Impact of long-term civil disorders and wars on the trajectory of HIV epidemics in sub-Saharan Africa

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ABSTRACT

From the mid-1970s, seven countries in sub-Saharan Africa have experienced civil disorders and wars lasting for at least 10 years. In two — Sierra Leone during 1991-2002, and Somalia from 1988 and continuing — adult HIV prevalence remained below 1%. In the Democratic Republic of the Congo, HIV prevalence appears to have stabilised during post-1991 civil disorder and war. Limited information from Angola (civil war 1975 -2002) and Liberia (civil disorder and war from 1989 and continuing) suggests low HIV prevalence. Mozambique's HIV prevalence was near 1% after its 1975 - 1992 civil war, but increased dramatically in the first post-war decade. Across African countries with long-term wars, HIV seems to have spread more slowly than in most neighbouring countries at peace. This evidence contributes to the ongoing debate about the factors that explain differential epidemic trajectories, a debate which is crucial to the design of HIV prevention programmes. One possible explanation for slow epidemic growth in wartime is that unsterile health care accounts for an important proportion of HIV transmission during peacetime, but much less when wars disrupt health services. However, other explanations are also possible. The roles of sex and blood exposures in HIV epidemics in war and peace await empirical determination.

Keywords: HIV, epidemiology, Africa, wars, risk factors.

RÉSUMÉ

A partir du milieu des années 1970, sept pays de l'Afrique sous-Sahara ont connu des désordres civils et des guerres qui ont duré au moins 10 ans. Dans deux pays — la Sierra Léone entre 1991 et 2002 et la Somalie depuis 1988 jusque-là — la prédominance du VIH chez des adultes est restée sous 1%. Dans la République Démocratique du Congo, la prédominance du VIH s'est stabilisée après le désordre civil et la guerre de 1991. L'information très limitée de l'Angola (guerre civile 1975 - 2002) et de Libéria (désordre et guerre civils depuis 1989) suggère une prédominance du VIH très basse. Au Mozambique, la prédominance du VIH était d'environ 1% après la guerre civile de 1975 à 1992. Cependant, il y a eu une augmentation importante au cours de la première décennie après la guerre. Dans tous les pays africains qui ont subit des guerres à longue durée, le VIH semble se propager plus doucement que dans des pays voisins qui sont en période de paix. Cette preuve contribue au débat actuel portant sur des facteurs responsables aux trajectoires différentielles de l'épidémie. Ce débat est important à la conception des programmes de prévention. Une explication possible de la dissémination lente de l'épidémie durant la guerre est que les soins non-stériles expliquent la proportion importante de transmission de VIH pendant la période de paix. Ce phénomène est encore plus réduit quand les services de santé sont interrompus par la guerre. Néanmoins, il y a d'autres explications possibles. Les rôles que jouent le sexe et la contamination par le sang sur l'épidémie du VIH pendant la guerre et durant la période de paix attendent une détermination empirique.

Mots clés: le VIH, I'épidémiologie, I'Afrique, les guerres, les facteurs de risque.

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Introduction

During the past several decades, civil disorders and wars have afflicted many countries in sub-Saharan Africa, and at the same time the region's AIDS epidemics have emerged as a major health crisis. Although most early analyses of Africa's AIDS epidemics ignored wars, some experts speculated that wars accelerated HIV transmission. For example, in one of the first AIDS reports from Uganda, Serwadda, Sewankambo, Carswell, Bayley, Tedder, Weiss *et al.* (1985, p. 852) proposed that 'movements of the Tanzanian army in 1980' might have propagated HIV in Uganda. In the mid-1990s, the World Health Organisation (WHO), United Nations High Commission for Refugees (UNHCR) and UNAIDS (1996, p. 6) generalised that 'HIV spreads fastest in conditions of poverty, powerlessness, and social instability - conditions that are often at their most extreme during emergencies'.

However, during the 1990s AIDS epidemic researchers recognised evidence that did not fit these views. Tarantola and Schwartlander (1997) noted that Mozambique, a country that had suffered a long-term civil war, had lower HIV prevalence than neighbouring countries. Nevertheless, they considered that available information did not demonstrate a causal relationship. Writing several years later, Melo, Beby-Defaux, Faria, Guiraud, Folgosa, Barreto *et al.* (2000, p. 203) hypothesised that 'The isolation of Mozambique during years of the civil war from 1976 to 1992 probably limited the spread of HIV epidemic'. Salama and Dondero (2001, p. S6) discuss migration and other social disruptions 'that may increase HIV transmission in complex emergencies,' but the data they present show relatively low HIV prevalence in many African countries 'affected by major complex emergencies' in the 1990s.

Evidence that Africa's long-term civil disorders and wars are associated with relatively slow epidemic expansion has accumulated in recent years. This paper summarises the evidence and discusses implications for understanding and preventing generalised HIV epidemics. This evidence contributes to an ongoing debate about the factors that explain differential HIV epidemic trajectories (Boerma, Gregson, Nyamukapa & Urassa, 2003; Buve, Caraël, Hayes, Auvert, Ferry, Robinson *et al.*, 2001; Garnett & Fraser, 2003; Gisselquist, Potterat & Brody, 2004). One unresolved issue in this debate — an issue crucial to the design of HIV prevention programmes — is the contribution of blood exposures during health care to generalised HIV epidemics. One possible explanation for relatively slow epidemic growth during civil wars is that HIV transmission through unsterile health care accounts for an important proportion of HIV transmission during peacetime, but much less when wars disrupt health care delivery. However, other explanations are also possible. Outstanding questions about the role of health care in Africa's HIV epidemics await conclusive empirical resolution.

Methods

During the last several decades, seven countries in sub-Saharan Africa have experienced civil disorders and wars lasting for at least 10 years and interrupting government functions over large areas of the affected countries (Peace Pledge Union Online, 2004a; see below for country-specific references). Moving counterclockwise from west Africa, these seven are Sierra Leone (civil war 1991 - 2002), Liberia (civil war from 1989 and continuing), Democratic Republic of the Congo (DRC; collapse of government functions followed by civil war from 1991 and continuing), Angola (civil war 1976 - 2002), Mozambique (civil war 1975 - 1992), Somalia (civil war from 1988 and continuing), and southern Sudan (civil war from 1983 and continuing). Africans have fought many other shorter or more localised wars in recent decades; however, data on HIV prevalence are generally too weak to convincingly demonstrate an impact on epidemic trends during these more limited events.

