that risk for HIV infection was associated with clinic attendance (Table 4). In two STD clinics in Rwanda, HIV prevalence in attendees was four to nine times higher than in controls (general population samples)[23,66]. Among STD outpatients in Zambia in 1985, HIV prevalence in those reporting previous attendance at an STD clinic was 37% compared to 23% for first-time attendees[72]. In another study in Zambia, 15% of HIV-negative STD patients seroconverted within two years[73]. Among men attending an STD clinic in Nairobi in 1986–87 after recent contact with prostitute women, 8% seroconverted within an average 15 weeks of follow-up[70]. Reported differences in HIV prevalence between clinic patients and controls and before and after STD treatment exceed differences in general population studies between persons with and without a history of STD. In 1985, among hospital and urban workers in Rwanda. HIV prevalence was only 1.4 times greater in persons reporting STD but no injections for STD in the past two years compared to persons reporting no STD (16.9% compared to 11.9%)[22]. Similarly, in a general population sample in Uganda in 1987, HIV prevalence in adults reporting STD in the past five years but no injections in the past year was only 1.6 times greater than for those reporting no STD or injections (10.6% compared to 6.8%)[27].

Discussion

As detailed, published epidemiological evidence from 1984–88 in Africa shows higher average crude PAFs for HIV associated with injections than with measures of sexual exposure. Information on distribution of HIV infections according to sexual activity in adults from the general population shows most HIV in sexually less active adults. Studies of HIV in infants and children show significant numbers with proven or presumed non-vertically acquired infections, implicating medical transmission. A large number of STD-clinic-based studies of prostitute women and STD patients reported relatively high rates of HIV prevalence, but did not show consistent associations with sexual variables (except STD), and none of these studies provided data and analyses to resolve confounding between sexual and correlated health care exposures.

A straightforward reading of this evidence would say that both health care and sexual exposures were important channels for HIV transmission, with the balance depending on whether or not one put more weight on general population or supposed core group studies. However, interpretations of several sorts are inevitable. First, the quality of the evidence is an issue. One option is to assume that the evidence is

Table 4. HIV prevalence, incidence, and risk measures in prostitute women and STD patients

Country, location, year	HIV prevalence in % or incidence per 100 PYs [cases/total]		Selected sexual risk factors	Injections
	Study population	General population		
Prostitutes				
Côte d'Ivoire, Abidjan, 1986[56]	38% [38/101]*	3% [11/331 ANC women]*	Syphilis: 42% prostitutes *vs* 6% ANC women are THPA+	No information
Côte d'Ivoire, Tortiya, 1986[56]	29% [38/131]*	3% [1/34 ANC women]*	Syphilis: 51% prostitutes *vs* 6% ANC women are TPHA+	No information
DRC, Kinshasa, 1985[57]	27% [101/377]	5.9% ANC women[58]	Median number of lifetime partners: 600 for HIV+ *vs* 338 for HIV− PAF for no condom use last yr: < 0% PAF for oral medications last yr: 38%	Reports no association w/injections last yr, but gives no data
DRC, rural, 1986[49]	11% [32/283]	2.2% [3/136 ANC women]		No information
DRC, Kinshasa, 1988[59]	35% [432/1,233]	4.8% [56/1, 160 ANC women in 1990][60]	Mean sex partners/wk: 7.9 for HIV+ *vs* 8.3 for HIV− PAF for regular use of oral antibiotics to prevent STD: 16% PAF for oral contraceptive last 5 yrs: 1% PAF for not regular condom use: 26% PAF for *Haemophilus ducreyi* antibodies: 47%	No information
Kenya, Nairobi, 1981[61]	4% [5/116]	0% [0/111 ANC women]	Mean sex partners/mo: 180 for HIV+ *vs* 54 for HIV− Mean yrs as prostitute: 0.7 for HIV+ *vs* 2.1 for HIV−	No information
Kenya, Nairobi, 1983[61]	82% [32/39]			No information
Kenya, Nairobi, 1984–85[61]	60% [219/362]	2.0% [22/1,100 ANC women in 1985	Mean sex partners/mo: 123 for HIV+ vs 117 for HIV− Mean yrs as prostitute: 2.9 for HIV+ vs 2.6 for HIV−	No information
Kenya, Nairobi, 1985[62]	66% [42/64 low class prostitutes]	2.0% [see above]	Sex acts/yr: 922 for HIV+ *vs* 1,042 for HIV−	95% of HIV+ vs 96% of HIV− women injected last 5 yrs
Kenya, Nairobi, 1985[62]	31% [8/26 high class prostitutes]	2.0% [see above]	Sex acts/yr: 143 for HIV+ *vs* 116 for HIV−	100% of HIV+ and HIV-women injected last 5 yrs
Kenya, Nairobi, 1985[63]	62% [259/418 low class prostitutes]	2.0% [see above]	Duration of prostitution: 35 mos for HIV+ *vs* 50 mos for HIV− Sex partners/day: 3.8 for HIV+ vs 3.6 for HIV− PAF for current oral contraceptive use: 6% PAF for current GU: 12% PAF for RPR/TPHA+: < 0%	No information ('Because this study was not designed to examine the epidemiology of HIV, data . . . were not collected . . .')
Kenya, Nairobi, 1985–87[64]	46 per 100 PYs [83/124]		PAF for oral contraceptive use: 10% PAF for no condom use: 49% Sex partners/day: 4.1 for seroconverters *vs* 3.3 for others GU episodes per year: 1.3 for seroconverters *vs* 0.5 for others	9% report injections or scarification outside research clinic during follow-up; PAF: 0%
Nigeria, Maiduguri, 1988[65]	5% [19/353]			No information
Rwanda, Butare, 1983[66]	74% [34/51]	12% [4/33 blood donor women]		No information
Rwanda, Butare, 1984[66]	88% [29/33]	12% [4/33 blood donor women]		No information
Tanzania, Dar es Salaam, 1986[67]	29% [65/224 women bar workers]	3.6% [7/192 ANC women]	PAF for reported STD last 2 yrs: 7% PAF for current STD on exam: 22%	No information
Uganda, SW roadside, 1986[55]	68% [125/185 barmaids]	11% [11/103 Kampala ANC women]		No information

