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SEX, POVERTY AND HIV

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Introduction

UNAIDS has recently been subject to a series of attacks for supposedly kow-towing to political correctness by overplaying the risks of generalised HIV epidemics and failing to concentrate on the risky behaviours of key groups (notably men who have sex with men, sex workers, and injecting drug users) for fear of stigmatising them and causing offense (e.g. Chin 2007; Pisani, 2008). It has also been taken to task for highlighting gender inequality and poverty as social drivers of the HIV epidemic in Africa rather than facing the challenge of addressing the multiple concurrent sexual partnerships which really fuel it (Chin, 2007: 54; Epstein, 2007). UNAIDS officials responded by defending the institution's record on prevention and by emphasising that the challenge is to know the local epidemic and its drivers, and to craft interventions accordingly (De Lay and De Cock, 2007; De Cock and De Lay, 2008).

This, of course, leaves open the question of the relationship between, and relative importance of, the social drivers of HIV (notably poverty) and sexual behaviour. This is especially contentious with regard to Africa. Some stress the importance of sexual culture (Epstein, 2007) whereas others point to the legacy of colonial exploitation and structural adjustment in underpinning behavioural vulnerability to HIV (e.g. Barnett and Whiteside 2002; Fenton 2004; Poku 2005) and even to a hypothesised biological vulnerability of poor people to HIV infection (Stillwaggon, 2006). This paper reviews the evidence on poverty, sexual behaviour and AIDS. It argues that contextual factors within Africa are more salient than economic factors and that a more nuanced and localised approach is indeed an appropriate way forward.

Poverty and AIDS

The most extreme version of the 'poverty causes AIDS' argument is that by Stillwaggon (2006). Drawing on her earlier work (Stillwaggon 2000, 2002) claiming that HIV took off in Africa because of its 'fertile terrain' of poverty and warning (incorrectly, as it turned out) that all poor countries faced explosive epidemics, Stillwaggon argues that malnourished people, especially those infected with worms, weakened by Tuberculosis (TB) and malaria, and burdened by untreated sexually transmitted infections (STIs) are particularly biologically vulnerable to HIV. This is because STIs cause genital lesions and

malnutrition weakens mucosal and skin integrity (both of which create more entry points for HIV), and infection with parasites and other diseases over-activates the immune system thereby creating more target cells for HIV infection. Accordingly, she argues that AIDS interventions should have focussed on addressing poverty, improving basic health care, and on eradicating poverty-related biological risk factors – such as worm infections, untreated ulcerative STDs and genital schistosomiasis.

There is certainly evidence that people who are infected with parasites and other diseases have an elevated immune response and that this increases susceptibility to HIV infection (e.g Bentwich *et al.* 2000; Clerici *et al.* 2001; Fauci 2007). But, while this points to the importance of addressing co-infections, it is not sufficient evidence for the claims made by Stillwaggon for the *primacy* of poverty-related biological pathways in driving the HIV epidemic. The fact that Malawian patients with worms were found to be less, rather than more, likely to have HIV (Hosseinipour *et al.* 2007) demonstrates the need for more caution in this regard.

On the issue of malnutrition, here too, there is some evidence to support her claims, but not enough to posit primacy for poverty-related nutritional deficiency as the key biological driver for the epidemic. Indeed, the evidence is weak and often contradictory. For example, a Rwandan study showed that those who became HIV-positive during the study period had previously lost an average of 1.5 kg, whereas those who remained negative did not (Moore *et al.* 1993). This suggests the possibility of a poverty-related caloric shortfall as a determinant of HIV infection. However as no relationship was found between sero-conversion and socio-economic variables or blood vitamin levels, the study concluded that more research was necessary. Similarly, there is some evidence that improvements in nutritional status might slow HIV disease progression (e.g. Ambrus and Ambrus 2004; Fawzi *et al.* 2004), there is no consistent or reliable evidence linking vitamin deficiency and HIV infection (Dreyfuss and Fawzi 2002; MacDonald *et al.* 2001). Not only have randomised controlled trials of micronutrient supplementation (notably Vitamin A) not shown any protective effect on HIV transmission, they may even have caused harm (Mills *et al.* 2005).