For each of the long-term wars, I report evidence on HIV prevalence (including HIV-1 and HIV-2 infections) in adults (usually persons aged 15 - 49 years) before, during, and at the end of the war, or the latest data in continuing wars, and the post-war epidemic trajectory (relevant for Mozambique only). For comparison, I report levels and movements of HIV prevalence in neighbouring countries over time. Long-term trends in HIV prevalence, as considered in this paper, imply matching long-term trends in HIV incidence (although short-term fluctuations in incidence may not be reflected in long-term trends in prevalence).

A major challenge in this study has been to choose the best data to demonstrate levels and movements in HIV prevalence. This is a problem not only in

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countries at war, where data are scarce, but also in neighbouring countries that are at peace. UNAIDS' estimates of country HIV prevalence, which are calculated from HIV prevalence in selected antenatal clinics, have serious problems for this study. Firstly, they are not available before 1994, whereas all wars considered in this paper began no later than 1991. Secondly, they are not comparable across years: country estimates may show large year-to-year fluctuations based on statistical adjustments rather than epidemic realities (e.g. UNAIDS estimated 2.9 million HIV infected Ethiopians in 2000 [UNAIDS, 2000a], but only 1.9 million in 2001 [UNAIDS, 2002a]). And finally, UNAIDS' estimates have an apparent upward bias: comparable estimates of HIV prevalence are available for six African countries from random sample national surveys; UNAIDS' estimates are 0 – 177% (mean of 64%) higher than these survey-based estimates (Table 1).

To avoid overestimating differences between HIV prevalence in countries at war and neighbours at peace, for comparison countries at peace I choose data that are not transparently biased toward the high end. When available, I report adult HIV prevalence from random sample national surveys (Table 2). In most cases, the best available information comes from sentinel surveys in selected antenatal clinics. Since urban clinics tend to report relatively high rates of HIV prevalence that are unrepresentative for the nation, for comparison countries I report medians of

HIV prevalence among all antenatal clinics in the country, or among antenatal clinics 'outside major urban areas'. In exceptional cases, I use other data to characterise HIV prevalence in countries at peace (see notes to Table 2).

For the seven countries with long-term civil disorders or wars, I report data as available from studies before, during, and in several cases after the war. In some cases, I report data from urban centres; since urban data may overestimate HIV prevalence in the country, use of these data tends to underestimate differences between countries at war and their more peaceful neighbours. For six of seven civil wars, I examine the impact on HIV prevalence for the country as a whole; for Sudan, I consider HIV prevalence for the south only, since the impact of the war has been highly concentrated in that region.

Results: trajectory of HIV epidemics during long-term civil wars

Sierra Leone
Sierra Leone entered a period of brutal civil war in 1991, when Revolutionary United Front (RUF) rebels entered from Liberia (United Nations, 2004). During the ensuing decade, the RUF terrorised much of the rural and, at times, urban population while living off profits from diamond mines (Campbell, 2004). The government, even with military assistance from the United Nations (UN) and United Kingdom, was unable to ensure security. Government services were

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TABLE 1. COMPARING ESTIMATES OF ADULT HIV PREVALENCE IN AFRICAN COUNTRIES FROM UNAIDS V. RANDOM SAMPLE NATIONAL SURVEYS

Country	UNAIDS' estimate		National survey estimate		UNAIDS' estimate as % of national survey estimate
	Year	Prevalence (%)	Year	Prevalence (%)	
Burundi	2001	8.3	2002	3.0*	277
Kenya	2001	15.0	2003	6.7	223
Mali	2001	1.7	2001	1.7	100
Rwanda	1997	12.8	1997	11.1	115
South Africa	2001	20.1	2002	15.6	129
Zambia	2001	21.5	2001/2	15.4*	140
Zimbabwe	2001	33.7	2001	16.1*†	NA
Mean					164

NA: not applicable.
* Averages of reported HIV prevalence for men and women.
† HIV prevalence for persons aged 15 - 29 years.
Sources: Kenya, Central Bureau of Statistics, 2003; Shisana & Simbayi, 2002; UNAIDS, 2002a; UNAIDS, 1988; WHO/AFRO, 2003; WHO, 2003.

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Table 2. HIV PREVALENCE IN COUNTRIES WITH LONG-TERM CIVIL DISORDER AND SELECTED NEIGHBOURING COUNTRIES

Region, country	Population (millions)	% adult literacy	1985-86	1987-88	1989-90	1991-92	1993-94	1995-96	1997-98	1999-00	2001-02	2003-04
West												
Guinea	8.2	36			OW: 0.7		OW: 1.4				NW: 2.8	
Sierra Leone	4.6	31				UW: 0.8		UW: 2.0			NA: 0.9	
Liberia	3.1	55		NA 0.0		UW: 3.7	UW: 4.0				NW: 4.3	
Côte d'Ivoire	16.1	50		OW: 2.6				OW: 9.8	OW: 8.8		NW: 7.3	
Central/Southwest												
CAR	3.8	48			OW: 6.5	OW: 5	OW: 10	OW: 14			NW: 15	
DRC	59.8	63	UW: 6.0	UW: 7.1	UW: 4.7	UW: 5.9	UW: 5.4	UW: 3.2	UW: 3	UW: 4.1	UW: 3.0	
Rwanda	8.1	68	NA: 3			OW: 2.5		OW: 8.8	NA: 11		NW: 4.2	
Angola	12.8	43	UW: 0.3			UW: 0.7		UW: 1.2			NW: 2.4	
Zambia	10.6	79			OW: 17	OW: 10	OW: 14		OW: 13		NA: 15	
Namibia	1.9	83				OW: 3.4	OW: 8.2	OW: 17	OW: 15	OW: 17	NW: 23	
Southeast												
South Africa	44.4	86				NW: 0.8	NW: 2.7	NW: 7.6	NW: 14	NW: 23	NW: 22	NA: 16
Zimbabwe	12.8	89				OW: 16	OW: 19	OW: 24	OW: 31	OW: 29	OW: 33	NW: 30
Mozambique	18.2	45			UW: 0.4	UW: 0.6	UW: 1.2	UW: 2.7	UW: 5.8	UW: 11	OW: 11	NW: 14
Malawi	11.6	61		UW: 8.2	UW: 17	OW: 5.5	OW: 16	OW: 16	OW: 18	OW: 22	NW: 17	
Northeast												
Kenya	31.1	83			OW: 3.2	OW: 7.9	OW: 8.9	OW: 11	OW: 14	OW: 23	NW: 6.5	NA: 6.7
Somalia	9.1	24							OW: 2.0	OW: 0.7		
Ethiopia	67.3	40					OW: 4.9	OW: 4.3	OW: 9.2		NW: 11	
Southern Sudan	11.1	nd						UW: 3.0	UW: 3.5			RA: <1
Uganda	24.2	68			OW: 11	OW: 13	OW: 8.7	OW: 8.4	OW: 9.3	OW: 5.2	NW: 5.6	

nd: no data; DRC: Democratic Republic of the Congo, CAR: Central African Republic; NA, RA: national or rural adult HIV prevalence; UW, OW, NW: HIV prevalence in antenatal women in urban areas, outside major urban areas, or nationally (medians among antenatal clinics, omitting medians based on one clinic only; where medians are available for 2 years, the Table 2 shows the one based on the most clinics; exceptions are described in the following notes on sources by country).