Continued

Table 4. *Continued*

Country, location, year	HIV prevalence in % or incidence per 100 PYs [cases/total]		Selected sexual risk factors	Injections
	Study population	General population		
STD patients				
Angola, Dundo, 1987–88[68]	24% [48/204]*	18% [ANC women]		No information
Kenya, Nairobi, 1980[61]	0% [0/118]			
Kenya, Nairobi, 1981[61]	3% [2/70]			
Kenya, Nairobi, 1982[61]	6% [4/68]			
Kenya, Nairobi, 1983[61]	14% [13/93]			
Kenya, Nairobi, 1985[61]	15% [29/194]		Sex partners/mo: 1.5 for HIV+; 1.1 for HIV−[†] PAF for prostitute contact: <0%[†]	No information[†]
Kenya, Nairobi, 1985[62]	7.5% [3/40]		MSM: 1 HIV+	Most injected last 5 yrs, but no data
Kenya, Nairobi, 1986[69]	11% [38/340 men w/STD from prostitutes]		PAF for >1 lifetime prostitute contact: 56% PAF for GU last 5 yrs (except current): 52% OR for not circumcised: 2.81	Reports no association with injections last yr, but no data
Kenya, Nairobi, 1986–87?[70]	29 per 100 PYs [24/293 men w/STD from prostitute] [cf: baseline prevalence: 12%]		RR for not circumcised: 8.2	11% report injections (outside research clinic?) during follow-up; PAF: <0%
Rwanda, Butare, 1984[66]	28% [7/25 men w/recent prostitute contact]	7.4% [2/27 blood donors]	PAF for >40 sex partners/yr: 76%	No information
Rwanda, rural, 1985[23]	29% [49/169]	3.4% [7/206 rural adults]	Cf: only 18% [4/22] controls reporting STD in last 2 yrs were HIV+	No information
Tanzania, Dar es Salaam, 1986[67]	10% [48/490]	5.5% [36/650 blood donors]		No information
Uganda, Kampala, 1987[71]	35% [95/270 dermatology and STD patients]	NA	PAF for prostitute contact: 26%	No information
Zambia, Lusaka, 1985[72]	29% [41/139]		Previous attendance at STD clinic: 37% [19/51] HIV+ vs 23% [18/79] for no previous attendance	No information
Zambia, Lusaka, 1985–87[73]	8.1 per 100 PYs [11/73]		OR for another STD during follow-up: 23	No information

PY = person-years; mos = months; yrs = years; ANC = antenatal clinic; STD = sexually transmitted disease; TPHA = *Treponema pallidum* haemagglutination test; RPR = rapid plasma regain test; GU = genital ulcers; PAF = population attributable fraction; OR = odds ratio; RR = risk ratio; NA = not available; MSM = men who have sex with men
*Including HIV-2
[†]Risk information applies to Nairobi STD patients 1982–85

so bad that it cannot be used to prove anything that is not intuitively obvious (ie, that disagrees with one's preconceptions). However, the consistency of much of the evidence—despite some outliers that may reflect poor data or peculiar circumstances—argues against this approach. Second, after accepting the data, there are two major adjustments of crude risk measures that could dramatically impact conclusions about proportions of HIV from health care and sexual transmission: for direction of causation between injections and HIV; and for confound, especially correlations and confound between injections, STD, and prostitute contact.