In short, there is some weak suggestive evidence that people living in poor conditions and with inadequate public health systems may be biologically more vulnerable to HIV infection. However, Stillwaggon takes her argument beyond what the evidence can support by claiming that ‘biomedical research provides sufficient evidence for examining the HIV/AIDS epidemic by conventional epidemiological and economic means’ – rather than treating HIV as an exceptional disease driven by sexual behaviour (2006: 80).

Stillwaggon mobilises cross-country regression analysis in an attempt to show that it was the decline in calorie consumption in Africa between 1970 and 1995 (itself a function of drought and economic hardship) which was the key driver of the AIDS epidemic. However, her analysis considers only 44 countries and excludes obvious alternative explanatory variables.[1] As argued below, neither calorie consumption nor change in calorie consumption is a statistically significant determinant of cross-country variation in HIV prevalence once regional and cultural factors are accounted for.

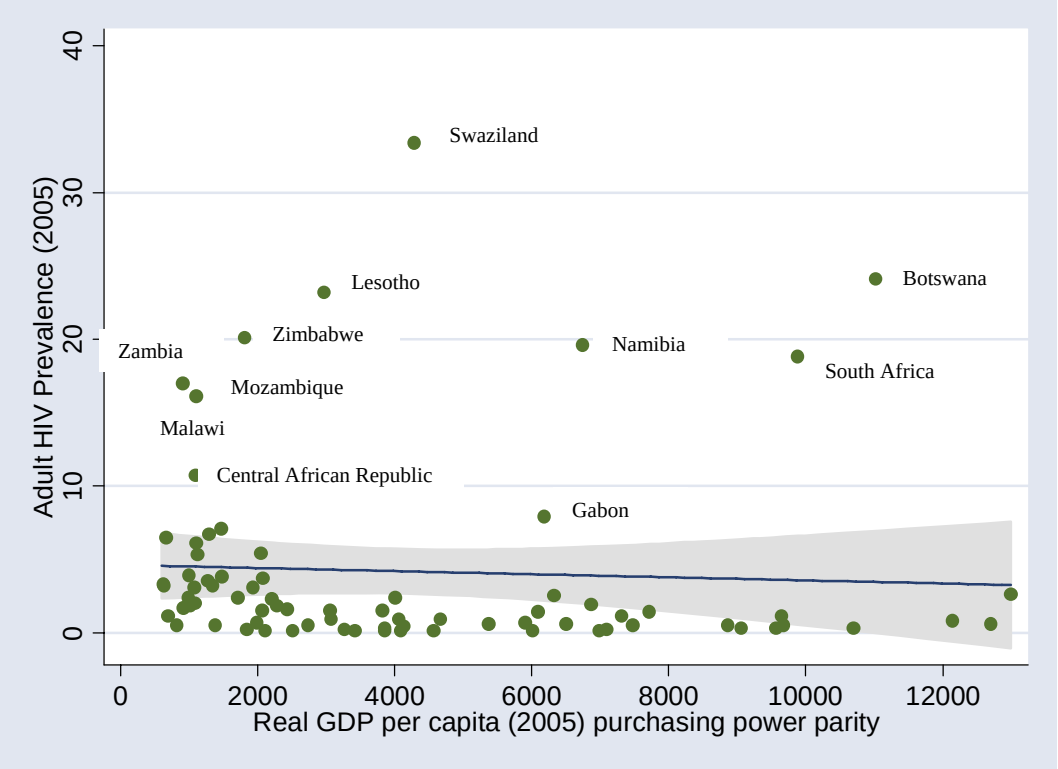

Figure 1: HIV Prevalence and GDP Per Capita

Figure 1 shows that there is no obvious relationship between the most basic development indicator – per capita income – and HIV prevalence. Zanakis *et al*. (2007) argue that the 'spoilers' of the anticipated relationship between economic development and HIV prevalence are the 'healthier of the wealthiest' and the 'wealthier of the sickest'. However, a quick inspection of the data instead suggests a strong regional dimension to the epidemic, with the middle-income countries of Southern Africa (notably Namibia, Botswana, and South Africa) appearing as major outliers. (These same countries also exercise a

[1] Stillwaggon's variables include the Gini coefficient, change in per capita income and urbanisation between 1970 and 1995, and change in calorie consumption between 1970 and 1995 (2006: 84-7).

disproportionate effect on the estimated cross-country relationship between HIV prevalence and income inequality (Nattrass 2008)).