Sources: Population and adult literacy (15 years and older) in 2001 are from United Nations Development Programme (2003) except for population in southern Sudan (which is estimated as 34% of Sudan's 2001 population [Prunier & Gisselquist, 2003]), and adult literacy for Guinea, Sierra Leone, and Angola in 1995 (from UNAIDS, 2000a).

Sources for HIV prevalence in each country:

Guinea: medians from five and six rural clinics in 1990 and 1996, respectively (UNAIDS, 2000f); 2001 survey of antenatal women (USAID, 2004).

Sierra Leone: 1990 and 1992 from one urban clinic (UNAIDS, 2000i); 2002 from a national survey of youth and adults aged 12-49 years (Kaiser et al., no date).

Liberia: 1987 from multiple studies of healthy individuals and blood donors (US Census Bureau, 2003); 1992-93 from one urban clinic (UNAIDS, 2000g); 2002 from national antenatal women (sampling and weighting not clear)(WHO/AFRO, 2003).

Côte d'Ivoire: 1987 - 2000 medians from 4 - 9 clinics outside major urban areas (UNAIDS, 2002b); 2002 national median of 28 clinics (WHO/AFRO, 2003).

CAR: 1990 - 96 medians from 3 - 10 clinics outside major urban areas (UNAIDS, 2000c); 2002 national median of 48 clinics (WHO/AFRO, 2003).

DRC: 1986 - 99 medians from 2 - 6 urban clinics (UNAIDS, 2000d); 2002 antenatal women in Kinshasa (WHO/AFRO, 2003).

Rwanda: 1986 and 1997 adult HIV prevalence from national surveys (Bizimungu, Ntilivamunda, Tshimana, Nzaramba, Bugingo, Ndikuyeze et al. 1989; WHO/AFRO, 2003); 1991 and 1996 medians from 6-10 clinics outside major urban areas (UNAIDS, 2000h); 2002 national median of 24 clinics (WHO/AFRO, 2003).

Angola: 1986 - 95 from one clinic in Luanda (UNAIDS, 2000b); 2002 median of clinics from 6 provinces (WHO/AFRO, 2003).

Zambia: 1990 - 98 medians of 4 - 22 clinics outside major urban areas (UNAIDS, 2002k); 2001/2 from a national Demographic and Health Survey of adults aged 15-49 years (WHO, 2003).

Namibia: 1992 - 2000 medians of 5 - 16 clinics outside major urban areas (UNAIDS, 2002g); 2002 national median of 21 clinics (WHO/AFRO, 2003).

South Africa: 1990 - 99 estimates for national antenatal women (Laurence, 2000); 2002 for adults 15 - 49 years from a national survey (Shisana & Simbayi, 2002).

Zimbabwe: 1990 - 2000 medians of 3-30 clinics outside major urban areas (Table 2 shows reported median prevalence of 19% for 1991 rather than 0% for 1992)(UNAIDS, 2002l); 2001 national median of 19 clinics (WHO/AFRO, 2003).

Mozambique: 1986 - 98 from Maputo clinics, and 2000 median of 18 clinics outside major urban areas (UNAIDS, 2002f); 2002 national median of 37 clinics (WHO/AFRO, 2003).

Malawi: 1987 - 89 medians of 2 urban clinics, and 1992-99 medians of 10 - 16 clinics outside major urban areas (UNAIDS, 2002e); 2001 national median of 19 clinics (WHO/AFRO, 2003).

Kenya: 1990 - 2000 medians from 11 - 25 clinics outside major urban areas (UNAIDS, 2002d); 2002 national median of 34 clinics (WHO/AFRO, 2003); 2003 from a national Demographic and Health Survey of adults 15 - 49 years (Kenya, Central Bureau of Statistics, 2003).

Somalia: 1997 - 99 medians of 2 - 3 clinics outside major urban areas (UNAIDS, 2002h).

Ethiopia: 1991 - 98 medians from 2 - 10 clinics outside major urban areas (UNAIDS, 2000e); 2001 national median of 34 clinics (WHO/AFRO, 2003). *Southern Sudan:* 1995 and 1998 from an antenatal clinic in Juba, a city in southern Sudan (UNAIDS, 2002i); 2003 from 2002-03 surveys in a rural area near Yei town and in Rumbek, a small southern town (Kaiser et al. 2004).

Uganda: 1990 - 2000 medians of 6 - 23 clinics outside major urban areas (UNAIDS, 2002j); 2001 national median of 17 clinics (WHO/AFRO, 2003).

minimal to non-existent. After an agreement with the rebels, the UN declared in January 2002 that the war was over (Peace Pledge Union Online, 2004b). In a national survey shortly after the end of the war, 0.6% of men and women aged 12 - 49 years reported a war-related amputated limb, 9.4% of youth aged 12 - 17 years reported one or both parents killed or missing, and 6% of women aged 12 - 49 years reported forced sex with someone other than their regular partner in the last 12 months (Kaiser, Spiegel, Salama, Brady, Bell, Bond et al., no date). The country is extremely poor; according to recent estimates, only 45% of adult men and 18% of adult women are literate, and life expectancy is below 40 years (UNAIDS, 2000a).

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At the beginning of Sierra Leone's civil war, HIV prevalence was low in the general population; for example, 0.8% among urban antenatal women and 0.4% among army recruits in 1990 (United States [US] Census Bureau, 2000). However, higher prevalence was found in some groups, e.g. 6.7% in Freetown blood donors in 1987 - 89 (US Census Bureau, 2000), and 4.7% in sexually transmitted disease (STD) patients in 1990 (Kosia, Makiu, Mansaray & Koker, 1992). During the war, UNAIDS (2002a) estimated 7.0% adult HIV prevalence in 2001, which was a significant over-estimate. Within months after the war ended in early 2002, a national serosurvey assisted by the US Centers for Disease Control and Prevention (CDC) found only 0.9% HIV prevalence in youth and adults aged 12 - 49 years, which was a weighted average of 0.6% HIV prevalence in rural areas and 2.1% in Freetown. The survey covered an estimated 79% of the population (Kaiser et al., no date).