Direction of causation

Faced with the frequently substantial PAFs associated with medical injections, acceptance by experts of the 1988 consensus that 90% of HIV transmission in Africa was of sexual origin made no sense unless one could attribute most of the association to reverse causation, ie, to people seeking injections for HIV-related symptoms[21]. Two contemporary studies attempted to make that case. A study among hospital workers in Kinshasa in 1984–86 reported no significant association between HIV incidence and injections after setting

aside six seroconverters with HIV-related symptoms. However, the data show a trend, and the PAF of HIV incidence associated with injections, calculated after setting aside six symptomatic seroconverters, is 21% (Table 1)[20].

In the second study, among hospital and urban workers in Rwanda, Van de Perre and colleagues argue that their data, showing a strong association between injections for STD or febrile illness and HIV status, but also showing no association between other injections and HIV, suggest that injections are not a risk—because HIV associated with injections for STD can be explained by subjects acquiring HIV through sexual contact during episodes of STD, while HIV associated with injections for febrile illness can be explained by people with HIV symptoms seeking injections[22]. This analysis is flawed, because injections for different symptoms may be given in different circumstances with greater or lesser risks. Importantly, data from this study show higher HIV prevalence among persons reporting injections for STD compared to persons reporting STD but no injections for STD (28.9 compared to 16.9%); if we assume that all reported STD have equal risks for HIV and equal probability of injection treatment, this difference shows risk from injections for STD without reverse causation (unfortunately, separate data are not available for ulcers, discharge, etc). If so, the PAF for HIV associated with injections for STD—net of reverse causation—is 20% (Table 1)[22].

Confound

During the mid-1980s, Potterat[74], Vachon[11], Wycoff[13], Imperato[14], and others pointed out that associations between HIV and sexual variables could be confounded by medical exposures to treat STD, while others argued that the association between injections and HIV was confounded by sexual transmission during episodes of STD[24]. As above, if we can assume that all reported STD have equal risks for HIV and equal probability of injection treatment, confounding between injections and STD—and *vice versa*—can be resolved by studies comparing HIV prevalence or incidence in (a) persons without STD, (b) persons with STD but without injections for STD, and (c) persons with injections for STD. Subtracting (a) from (b) shows enhanced risk for sexual acquisition of HIV while infected with STD; subtracting (b) from (c) shows risk from HIV associated with injections for STD. In two contemporary studies, data are available for at least partial application of this analysis (Table 1); there is, however, no indication that data were so analysed. In the 1985 Rwanda study discussed in the previous paragraph, the PAF of prevalent HIV associated with STD falls from 33% to 13% after adjusting for injections for STD as a confounding variable, and the adjusted PAF for HIV associated with injections for STD is 20%[22]. In a 1987 study in Uganda, adjustment for all injections as a confound reduces the PAF for reported STD from 11% to 7%; the study was 'unable to show a significant association with STD independent of injections received, although a trend was noted . . .'[27]. In the same study, the crude PAF for all injections of 31% falls to 30% after stratified analysis across persons with and without STD. The authors also noted that 'Injections in Uganda are often not given under aseptic conditions and themselves could theoretically be a vehicle of transmission'.

Most studies of prostitute women in Africa during this period reported HIV prevalence levels that were 5–10 times higher than levels among control women (usually antenatal clinics attendees). During the early 1980s, Nairobi was an exception, with rates in prostitutes more than 30 times higher than in control women. Such data do encourage interpretation of prostitutes and customers as core groups responsible for a large share of transmission, particularly in early epidemics. For example, if 1–2% of women are prostitutes with HIV prevalence 5–20 times higher than other women, they would account for 5–29% of HIV in women. And if each prostitute infected several men each year, one could expect high PAFs among men for prostitute contact. Comparing these calculations to findings from general population studies (Table 1), there is a good fit with one reported high PAF for prostitute contact—71% in Rwanda[24]—but two other PAFs for prostitute contact are considerably lower (8% in Kinshasa[21] and 28% in Zimbabwe[30]). Furthermore, as already reported, 1980s general population studies showed only modest concentration of HIV in adults according to sexual activity. Finally, however important prostitutes and their patrons might be as a core group, questions remained unanswered about the modes of transmission: did they contract and transmit HIV via sexual intercourse or via reuse of injection equipment and multidose vials during STD treatment and antenatal care?[11,75].