Table 1 explores the relationship between HIV prevalence and poverty more systematically, and in the context of other potential determinants.[2] Regression 1.1 shows that when controlling for per capita income and type of epidemic, calories per capita are significantly (and negatively) associated with HIV prevalence (a one percent increase in calories per capita is associated with a 2.9% decrease in HIV prevalence). However, the relationship between HIV prevalence and per capita income is statistically insignificant.

Type of epidemic is proxied by a dummy variable which takes a value of one if the country reported HIV prevalence data for either 'men who have sex with men' (MSM) or for injecting drug users.[3] This is to account for cross-country variation arising from the fact that countries with these 'concentrated' epidemics are likely to have smaller national HIV prevalence rates. Regression model 1.1 predicts that after controlling for per capita income and calorie consumption, concentrated epidemics are expected to have HIV prevalence rates 15% of that of countries with heterosexual epidemics.[4]

[2] Log transformations are used to reduce the effect of outliers (and to provide a more normal distribution of the variables).
[3] This variable was constructed on the basis of country fact sheets available on www.unaids.org
[4] The antilog of the coefficient on the MSM/IDU dummy variable is 0.15.

Table 1: Per Capita GNP, Inequality and Adult HIV Prevalence

Dependent variable: Log of Adult HIV Prevalence (2005)	1.1	1.2	1.3	1.4#	1.5	1.6	1.7
Constant	***	**	*				*
Coefficient	20.646	16.479	11.094	7.787	6.478	8.170	15.169
(standard error)	(7.673)	(9.099)	(6.561)	(5.665)	(7.209)	(6.280)	(8.001)
P>|t|	0.009	0.075	0.095	0.173	0.373	0.198	0.063
Log of per capita GDP in 2005			*				
Coefficient	0.345	0.057	0.392	0.248	0.216	0.238	0.319
(standard error)	(0.213)	(0.254)	(0.226)	(0.194)	(0.252)	(0.201)	(0.218)
P>|t|	0.109	0.824	0.086	0.206	0.395	0.241	0.148
Log of calories per capita 2002/04							
Coefficient	***	*	**				*
(standard error)	-2.878	-1.804	-1.852	-1.282	-1.002	-1.347	-2.304
P>|t|	(1.104)	(1.318)	(0.931)	(.807)	(1.054)	(0.887)	(1.142)
	0.012	0.176	0.050	0.116	0.346	0.133	0.048
MSM/IDU epidemics	***	***	*		*		*
Coefficient	-1.872	-1.891	-0.659	-0.502	-0.673	-0.493	-0.607
(standard error)	(0.309)	(0.344)	(0.478)	(0.287)	(0.361)	(0.310)	(0.310)
P>|t|	0.000	0.000	0.053	0.084	0.066	0.116	0.054
Southern Africa			***	***	***	***	***
Coefficient			3.067	2.642	2.206	2.681	2.409
(standard error)			(0.531)	(0.478)	(0.626)	(0.502)	(0.507)
P>|t|			0.000	0.000	0.001	0.000	0.000
Rest of Sub-Saharan Africa			***	***	***	***	***
Coefficient			1.634	1.673	1.578	1.801	1.471
(standard error)			(0.428)	(0.379)	(0.464)	(0.473)	(0.421)
P>|t|			0.000	0.000	0.001	0.000	0.001
Predominantly Muslim				***	***	***	**
Coefficient				-0.855	-0.832	-0.768	-0.725
(standard error)				(0.246)	(0.289)	(0.268)	(0.291)
P>|t|				0.001	0.007	0.006	0.015
Predominantly Protestant				***	**	***	***
Coefficient				0.693	0.754	0.715	0.882
(standard error)				(0.261)	(0.319)	(0.265)	(0.296)
P>|t|				0.010	0.022	0.009	0.004
Log of income share bottom 20%		***					
Coefficient		-1.109			-0.331		
(standard error)		(0.324)			(0.289)		
P>|t|		0.001			0.256		
State Legitimacy (Englebert)							
Coefficient						0.259	
(standard error)						(0.258)	
P>|t|						0.319	
Change in per capita calorie consumption 1979/81 to 2002/4							
Coefficient							0.000
(standard error)							(0.001)
P>|t|							0.336
Adjusted R-squared	0.4322	0.5418	0.6120	0.7431	0.7349	0.7133	0.7181
Number of observations	81	66	81	81	66	79	74

* Significant at the 10% level, ** significant at the 5% level, *** significant at the 1% level