Sierra Leone is bounded by Guinea and Liberia. The best recent estimate of HIV prevalence in Guinea is 2.8% from a national survey of antenatal women in 2001 (WHO, Regional Office for Africa [WHO/AFRO], 2003), which is triple the HIV prevalence in Sierra Leone. Information on HIV in Liberia is too limited to make any sound comparisons with the situation in Sierra Leone (see below).

Liberia
Liberia's civil war began in 1989 when Charles Taylor's National Patriotic Front of Liberia entered from Côte d'Ivoire. Taylor seized power in 1990, but for years competing armed factions controlled parts of the country. Despite a peace agreement and elections in 1997, fighting continued. In 2003, as part of another peace agreement, Taylor resigned as President and moved to Nigeria, but 'fighting and marauding ...has continued' (International Crisis Group, 2004, p. 1). In early 2004, the UN Mission in Liberia increased troop levels, with plans to demobilise and disarm warring factions (Europaworld, 2004).

HIV was present in Liberia at low levels at the beginning of the civil war. Studies in 1987 - 89 reported 1 - 4% HIV prevalence in tuberculosis patients, 0 - 0.6% in blood donors, and 0 - 0.7% in visa applicants (US Census Bureau, 2003). Other studies in 1988 reported no HIV in 30 prostitutes, 35 STD patients, or 941 healthy individuals (US Census

Bureau, 2003). From 1995, rates of HIV prevalence in multiple studies of inpatients, outpatients, and tuberculosis patients range upward from 11.3% (US Census Bureau, 2003), pointing to an expanding pool of infected people in the non-patient population. UNAIDS (2000a) estimated 2.8% adult HIV prevalence nationally in 2000, but the basis for that estimate is unclear. WHO/AFRO (2003) reported 4.3% HIV prevalence for women attending antenatal clinics throughout Liberia in 2002. However, it is not clear how representative this is for all women.

Liberia shares borders with Sierra Leone and Côte d'Ivoire. As already stated, a national survey in 2002 reported 0.9% HIV prevalence in Sierra Leone (Kaiser, Spiegel et al., no date). Côte d'Ivoire has consistently reported the highest levels of HIV prevalence in the region: in 2002, the median HIV prevalence in 28 sentinel antenatal clinics was 7.3% (WHO/AFRO, 2003).

DRC
As President Mobutu aged and aid to the DRC faltered at the end of the cold war, government mismanagement led in the early 1990s to a collapse of services and cessation of many aid projects. In September 1991, soldiers protesting low pay rioted in Kinshasa; as others joined, rioting and looting spread to other cities. French and Belgian soldiers entered Kinshasa to protect and evacuate expatriates; foreign staff of Project SIDA, a major US and Belgian-funded AIDS research project, left Kinshasa at this time, and the project soon closed (Cohen, 1997). In 1993, soldiers protesting low pay instigated a second episode of urban riots. Civil disorder gave way to civil war in 1996 as Laurent Kabila, with assistance from Uganda and Rwanda, drove from eastern DRC to overthrow Mobutu in 1997. Civil war resumed in 1998 with the additional involvement of troops from Angola, Zimbabwe, Namibia and Sudan. Since Joseph Kabila replaced his assassinated father in 2001, fighting has subsided (Peace Pledge Union Online, 2004c), but as of early 2004, civil order and government services have yet to be restored in much of the country.

AIDS is an old epidemic in the DRC. Sequencing of HIV collected in 1997 found many samples that did not cluster with known clades (Vidal, Peeters, Mulanga-Kabeya, Nzilambi, Robertson, Ilunga et al., 2000), indicating that HIV was spreading in the DRC not long after the most recent common ancestor,

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which has been dated to 1931 (95% confidence interval: 1915 – 1941)(Korber, Muldoon, Theiler, Gao, Gupta, Lapedes *et al.*, 2000). The oldest known HIV was isolated from a blood sample collected in or near Kinshasa in 1959 (Nahmias, Weiss, Yao, Lee, Kodsi, Schanfield *et al.*, 1986). Blood collected in rural Equateur Province (northwest DRC) in 1976 and 1986 showed a stable 0.8% HIV prevalence over a decade (Nzilambi, De Cock, Forthal, Francis, Ryder, Malebe *et al.*, 1988). In the early 1980s, the DRC was one of the first countries in Africa in which AIDS was recognised.

During 1984 – 91, multiple studies found HIV prevalence around 5% in low-risk adults in Kinshasa. A 1984 survey of health workers found 8.1% of women and 5.2% of men with HIV infection (Mann, Francis, Quinn, Bila, Asila, Bosenge *et al.*, 1986). In 1987 – 88, HIV prevalence among men working at a bank and their wives was 5.8% and 5.7%, respectively, somewhat higher than the 2.8% and 3.3% found among men working at a textile factor and their wives (Ryder, Ndilu, Hassig, Kamenga, Sequeira, Kashamuka *et al.*, 1990). In this, as in many other early studies in Africa, higher status and income was associated with higher HIV prevalence. During 1986 – 91, median HIV prevalence in urban antenatal clinics ranged from 4.7% to 7.1% and outside major urban areas from 1.1% to 3.8% (omitting years with only one clinic reporting in a category)(UNAIDS, 2000d).

From the mid-to-late 1980s, evidence suggests stable or declining HIV prevalence in the DRC. After a 1997 study of HIV in antenatal women, blood donors, tuberculosis patients, commercial sex workers, and STD patients in three locations (Kinshasa, Mbuyi-Mayi, a south-central city, and Bwamanda, a northern town), the study team reported that 'Despite the social disruption, the rapid decline in health-care provision, and the decrease in funding in health education programmemes, our results show that the HIV seroprevalence rates remain relatively low and stable in DRC' (Mulanga-Kabeya, Nzilambi, Edidi, Minlangu, Tshimpaka, Kambembo *et al.*, 1998, p. 908). The median HIV prevalence in three urban antenatal clinics in 1997 was 3% and in 14 clinics outside major areas it was 3.7% (UNAIDS, 2000d). In 2002, HIV prevalence among antenatal women in Kinshasa was 3.0%. Moreover, WHO/AFRO (2003, vol. 2, p. 124) assessed that 'available data suggest a stabilization of the trend in some parts of the country', but also noted

evidence of rising HIV prevalence in Lubumbashi (near Zambia). HIV testing in rural areas with presumably lower prevalence has been limited.