Deriving and defending consensus estimates

The post-1988 consensus that ascribed over 90% of adult HIV to heterosexual transmission and an insignificant proportion to unsafe injections was not at the time—or later—supported by calculations from evidence associating HIV with sexual behaviours. Instead, the numerical estimate seems to have been derived by a process of elimination. Most experts accepted available evidence that few African adults with HIV were MSMs or IDUs, so that most infections were assumed to derive from either sexual or health care exposures. The 90% estimate for adult HIV from sexual transmission hence rested on the belief that health care transmission was very low, despite abundant evidence to the contrary. For example, one 1988

review of HIV in Africa stated counterfactually that 'multiple heterosexual partners was the only major risk factor identified during epidemiological studies'[76], and a 1989 review described evidence for health care transmission except by transfusion as 'anecdotal or speculative'[77]. Weak analyses of direction of causation between injections and HIV and of confound between sexual and correlated parenteral risks encouraged discounting of evidence for iatrogenic transmission[20,22]. Experts may have also been lulled by reported low rates of HIV transmission to health care workers through needlestick accidents (through 1989, nine of 10 studies reported 0–0.47% transmission)[78], although injections are deeper than most needlesticks and effectively wash the inside of a syringe and needle into the wound.

Influential epidemiologic reviews published between 1987 and 1990 presented a variety of inferential arguments and hypotheses to support consensus estimates of sexual transmission[2,3,10,79]. Their authors argued, for example, that near-parity in gender distribution of cases was evidence for sexual transmission. Why this should have been viewed as pathognomonic, when such parity was not often observed with truly sexually transmitted diseases such as gonorrhoea, syphilis, and genital herpes, is puzzling. Another inferential argument was that little HIV was found outside sexually active age groups—an argument that did not agree with the evidence. These and other papers presented a number of hypotheses—high rates of partner change, more STD and more efficient HIV transmission in the presence of STD, lack of circumcision, genetic susceptibility to HIV infection, activated or depressed immune system—to explain how sexual transmission could be more important in Africa than in Western Europe. But that was not really the question; to argue that sexual transmission was more important in Africa was not at all the same as arguing that it was responsible for 90% of adult infections; 'more important' could mean 50% of adult infections, or even 20%, for that matter.

Insufficient attention to differential sexual risks

A number of early epidemiological investigations in Africa looked for HIV/AIDS associated with anal intercourse, both men with men and men with women[28,38,62,66,67,69,80]. Many reported only small numbers of infections linked to anal intercourse, which the post-1988 consensus has largely ignored. The importance of these behaviours may have been under-reported[81,82]. Since PAFs for multiple partners and prostitute contact measure HIV risks associated with all sexual contact (including anal intercourse) between men and women, misreporting of anal intercourse would have no impact on PAFs discussed in this paper for heterosexual exposures. However, if the proportion of adult infections from heterosexual transmission is much less than the 90% that has been assumed, any under-reporting of anal sex—which is much more dangerous than vaginal sex—becomes proportionately more important as a component of all sexual transmission, including sexual transmission among men. Hence, this is one issue where the consensus may have overlooked sexual risks.

Why was evidence ignored?

It has been said that people often see what they wish to see. Papers published around 1988 reveal a number of considerations that might have encouraged a mindset prepared to see heterosexual transmission as the driving force in Africa's HIV epidemic. First, it was in the interests of AIDS researchers in developed countries—where HIV seemed stubbornly confined to MSMs, IDUs, and their partners—to present AIDS in Africa as a heterosexual epidemic; 'nothing captured the attention of editors and news directors like the talk of widespread heterosexual transmission of AIDS' (quoted from p. 513[83]). In a prominent 1988 article in *Science*, Piot and colleagues generalize with arguably more public relations savvy than evidence that 'Studies in Africa have demonstrated that HIV-1 is primarily a heterosexually transmitted disease and that the main risk factor for acquisition is the degree of sexual activity with multiple partners, not sexual orientation'[10].