This is a robust regression (to address heteroskedacticity, which was a problem only for this model)

Regression 1.2 adds a further poverty indicator: the share of income going to the poorest quintile. If poverty drives the spread of HIV for either biomedical or behavioural reasons (e.g. if poor women are more likely to engage in risky sex (as suggested, for example, by Letamo and Bainame 1997; Hunter 2002), Weiser *et al* 2007) then we would expect a negative relationship between the share of income going to the poor and HIV prevalence. However, there is evidence that better off people may be as, if not more, vulnerable to HIV (e.g. Gillespie *et al.* 2007; Bärnighausen *et al.* 2007; Mishra *et al.* 2007). This may well be a consequence of the relationship between wealth and participation in concurrent sexual partnerships – a practice which has been shown to increase HIV risk enormously (e.g. Morris and Kretzschmar 1997a, 1997b; Epstein 2007):

> "Wealth is the key for such networks, because wealth is associated with the mobility, time and resources to maintain concurrent partnerships. Clearly such relationships might often have a strong economic element, but poverty itself may not be a major factor. Similarly, wealth and social interaction are inextricably linked, and wealth might increase the number of opportunities for partnerships to develop" (Shelton *et al.* 2005: 1058).

As the relationship between wealth and HIV is even stronger for women than men, they observe that this undermines the oft-made claim that poverty drives women to engage in risky sex. Shelton *et al.* conclude: "it appears that both wealth and economic disadvantage, or at least the desire for economic advancement) play pivotal roles in HIV transmission (2005: 1058). This suggestion was supported by a subsequent comparative study of socio-economic status and HIV infection in eight sub-Saharan African countries which found that adults in the wealthiest quintiles had a higher HIV prevalence than those in the poorer quintiles, but that this association became insignificant in multivariate models (Mishra *et al.* 2007).

In other words, one should not necessarily expect there to be a cross national relationship between relative poverty and HIV prevalence. As it turns out, regression 1.2 predicts that controlling for the other variables in the model, a one percent increase in the share of income going to the bottom 20% is associated with a decrease of 1.1% in HIV prevalence and that an increase of one percent in per capita calorie consumption is associated with a 1.8 percent decrease in HIV prevalence. Unfortunately, adding the share of income going to the lowest quintile to the model reduces the number of observations to 66 because of missing data but it nevertheless indicates that across a large sample of countries both inside and outside of sub-Saharan Africa, greater relative poverty is

associated with higher HIV prevalence. But it is important to note that this relationship falls away when regional and cultural variables are included (see Regression 1.5).

Regression 1.3 includes two dummy variables; one for being a Southern African[5] country and the other for countries in the rest of Sub-Saharan Africa. This is to pick up effects which are unique to Africa and which have not been captured by the inclusion of other variables. An obvious reason for including African dummy variables is the key point that the AIDS epidemic started in Africa (Iliffe 2006: 58) and spread through the continent as a consequence of 'massive demographic growth, urbanisation and social change during the latter 20th century' taking its shape from 'the structure of the commercial economy that had grown up during the colonial period' (*ibid*: 2). Accounting for the specific context of Africa thus makes sense. Furthermore, separating the impact of being a Southern African country from the rest of Sub-Saharan Africa is useful to account for its greater economic power and history of migrant labour within the region. This is reflected in the virus itself: whereas the rest of Africa displays a medium to high degree of genetic diversity, Southern Africa is dominated by HIV Sub-type C, and South African viruses are interspersed among sequences from Zambia, Malawi, Botswana and Malawi (Williamson and Martin 2005: 113-4). This suggests that there are factors that are unique to the Southern African AIDS epidemic which could usefully be captured by a Southern African dummy variable. As can be seen in regression 1.3, the coefficients on the African dummy variables are large, positive and highly significant. This is discussed in more detail below.

Model 1.4 adds two cultural variables: a dummy variable for whether the country is predominantly Muslim and a dummy variable for whether the country is predominantly Protestant. This is to capture the potential impact of religion on sexual behaviour given that Muslim societies are less tolerant of sexual freedom[6] than other religions, whereas Protestant countries tolerate a greater degree of sexual freedom than other countries. There is also a biological reason for including these religious dummy variables because Muslim's practice male circumcision (and Protestants do not). Men with foreskins are known to be at a higher risk of HIV infection as the foreskin is rich in HIV target cells (Lagerhans' and dendritic cells, CD4+ T cells and macrophages) and is subject to small tearing during sex (Donoval *et al.* 2006; Szabo and Short 2000). Observational studies have demonstrated a clear link between reduced HIV

[5] This includes the Southern African countries of Botswana, Lesotho, Namibia, Swaziland, South Africa.