In the early 2000s, HIV prevalence in the DRC appears to be lower than in most if not all neighbours. A 2001/2 national survey in Zambia found 15% HIV prevalence among adults (WHO, 2003); the median from 48 antenatal clinics across the Central African Republic (CAR) in 2002 was 15% (WHO/AFRO, 2003); median HIV prevalence in 24 Tanzanian antenatal clinics was 8.1% in 2002 (WHO/AFRO, 2003); a 1997 national survey in Rwanda reported 11% adult HIV prevalence (though prevalence seems to have fallen in recent years); and median HIV prevalence in 17 antenatal clinics across Uganda in 2001 was 5.6% (WHO/AFRO, 2003). On the other hand, a 2002 national survey in Burundi reported 3% adult HIV prevalence (WHO, 2003), Congo's 2002 median HIV prevalence across antenatal clinics was 4.3% (WHO/AFRO, 2003), and HIV prevalence in Angola is also low (see below).

Angola
From Angola's independence in 1975, the government faced civil war with União Nacional para Independencia Total de Angola (UNITA) rebels led by Savimbi. War continued with occasional truces until Savimbi's death in 2002. Agreements and ceremonies in August 2002 marked the end of the war. Over 27 years an estimated 600 000 Angolans were killed and a third of the population displaced by fighting (Peace Pledge Union Online, 2004d).

In the mid-to-late 1980s, after more than 10 years of civil war, HIV was found in Angola at low rates, with differences across provinces. A 1986 study found 0.3% of antenatal women, 4% of inpatients, and 1% of tuberculosis patients in Luanda, the capital, to be HIV-positive (Bottinger, Palme, da Costa, Dias & Biberfeld, 1988). In 1987 – 88 another study confirmed low prevalence in Luanda, but found higher prevalence in other provinces, including Lunda Norte (22% in blood donors, 12% in urban healthy persons, 22% among tuberculosis patients), Huambo, Zaire province of Angola, and Kuando-Kubango (Santos-Ferreira, Cohen, Lourenço, Almeida, Chamaret & Montagnier, 1990). In 1992, HIV prevalence was 1.7% in 11 333 blood donors from nine provinces (US Census Bureau, 2000).

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In the last decade of the war to 2002, evidence suggests a minor increase in HIV prevalence. As of 2001, UNAIDS (2002a) estimated adult HIV prevalence at 5.5% for Angola, but this may be high. Prevalence was 3.2% among military personnel in Luanda in 2001, and in 2002 the median HIV prevalence in antenatal clinics in six provinces was 2.4%. Angola's Cabinda enclave, which borders Congo and the DRC, has had somewhat higher HIV prevalence, i.e. 6.8 – 8.5% among women at the one reporting antenatal clinic during 1992 - 96 (UNAIDS, 2000b), but falling to 3.3% in 2002 (WHO/AFRO, 2003).

The US Agency for International Development (USAID) (2003, p. 1) noted that 'Angola's HIV prevalence has remained significantly lower' than in neighbouring countries, and speculated that this is 'perhaps due to the initially isolating effects of civil war...' In 2002, the median HIV prevalence in 21 antenatal clinics across Namibia, Angola's southern neighbour, was 23% (WHO/AFRO, 2003). As already discussed, a 2002 national survey in Zambia found 15% adult HIV prevalence (WHO, 2003), while prevalence in the DRC is low and may be comparable with Angola's.

Mozambique
Mozambique's civil war during 1976 – 92 devastated much of the country. South Africa supported the Resistência Nacional de Moçambique (RENAMO) to destabilise Mozambique's government and to block its support for South Africa's African National Congress. RENAMO rebels attacked government facilities; among other damage, 46% of clinics and health posts were looted, destroyed, or closed through 1988 (Finnegan, 1992). The peace agreement in 1992 led to multiparty elections in 1994, demobilisation, and extension of government services with donor support.

To the end of the war, Mozambique's HIV prevalence remained low. Among antenatal women in Maputo, HIV prevalence was 1.2% in 1992 (UNAIDS, 2002f). Prevalence was also low in regions of the country that had suffered intensively from rebel attacks. A 1992 – 93 survey of antenatal attendees among displaced persons (mean duration of displacement 5 years; only 1% had left Mozambique) in Zambezia Province, which borders Malawi, found 26 (1.5%) of 1 728 women to be HIV-positive. One hundred and forty-five women (8%) had been raped (half more than once), of whom

4 were HIV-positive (Cossa, Gloyd, Vaz, Folgosa, Simbine, Diniz et al. 1994). A 1991 – 92 study of antenatal women and STD patients in Inhambane Province in southern Mozambique found no HIV among antenatal women, although 51% had one or more STD; while among 155 men and women with STD-related complaints, three men (2%) were HIV-positive (Vuylsteke, Bastos, Barreto, Crucitti, Folgosa, Mondlane et al., 1993). A 1990 – 91 survey of 1 338 prisoners in Maputo found 0.6% HIV prevalence (Vaz, Gloyd, Folgos, & Kreiss, 1995). On the other hand, a 1990 survey of displaced persons on Inhaca Island near Maputo found 10 (4.7%) of 216 volunteers with HIV infection (Fernandes, Vaz, Esteves & Noya, 1992).

From 1992, rates of HIV prevalence in Mozambique began a sharp increase. In Maputo, prevalence in antenatal women rose from 1.2% in 1992 to 5.8% in 1996 (UNAIDS, 2002f) and 18% in 2002 (WHO/AFRO, 2003). In 2002, the national median HIV prevalence across 36 antenatal clinics was 13.7%; prevalence was lower in the north than in the centre and south (WHO/AFRO, 2003). In Chimoio, a city along the Beira corridor to Zimbabwe in the centre of the country, HIV prevalence among blood donors increased to 21% (49% in military donors, and 16% in others) in 1999 (Newman, Miguel, Jemusse, Macome & Newman, 2001). These rates may be compared with 0% prevalence among blood donors in Chimoio in 1987 (Barreto, Ingold, De La Cruz, Jorge, De Sousa, Leandro et al., 1988), and less than 4% among soldiers in Tete in 1987 and Pemba in 1990 (US Census Bureau, 2000). A study of post–war HIV prevalence in Mozambique (Melo et al., 2000, p. 203) speculated that 'the opening of the borders, the return of refugees, and widespread prostitution have contributed to a dramatic increase in HIV prevalence'.

HIV prevalence in Mozambique was far below most of its neighbours at the end of the war. In 1991 – 92, when antenatal prevalence in Maputo was 1.2%, median HIV prevalence in antenatal clinics outside major urban areas was 8% in Tanzania (UNAIDS, 2000j), 19% in Zimbabwe (UNAIDS, 2002l), 10% in Zambia (UNAIDS, 2002k), and 5.5% in Malawi (UNAIDS, 2002e), while estimated national HIV prevalence in antenatal women in South Africa was 2.7% (Laurence, 2000). In the first decade after the war, Mozambique's HIV prevalence soared. Among antenatal women in sentinel surveys, rates of HIV prevalence are now higher in Mozambique than in

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Tanzania, are comparable with Malawi's, and remain somewhat lower than in Zambia, Zimbabwe, and South Africa (WHO/AFRO, 2003). However, unmeasured HIV prevalence may be lower in rural populations.