Second, there may have been an inclination to emphasize sexual transmission as an argument for condom promotion, coinciding with pre-existing programmes and efforts to curb Africa's rapid population growth. Third, 'the role of sexual promiscuity in the spread of AIDs in Africa appears to have evolved in part out of prior assumptions about the sexuality of Africans', as Packard and Epstein document in a regrettably ignored 1991 article[84]. Fourth, health professionals in WHO and elsewhere worried that public discussion of HIV risks during health care might lead people to avoid immunizations. A 1990 letter to the *Lancet*, for example, speculated that 'a health message—eg, to avoid contaminated injection materials—will be misunderstood and that immunization programmes will be adversely affected'[85]. In short, tangential, opportunistic, and irrational considerations may have contributed to ignoring and misinterpreting epidemiologic evidence.

Conclusions

Our review of the evidence from 1984–88 suggests several conclusions. First, the post-1988 consensus that sexual transmission is responsible for 90% of adult HIV infections in Africa emerged despite, rather than from, the available evidence. Second, the consensus reflected poor management of research programmes and projects, since key

questions suitable for empirical resolution (eg, direction of causation between injections and HIV, resolution of confound between injections and STD, and why HIV was often correlated with socioeconomic status) were settled with assumptions 'unencumbered by data' (in the words of a colleague).

Third, further delay in reopening and resolving the mid-1980s debate about the relative proportions of African HIV from sexual and health care exposures could have serious consequences for the trajectory of HIV epidemics in many African and Asian countries with nascent or continuing generalized HIV epidemics. Lastly, the emergence of the post-1988 consensus in the face of conflicting evidence serves as a warning to those who would reopen the debate: Now as then, experts may ignore evidence they do not want to see. Nevertheless, the climate for debate may well be different now than in 1988: more are infected; there is less optimism about vaccines; and there is less worry that people are not aware of sex as a risk. In these new circumstances, both epidemiologists and public health managers may be more willing to seek and respect evidence about the proportion of HIV in Africa from medical procedures.