Somalia

Somalia's civil war began in the north in 1988 with a rebellion that led to the secession of Somaliland in 1991 (which has not been recognised by the international community). In 1991, armed militias overthrew the central government (Peace Pledge Union Online, 2004e). From 1991, militias have successfully rejected a new central government, and intermittent civil war continues (as of early 2004).

Somalia had few HIV infections up to the end of the 1980s. Three studies during 1985 – 89 found no HIV in prostitutes, STD patients, or tuberculosis patients in Mogadishu and two southern cities (Burans, Fox, Omar, Farah, Abbass, Yusef *et al.*, 1990; Jama, Grillner, Biberfield, Osman, Isse, Abdirahman *et al.*, 1987; Scott, Corwin, Constantine, Omar, Guled, Yusuf *et al.*, 1991). In 1990, HIV appeared at low levels in prostitutes (5 of 245) and military personnel (1 of 79) in Mogadishu and two southern cities, and none of 80 STD patients or 43 tuberculosis patients was infected (Watts, Corwin, Omar & Hyams, 1994). In 1995, a study in Mogadishu found no HIV infection among 157 blood donors, 57 inpatient adults, and 42 inpatient children (Nur, Groen, Elmi, Ott & Osterhaus, 2000). The authors observe that 'during the civil war in Somalia, no evidence of an increase in HIV-1 infections was found (p. 137)'. The latest information on antenatal women in Mogadishu showed no HIV infections in 1998 (UNAIDS, 2002h). However, in northern Somalia (Somaliland), HIV has been found at low levels in the general population: a 1999 study in three cities reported 0.9% HIV prevalence in antenatal clinics and 4.6% among 314 tuberculosis patients (Abokor, 2000).

HIV prevalence in Somalia has been much lower than in neighbouring countries. Kenya's and Ethiopia's HIV epidemics reached double-digit rates of infection in at least one urban antenatal clinic by 1990 – 91 (UNAIDS, 2002d; UNAIDS, 2000e). A Demographic and Health Survey in Kenya in 2003 reported 6.7% adult HIV prevalence (Kenya, Central Bureau of Statistics, 2003). In Ethiopia, the median HIV prevalence among 34 antenatal clinics nationally was

11% in 2001 (WHO/AFRO, 2003). In Djibouti, the latest data from antenatal clinics showed 2.9% HIV prevalence in 1996, while 17.2% of tuberculosis patients were HIV-positive in 1998 (UNAIDS, 2002c).

Southern Sudan

Rebel armies in Sudan's southern regions have fought for autonomy or secession for decades. The first civil war began when Sudan became independent in 1956 and lasted 16 years. The second began in 1983 with a rebellion led by the Sudan People's Liberation Movement/Army (SPLM/A). From 1991, a split within the rebel movement led to additional fighting. Up to 2002, an estimated two million Sudanese had died during the second civil war, four million had been internally displaced, and 400 000 had sought refuge in neighbouring countries (Prunier & Gisselquist, 2003). Despite progress in talks, fighting continues in early 2004.

In 1986, a study of 130 inpatients with fever in Juba, a southern city, found HIV in two, a prisoner and a Ugandan soldier (Woodruff, Morrill, Burans, Hyams & Woody, 1988). Three years later, another study in Juba identified HIV in 3% of outpatients, 19% of tuberculosis inpatients, and 16% of prostitutes (McCarthy & El Hag, 1990). Subsequent surveys in an antenatal clinic in Juba reported 3% and 3.5% HIV prevalence in 1995 and 1998 respectively (UNAIDS, 2002i). HIV prevalence in Juba, which has been under central government control, may not be characteristic for surrounding contested and rebel-held territory. In 2002 and 2003, random sample surveys in southern Sudan (assisted by the US CDC) reported adult HIV prevalence around 4% in Yei town near Sudan's border with Uganda, less than 1% in rural areas near Yei town, and less than 0.5% in Rumbek, a small town further north from the border (Kaiser, Kebdamo, Lane, Kessia, Handzel, Downing *et al.*, 2004). Figures for rural Yei and Rumbek may be the best indicators of HIV prevalence in rural southern Sudan.

Populations in southern Sudan affected by war have experienced later and fewer HIV infections than populations in neighbouring countries. Southern Sudan borders Ethiopia, Kenya, Uganda, DRC, CAR and Chad. By 1992, median HIV prevalence in antenatal clinics outside major urban areas had been reported as high as 4.9% in Ethiopia (UNAIDS, 2000e), 7.9% in Kenya (UNAIDS, 2002d), 11% in Uganda (UNAIDS, 2002j), and 6.5% in CAR

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(UNAIDS, 2000c). Antenatal HIV prevalence in Gulu District in northern Uganda was reported at 27% in 1993 (UNAIDS, 2002j).

A note on other wars

Accounts of HIV epidemics in Tanzania, Uganda, and Guinea Bissau have speculated on a role for civil disorder and wars. Hooper (2000) proposed that Tutsi refugees fleeing ethnic violence in Rwanda introduced HIV into northwest Tanzania and southwest Uganda around 1960. In the early 1960s, medical authorities in Uganda and Tanzania recorded unusual cases among recent Rwandan immigrants of Kaposi's sarcoma and other diseases now recognised as opportunistic infections associated with AIDS. This timing is plausible: from genetic evidence, HIV was circulating in central Africa well before 1950 (Korber et al., 2000; Vidal et al., 2000). One hypothesis that fits this evidence is that immunodeficiency viruses crossed from chimpanzees to humans through a cut hunter, and then spread through unsterile health care, including especially intensive programmes to find and treat trypanosomiasis in French and Belgian colonies in central Africa during 1930 – 1950 (Gisselquist, 2004). However, Hooper's (2000) hypothesis that oral polio vaccines introduced HIV into Rwanda in the 1950s does not fit the genetic evidence. The regions of Tanzania and Uganda where Tutsi refugees settled around 1960 were recognised over 20 years later with high HIV prevalence. Hence, ethnic violence in Rwanda may have spread HIV to neighbouring countries through refugee resettlement.