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References

1 Chin J, Sato PA, Mann JM. Projections of HIV infections and AIDS cases to the year 2000. *Bull WHO* 1990;**68**:1–11
2 N'Galy B, Ryder R. Epidemiology of HIV infection in Africa. *J Acquir Immune Defic Syndr* 1988;**1**:551–8
3 Piot P, Laga M, Ryder R, *et al*. The global epidemiology of HIV infection: continuity, heterogeneity, and change. *J Acquir Immune Defic Syndr* 1990;**3**:403–12
4 Taelman H, Dasnoy J, Van March E, Eyckmans L. Syndrome d'immunodéficience acquise chez trois patients du Zaire. *Ann Soc Belge Méd Trop* 1983;**63**:73–4
5 Clumeck N, Sonnet J, Taelman H, *et al*. Acquired immunodeficiency syndrome in African patients. *N Engl J Med* 1984;**310**:492–7
6 Katlama C, Leport C, Matheron S, *et al*. Acquired immune deficiency syndrome in Africans. *Ann Soc Belge Méd Trop* 1984;**64**:379–89
7 Piot P, Quinn TC, Taelman H, *et al*. Acquired immune deficiency syndrome in a heterosexual population in Zaire. *Lancet* 1984;**ii**:65–9
8 Van de Perre P, Rouvroy D, Lepage P, *et al*. Acquired immune deficiency syndrome in Rwanda. *Lancet* 1984;**ii**:62–5
9 World Health Organization (WHO). Acquired immunodeficiency syndrome—an assessment of the present situtation in the world: memorandum from a WHO meeting. *WHO Bull* 1984;**62**:419–32
10 Piot P, Plummer FA, Mhalu FS, Lamboray J-L, Chin J, Mann JM. AIDS: an international perspective. *Science* 1988;**239**:573–9
11 Vachon F, Coulaud JP, Katlama C. Epidémiologie actuelle du syndrome d'immunodéficit acquis en dehors des groupes a risque. *Presse Medicale* 1985;**14**:1949–50
12 Wyatt HV. Injections and AIDS. *Trop Doc* 1986;**16**:97–8
13 Wycoff RF. Female-to-male transmission of AIDS agent [letter]. *Lancet* 1985;**ii**:1017–18
14 Imperato PJ. The epidemiology of the acquired immunodeficiency syndrome in Africa. *New York State J Med* 1986;**86**:118–21
15 Clumeck N, Van de Perre PH, Rouvroy D, Nzaramba D. Heterosexual promiscuity among African patients with AIDS. *N Engl J Med* 1985;**310**:492–7
16 Fleming AF. Seroepidemiology of human immunodeficiency viruses in Africa. *Biomed Pharmacother* 1988;**42**:309–20
17 van der Geest S. The illegal distribution of Western medicines in developing countries: pharmacists, drug pedlars, injection doctors, and others. A bibliographic exploration. *Med Anthropol* 1982;**6**:197–219
18 Lallemant M, Lallemant-Le Coeur S, Cheynier D, *et al*. Characteristics associated with HIV-1 infection in pregnant women in Brazzaville, Congo. *J Acquir Immune Defic Syndr* 1992;**5**:279–85
19 Mann JM, Francis H, Quinn TC, *et al*. HIV seroprevalence among hospital workers in Kinshasa, Zaire. *JAMA* 1986;**256**:3099–102
20 N'Galy B, Ryder RW, Bila K, *et al*. Human immunodeficiency virus infection among employees in an African hospital. *N Engl J Med* 1988;**319**:1123–7
21 Ryder RW, Ndilu M, Hassig SE. Heterosexual transmission of HIV-1 among employees and their spouses at two large businesses in Zaire. *AIDS* 1990;**4**:725–32
22 Van de Perre P, Carael M, Nzaramba D, Zissis G, Kayihigi J, Butzler JP. Risk factors for HIV seropositivity in selected urban-based Rwandese adults. *AIDS* 1987;**1**:207–11
23 Van de Perre P, Le Polain B, Carael M, Nzaramba D, Zissis G, Butzler J-P. HIV antibodies in a remote area in Rwanda, central Africa: an analysis of potential risk factors for HIV seropositivity. *AIDS* 1987;**1**:213–15
24 Carael M, Van de Perre PH, Lepage PH, *et al*. Human immunodeficiency virus transmission among heterosexual couples in Central Africa. *AIDS* 1988;**2**:201–5
25 Allen S, Lindan C, Serufilira A, *et al*. Human immunodeficiency virus infection in urban Rwanda. *JAMA* 1991;**266**:1657–63
26 Killewo J, Nzamuryekunge K, Sandstrom A, *et al*. Prevalence of HIV-1 infection in the Kagera region of Tanzania: a population based study. *AIDS* 1990;**4**:1081–5
27 Konde-Lule J, Berkeley SF, Downing R. Knowledge, attitudes and practices concerning AIDS in Ugandans. *AIDS* 1989;**3**:513–18
28 Hira SK, Kamanga J, Bhat GJ, *et al*. Perinatal transmission of HIV-1 in Zambia. *BMJ* 1989;**299**:1250–2
29 Hira SK, Magrola UG, Mwale C, *et al*. Apparent vertical transmission of human immunodeficiency virus type 1 by breast-feeding in Zambia. *J Pediatr* 1990;**117**:421–4
30 Bassett MT, Latif AS, Katzenstein DA, Emmanuel JC. Sexual behavior and risk factors for HIV infection in a group of male factory workers who donated blood in Harare, Zimbabwe. *J Acquir Immune Defic Syndr* 1992;**5**:556–9
31 Kayembe K, Mann JM, Francis H, *et al*. Prévalence des anticorps anti-HIV chez les patients non atteints de SIDA ou de syndrome associé au SIDA a Kinshasa, Zaire. *Ann Soc Belge Méd Trop* 1986;**66**:343–8
32 Mann JM, Francis H, Davachi F, *et al*. Human immunodeficiency virus seroprevalence in pediatric patients 2 to 14 years of age at Mama Yemo Hospital, Kinshasa, Zaire. *Pediatrics* 1986;**78**:673–7

33 Mann JM, Francis H, Davachi F, *et al*. Risk factors for human immunodeficiency virus seropositivity among children 1–24 months old in Kinshasa, Zaire. *Lancet* 1986;**ii**:654–7

34 Nguyen-Dinh P, Greenberg AE, Mann JM, *et al*. Absence of association between *Plasmodium falciparum* malaria and human immunodeficiency virus infection in children in Kinshasa, Zaire. *Bull WHO* 1987;**65**:607–13

35 Shaffer N, Hedberg K, Davachi F, *et al*. Trends and risk factors for HIV-1 seropositivity among outpatient children, Kinshasa, Zaire. *AIDS* 1990;**4**:1231–6

36 Lepage P, Van de Perre P, Carael M, Butzler JP. Are medical infections a risk factor for HIV infection in children? *Lancet* 1986;**ii**:1103–4

37 Lepage P, Van de Perre P. Nosocomial transmission of HIV in Africa: what tribute is paid to contaminated transfusions and medical injections. *Infect Control Hosp Epidemiol* 1988; **9**:200–3

38 Berkley SF, Widy-Wirski R, Okware SI, *et al*. Risk factors associated with HIV infection in Uganda. *J Infect Dis* 1989; **160**:22–33

39 Hudson CP, Hennis AJM, Kataaha P, *et al*. Risk factors for the spread of AIDS in rural Africa: evidence from a comparative seroepidemiological survey of AIDS, hepatitis B, and syphilis in southwestern Uganda. *AIDS* 1988;**2**: 255–60

40 Kebede Y, Pickering J, McDonald JC, Wotton K, Zewde D. HIV infection in an Ethiopian prison. *Am J Public Health* 1991;**81**:625–7

41 Carswell JW, Lloyd G, Howells J. Prevalence of HIV-1 in east African lorry drivers. *AIDS* 1989;**3**:759–61

42 McCarthy MC, Hyams KC, El-Hag AE-L, *et al*. HIV-1 and hepatitis B transmissions in Sudan. *AIDS* 1989;**3**:725–9

43 Vincent-Ballereau F, Lafaix C, Haroche G. Incidence of intramuscular injections in rural dispensaries in developing countries. *Trans Roy Soc Med Hygiene* 1989;**83**:106

44 Potterat JJ. ''Socio-geographic space'' and sexually transmissible diseases in the 1990s. *Today's Life Science* 1992;**4**: 16–22,31

45 Thomas JC, Tucker MJ. The development and use of the concept of a sexually transmitted disease core. *J Infect Dis* 1996;**174**(Suppl 2):S134–43

46 Rwandan HIV Seroprevalence Study Group. Nationwide comunity-based serological survey of HIV-1 and other human retrovirus infections in a central African country. *Lancet* 1989;**i**:941–3

47 Allen S, Tice J, Van de Perre P, *et al*. Effect of serotesting with counselling on condom use and seroconversion among HIV discordant couples in Africa. *BMJ* 1992;**304**:1605–9

48 Aral SO, Holmes KK. Social and behavioral determinants of the epidemiology of STDs: industrialized and developing countries. In: Holmes KK, Sparling PF, Mardh P-A, *et al*. *Sexually transmitted diseases*. 3rd edn. New York: McGraw-Hill, 1999. p. 1381–90

49 Nzilambi N, De Cock KM, Forthal DN, *et al*. The prevalence of infection with human immunodeficiency virus over a 10-year period in rural Zaire. *N Engl J Med* 1988;**318**:276–9

50 Quinn TC, Mann JM, Curran JW, Piot P. AIDS in Africa: an epidemiologic paradigm. *Science* 1986;**234**:955–63

51 Central Intelligence Agency. Democratic Republic of the Congo. The World Fact Book 2002. Available from : URL: http://odci/gov/cia/publication/factbook/geos/cg.html.

52 Central Intelligence Agency. Burundi. The World Fact Book 2002. Available from http://odci/gov/cia/publication/factbook/geos/bu.html.

53 Outtara SA, Meite M, Cot MC, de-The G. Compared prevalence of infections by HIV-1 and HIV-2 during a 2-year period in suburban and rural areas of Ivory Coast. *J Acquir Immune Defic Syndr* 1989;**2**:94–9

54 Merlin M, Josse R, Trebucq A, Mouanda V, Kouka-Bemba D. Surveillance epidémiologique du syndrome d'immunodépression acquise dans six états d'Afrique centrale. *Med Trop* 1988;**48**:381–9

55 Carswell JW. HIV infection in healthy persons in Uganda. *AIDS* 1987;**1**:223–7

56 Denis F, Barin F, Gershy-Damet G, *et al*. Prevalence of human T-lymphotropic retroviruses type III (HIV) and type IV in Ivory Coast. *Lancet* 1987;**i**:408–11

57 Mann JM, Nzilambi N, Piot P, *et al*. HIV infection and associated risk factors in prostitute women in Kinshasa, Zaire. *AIDS* 1988;**2**:249–54

58 UNAIDS. Democratic Republic of the Congo. Epidemiological Fact Sheet, 2000 update. WHO: UNAIDS, 2000

59 Nzilambi N, Laga M, Thiam MA, *et al*. HIV and other sexually transmitted diseases among prostitute women in Kinshasa. *AIDS* 1991;**5**:715–21

60 Vuylsteke B, Laga M, Alary M, *et al*. Clinical algorithm for the screening of women for gonococcal and chlamydial infection: evaluation of pregnant women and prostitutes in Zaire. *Clin Infect Dis* 1993;**17**:82–8

61 Piot P, Plummer FA, Rey M-A, *et al*. Retrospective seroepidemiology of AIDS virus infection in Nairobi populations. *J Infect Dis* 1987;**155**:1108–12