Serwadda et al. (1985) and Hooper (2000) speculate that Uganda's 1978-79 war with Tanzania contributed to Uganda's HIV epidemic. In October 1978, Ugandan soldiers crossed the border with Tanzania west of Lake Victoria and occupied over 1 000 km², and then withdrew several weeks later. Tanzanian troops assembled in northwest Tanzania from November 1978, invaded Uganda in January 1979, and took Kampala in April (Kasozi, 1999). While the occupied area in Tanzania and invasion routes through southwest Uganda were later found to have high HIV prevalence, large groups of soldiers spent no more than a few months in these regions. This was probably too little time for more than about 1% to have become HIV-infected, which in turn limited their potential role as vectors for HIV transmission (cf. in five studies of African men in serodiscordant couples in which many men were unaware of their wife's HIV status, their unweighted average HIV incidence was 7.1 per 100 person years (PYs) [Gisselquist & Potterat, 2003]; other studies report HIV incidence of 6.6 per 100 PYs among Kenyan truckers [Martin, Jackson, Mandaliya, Bwayo, Rakwar, Nyange et al., 1994] and 2.0 per 100 PYs among Tanzanian policemen [Bakari, Lyamuya, Mugusi, Aris, Chale, Magao et al., 2000]). Hence, it seems more likely that high HIV prevalence in border regions built up over time, both before and after the war, and that peacetime commerce and travel contributed more than troop movements to the spread of HIV to other parts of Uganda and Tanzania.

Several authors have hypothesised that Guinea-Bissau's liberation war in 1963 – 74 contributed to the spread of HIV-2 through increases in sexual and health care exposures (Hooper, 2000; Poulsen, Aaby, Jensen & Dias, 2000). A variety of evidence supports this hypothesis, but also fits patterns of peacetime, urban-centered HIV epidemic growth common in Africa. Genetic analyses of HIV-2 samples collected in Guinea-Bissau in 1989 – 92 suggest a transition around 1955 – 70 from no growth to 20% annual epidemic growth (Lemey, Pybus, Wang, Saksena, Salemi & Vandamme, 2003). According to this evidence, whether or not growth began during the war, it continued for 15 years after the war. The geographic distribution of infections in 1980 was centered on Bissau, the capital (Piedade, Venenno, Prieto, Alburquerque, Esteves, Parreira et al., 2000). Many of those found infected in samples collected during 1980 – 92 were too old or too young to have been involved in much sexual transmission during the war (Poulsen, Aaby, Jensen & Dias, 2000; Wilkins, Ricard, Todd, Whittle, Dias & Da Silva, 1993). Moreover, assumptions about the survival of persons with HIV-2 infections are required to interpret this data.

Discussion

This paper describes levels and changes in HIV prevalence in seven African countries experiencing long-term civil disorders or wars over the last several decades. In one country (DRC), adult HIV prevalence appears to have stabilised; in two (Sierra Leone and Somalia), it increased from near zero to less than 1%; in three (Angola, Mozambique, and southern Sudan), it increased from near zero to undetermined but low rates; and in Liberia, adult HIV prevalence increased from near zero, but current rates are unknown. In Mozambique, the one country where post-war

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movements in HIV prevalence have been observed, prevalence appears to have increased by more than 10% (from roughly 1% to 14%) over the decade from 1992 (UNAIDS, 2002f). Only a handful of countries in southern Africa have experienced greater increases in HIV prevalence over a decade.

These findings are relevant to ongoing research and debate to identify factors that explain differential epidemic trajectories, as well as related debates about what to include in HIV prevention programmes. Some key conclusions are discussed below.

Communities with HIV prevention programmes often do no better than communities at war, with no prevention programmes

Observed trends in HIV prevalence in countries with long-term civil wars may be compared with UNAIDS' (2002a) estimate of 9% adult HIV prevalence in sub-Saharan Africa in 2001. Allowing for some overestimate, this suggests an average increase in adult HIV prevalence of 3 – 4% per decade from 1980 to 2000, which exceeds what has been observed in countries with long-term civil wars. On the other hand, a number of African countries at peace have demonstrated low or falling HIV prevalence that compares favourably with what has been observed in countries with long-term civil wars. Senegal and Madagascar have maintained adult HIV prevalence below 1% (UNAIDS, 2000a). Declining HIV prevalence in Uganda from 1990 has been widely recognised (Green, 2003). Less well known are comparable declines suggested by some evidence in Burundi (WHO, 2003), Rwanda (WHO/AFRO, 2003), Kagera District in Tanzania (Kwesigabo, 2001), and possibly other countries and communities as well.

During civil disorders and wars, WHO, UNHCR & UNAIDS (1996, p. 6) recognise 'a strong likelihood that AIDS control activities, whether undertaken by national governments or NGOs, will have been severely disrupted or have broken down altogether'. Hence, it is noteworthy that epidemic trajectories in countries at war compare favourably with what is observed in many countries at peace. Although there is no question that HIV prevention programmes do prevent some HIV transmission (e.g. providing condoms to men who would otherwise contract or spread HIV), the failure to outperform consistently the 'no programme' option observed in countries at war demonstrates that these positive impacts are often

outweighed by other factors. However, with few exceptions, even the lowest rates of HIV prevalence in African countries at war or peace are high on a world scale, and represent a failure to prevent spread of HIV beyond high-risk groups such as men who have sex with men, injection drug users, and their sexual partners. Prostitute women are also often a high-risk group in Africa and Asia (which may be due to unsterile health care [Packard & Epstein, 1991] as well as sexual exposures), although not in Europe, where 'prevalence is generally less than 2%, except in settings where most HIV-infected prostitutes seem to be injecting drug users' (Hamers & Downs, 2004, p. 89).

Low risk for HIV in war zones corresponds to low risk in remote rural populations and in persons with low socioeconomic status

Within African countries at peace, HIV is associated with residence in or convenient access to cities or towns. In a 1987 survey in Tanzania, adult HIV prevalence in Bukoba town was 24%, dropping to 10%, 4.5%, and 0.4% in progressively more remote rural wards (Killewo, Dahlgren & Sandstrom, 1994; Killewo, Nyamuryekunge, Sandstrom, Bredberg-Raden, Wall, Mhalu et al., 1990). At maternity units in Kimpete, DRC, during 1988 – 91, HIV prevalence was 11.7% in women from large cities, 4.3% from small towns, 3.7% from villages within 1.7 kilometers from a main road, and 1.7% from more remote villages (Green, Mokili, Nganzi, Davies, Hardy, Jackson et al., 1992). In Hlabisa, South Africa, in 1997, HIV prevalence in 11 antenatal clinics varied inversely according to the isolation of the population in each clinic's catchment area (measured as the mean distance from homesteads to a primary or secondary road). Thus, across clinics, HIV prevalence fell roughly 2% per kilometer ($r = 0.66$, $p = 0.002$) (Tanser, LeSueur, Solarsh & Wilkinson, 2000). In a rural district in Malawi in 1987 – 90, HIV prevalence in antenatal women living more than 8 km from a clinic was 4.7% compared with 8.4% for those living closer. In the same study, 17% of women with more than 10 years of education were HIV-positive, compared with 8% with less than four years (Slutsker, Cabeza, Wirima & Steketee, 1994). In a 1989 – 93 study in Rwanda, HIV incidence was 8.6 times greater among urban than rural women (Bulterys, Chao, Habimana, Dushimimana, Nawrocki & Saah, 1994). Botswana is an exception to this pattern, with comparable levels of

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HIV prevalence in urban and rural antenatal clinics (WHO/AFRO, 1993).