62 Kreiss JK, Koech D, Plummer FA, *et al*. AIDS virus infection in Nairobi prostitutes. *N Engl Med* 1986;**314**:414–17

63 Simonsen JN, Plummer FA, Ngugi EN, *et al*. HIV infections among lower socio-economic strata prostitutes in Nairobi. *AIDS* 1990;**4**:139–44

64 Plummer FA, Simonsen JN, Cameron DW, *et al*. Cofactors in male–female sexual transmission of human immunodeficiency virus type 1. *J Infect Dis* 1991;**163**:233–9

65 Chikwem JO, Mohammed I, Ola T. Human immunodeficiency virus type 1 (HIV-1) infection among female prostitutes in Borno State of Nigeria: one year follow-up. *E Afr Med J* 1989;**66**:752–6

66 Van de Perre P, Clumeck N, Carael M, *et al*. Female prostitutes: a risk group for infection with human T-cell lymphotrophic virus type III. *Lancet* 1985;**ii**:524–7

67 Mhalu F, Bredberg-Raden U, Mbena E, *et al*. Prevalence of HIV infection in healthy subjects and groups of patients in Tanzania. *AIDS* 1987;**1**:217–21

68 Santos-Ferreira MO, Cohen T, Lourenco MH, Almeida MJM, Chamaret S, Montagnier L. A study of seroprevalence of HIV-1 and HIV-2 in six provinces of People's Republic of Angola: clues to the spread of HIV infection. *J Acquir Immune Defic Syndr* 1990;**3**:780–6

69 Simonsen JN, Cameron DW, Gakinya MN, *et al*. Human immunodeficiency virus infection among men with sexually transmitted diseases. *N Engl J Med* 1988;**319**:274–8

70 Cameron DW, Simonsen JN, D'Costa LJ, *et al*. Female to male transmission of human immunodeficiency virus type 1: risk factors for seroconversion in men. *Lancet* 1989;**ii**: 403–7

71 Nsubuga P, Mugerwa R, Nsibambi J, Sewankambo N, Katabira E, Berkley S. The association of genital ulcer disease and HIV infection at a dermatology–STD clinic in Uganda. *J Acquir Immune Defic Syndr* 1990;**3**:1002–5

72 Melbye M, Njelesani EK, Bayley A, *et al*. Evidence for heterosexual transmission and clinical manifestations of human immunodeficiency virus infection and related conditions in Lusaka, Zambia. *Lancet* 1986;**ii**:1113–15

73 Hira SK, Kamanga J, Macaucua R, Mwansa N, Cruess DF, Perine PL. Genital ulcers and male circumcision as risk factors for acquiring HIV-1 in Zambia. *J Infect Dis* 1990; **161**:584–5

74 Potterat JJ. The AIDS epidemic and media coverage: a critical review. *Critique* 1987;**26**:36–8

75 Minkin SF. Iatrogenic AIDS: unsafe medical practices and the HIV epidemic. *Soc Sci Med* 1991;**33**:786–90
76 Ndinya-Achola J, Plummer FA, Simonsen JN, Cameron DW, Ngungi EN, Pamba H. A review of HIV-1 in Africa. *Bull N Y Acad Med* 1988;**64**:480–90
77 Pela AO, Platt JJ. AIDS in Africa: emerging trends. *Soc Sci Med* 1989;**28**:1–8
78 Marcus R, Kay K, Mann JM. Transmission of human immunodeficiency virus (HIV) in health-care settings worldwide. *Bull WHO* 1989;**65**:577–82
79 Piot P, Mann M. Bidirectional heterosexual transmission of human immunodeficiency virus (HIV). *Ann Inst Pasteur/ Virol* 1987;**138**:125–32
80 Serwadda D, Mugerwa RD, Sewankambo NK, *et al*. Slim disease: a new disease in Uganda and its assocation with HTLV-III infection. *Lancet* 1985;**ii**:849–52
81 Fumento M. *The myth of heterosexual AIDS*. Washington DC: Regnery Gateway, 1990
82 Brody S. *Sex at risk: Lifetime number of partners, frequency of intercourse, and the low AIDS risk of vaginal intercourse*. New Brunswick: Transaction Publishers, 1997
83 Shilts R. *And the band played on: politics, people, and the AIDS epidemic*. New York: St Martin's Press, 2000
84 Packard RM, Epstein P. Epidemiologists, social scientists, and the structure of medical research on AIDS in Africa. *Soc Sci Med* 1991;**33**:771–83
85 Berkley S, Weeks M, Barenzi J. Immunisation and fear of AIDS [letter]. *Lancet* 1990;**335**:47–8

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