Within urban and rural African communities, HIV is often associated with more education and other measures of high socioeconomic status. A recent review of evidence from published studies found that Africans with more years of schooling were often at higher risk for HIV-positivity. Moreover, the association between education and HIV was comparable in men and women, and stronger in rural populations and older cohorts (Hargreaves & Glynn, 2002). In a study of urban antenatal women in Malawi in 1989 - 90, having more compared with less than 8 years of education more than doubled the woman's risk for prevalent HIV infection. Similarly, having a husband with more compared with less than 8 years of education more than doubled the woman's risk for HIV, but reduced her risk for STDs (Dallabetta, Miotti, Chiphangwi, Saah, Liomba, Odaka et al., 1993). In Lusaka in 1985, HIV prevalence ranged from 8% in adults with 0-4 years of education to 33% in those with more than 14 years (Melbye, Njelesani, Bayley, Mukelabai, Manuwele, Bowa et al., 1986). However, associations between education — and more generally, higher socioeconomic status — and HIV are weaker or even reversed in some recent studies. For example, a 1996 study in Kisumu, Kenya, reported higher socioeconomic status associated positively or negatively with HIV for various age and sex cohorts (Hargreaves, Morison, Chege, Rutenberg, Kahindo, Weiss et al., 2002).

Alternate views on the importance of HIV transmission through unsterile health care lead to alternate hypotheses for slow spread of HIV during civil wars
Since the late 1980s, most AIDS experts have accepted the hypothesis that sexual transmission accounts for almost all HIV infections among African adults. From this viewpoint, differences in sexual risk factors should explain differences in HIV epidemic trajectories across countries. Although hundreds of studies find that sexual behaviour and biological variables are personal risks for HIV infection, research to date has been unable to identify even one such variable that is consistently more common in countries with high compared with low HIV prevalence (Gisselquist et al., 2004). For example, a recent African study found adults in cities with high HIV prevalence not to have more syphilis, gonorrhoea, chlamydia, and non-marital

and concurrent sexual partners than adults in cities with low HIV prevalence (Buve et al., 2001). In other studies, differences in prevalence of herpes simplex virus 2 (Orroth, Korenromp, White, Changalucha, de Vlas, Gray et al., 2003) and lack of male circumcision (Boerma et al., 2003) across communities do not correlate with differences in HIV prevalence. Hence, the view that sex accounts for almost all HIV transmission to African adults remains an hypothesis in search of evidence.

A competing hypothesis proposes that differences in HIV transmission through blood exposures — particularly during unsterile health care — drive epidemics in countries where HIV is not concentrated among men who have sex with men, injection drug users, and their sexual partners (Gisselquist & Potterat, 2003; Gisselquist, 2004). From this perspective, HIV prevention in countries with generalised epidemics should address health care transmission along with sexual risks. Between these two views, there are many possible compromise positions, with health care accounting for more than 5% to 10% of infections, but not enough to drive generalised epidemics. In countries with generalised epidemics, there are to date no reliable estimates of the proportions of HIV infections from sexual and health care exposures, so it is not yet possible to end the debate with an empirical resolution.

Experts who accept the view that sex explains most HIV infections in Africa have tried to explain observed low HIV prevalence during wars as due to low rates of sexual transmission. For example, Mulanga-Kabeya et al. (1998, p. 909) suggest for DRC that 'the high degree of poverty experienced in the country as a result of the political and socio-economic crisis could have contributed in the change of sexual behavior.' For Mozambique, Barreto, Liljestrand, De Sousa, Bergstrom, Bottinger, Biberfeld et al. (1993, p. 687) suppose that 'closed roads and isolation from neighboring countries' limited HIV entry. Although war may reduce the frequency of HIV-infected truck-drivers copulating with sex workers, the level of HIV infection in soldiers and rebels and their sexual activities must be considered as well. It is not at all obvious that civil wars cause a net reduction in sexual risk. WHO, UNHCR & UNAIDS (1996) speculate that wars increase sexual risks (abuse of women and boys, earlier sexual activity, and prostitution) as well as

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non-sexual risks (injection drug use and transfusion of untested blood).

On the other hand, if one supposes that unsterile health care accounts for an important proportion of HIV infections in countries with generalised epidemics, then slower epidemic growth during wars may be explained by wars reducing exposures to unsterile health care. Health care services diminish as aid and government health expenditures collapse, and violence drives away health workers and destroys facilities. Lack of access to health care in southern Sudan is demonstrated by a finding of 0% of common pathogens (pneumococci, *S. pyogenes, H. influenzae*) resistant to common antibiotics in samples collected in 2000 in a rural hospital that had been isolated by civil war for 18 years (Doczeova, Liskova & Kremery Jr., 2003).

In what might be a macabre parallel to wartime conditions reducing public access to health care and HIV transmission, Seale (1986, p. 123) explains the spread and end of the Ebola outbreak in Yambuka, DRC, in 1976: 'Once the hospital closed on 28 September because of the death of three-quarters of the hospital staff, the epidemic vanished as abruptly as it started, leaving 280 people dead... The cause, and amplifier, of this particularly explosive epidemic was traced by the international team to five hypodermic needles and syringes, the daily allocation for the nurses for use on all ward patients in the 120-bed hospital and on all 400 outpatients per day... [T]he five hypodermics...were never sterilised between use all day.'

Evidence for slow growth of HIV epidemics during long-term civil wars may be easier to correlate with wartime reductions in health care exposures than with unknown differences in sexual exposures. If so, the evidence would seem to favour the view that unsterile health care accounts for an important proportion of HIV transmission in Africa. However, alternate interpretations are possible, so that evidence is suggestive, not decisive. Further research in early post-war populations might help to characterise better wartime sexual and blood exposures as risks for HIV infections.

Conclusion

In African communities subject to civil disorder and war, people are impoverished and cut off from government services. Setting aside war-related violence, these wartime conditions are approximated in peacetime among remote rural populations with low socioeconomic status. Factors that explain relatively low HIV prevalence in countries after long-term civil wars may help to explain much of the diversity in HIV epidemic trajectories across African countries and communities. Identifying what those factors might be (e.g. more or different sexual or medical exposures in peacetime than in war) may lead to more effective prevention programmes that will consistently outperform the 'no programme' option.

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