[6] Iliffe argues that the Muslim social order was probably an important contributing factor to the lower HIV prevalence in West Africa (2006: 48-56).

prevalence and circumcision (Siegfried *et al.* 2005) and clinical trials have demonstrated that circumcision reduces the risk of HIV infection by about 60% (Auvert *et al.* 2005; Bailey *et al.* 2007 and Gray *et al.* 2007).

Note that including the two religious dummy variables increases the explanatory power of the model dramatically whilst rendering the economic explanatory variables insignificant. More specifically, regression 1.4 shows that controlling for the other variables in the model, Southern African countries are expected to have HIV prevalence rates 14 times higher than that of other countries (and other Sub-Saharan countries are expected to have HIV prevalence rates 5 times higher than anywhere else);[7] predominantly Muslim countries are likely to have HIV prevalence rates that are 58% of the levels manifest in other countries and predominantly Protestant countries are expected to have HIV prevalence rates twice that of other countries.[8] Regression 1.5 shows that including a proxy for degree of poverty makes no substantive difference to the size and significance of most of the other variables and is itself statistically insignificant.

This analysis suggests that poverty (as proxied by per capita income, calories per capita and relative inequality) may play a role in the HIV epidemic in some countries but that its overall impact is dwarfed by that of religion, the nature of the epidemic, and by other factors specific to Africa. This is consistent with survey evidence from Africa (see review article by Mishra *et al.* 2007) and Iliffe's assessment that poverty was not a major driver of the African AIDS epidemic. Citing Noreen Kaleeba (who started the AIDS support organisation (TASO) in Uganda) that 'AIDS affects ordinary people' – i.e. not just the poor – Iliffe concludes that poverty did not 'give birth to HIV' but was an 'effective incubator' which 'accentuated the suffering of AIDS patients' (2006: 63-4; see also Poku 2005; Collins and Leibbrandt 2007). Interestingly, recent evidence from rural KwaZulu-Natal shows that deaths amongst young adults between 1993 and 2004 fitted the pattern predicted by demographic modelling, that those who died were not poorer (prior to getting sick) than those who did not (Sienaert 2007), and that there was no relationship between socio-economic status and HIV incidence (Bärnighausen *et al.* 2007). In other words, 'ordinary' people died of HIV in rural KwaZulu-Natal, not just the poor and marginalised. Similarly, data analysis from the South African Demographic and Health Survey found no significant links between socio-economic status, AIDS knowledge and risky sexual behaviour (Booysen and Summerton, 2002). As a recent overview of existing international studies of the relationship between poverty and HIV

[7] The antilog of the coefficient on the Southern African dummy is 14.06 and for the rest of Africa, it is 5.34.

[8] The antilog of the coefficient on the Muslim dummy is 0.42 and on the Protestant dummy, 2.0.

concludes, that 'AIDS cannot accurately be termed a 'disease of poverty'" (Gillespie *et al* 2007: s15).

Addressing poverty is clearly a top priority for any development agenda and it goes without saying that improving the incomes and living conditions of the poor will improve public health. Targeting assistance to poor HIV positive people may also make sense from a humanitarian point of view as it is the poor who are likely to be least able to deal with the negative consequences of AIDS (Poku 2005). But it would be a mistake to assume that channelling AIDS-related international assistance away from AIDS prevention and treatment interventions and towards interventions to alleviate poverty is an appropriate way of combating the epidemic itself. It is far from clear that, as claimed by Stillwaggon, we would be further down the road of combating AIDS if more AIDS-related resources had been diverted to broader developmental objectives. Whereas there is some evidence that increasing educational attainment may help combat the AIDS pandemic, there is no evidence that reducing poverty has the same effect (Bärnighausen *et al.* 2007; Gillespie *et al.* 2007).

AIDS in Africa

What are the Africa-specific factors which contribute to the AIDS epidemic, and which are probably being picked up by the Africa dummy variables in Table 1? A relevant factor may be the impact of Africa's past history of colonisation (as suggested by Iliffe 2006; Poku 2005: 17-22 and Barnett and Whiteside, 2002, chapter 5). This impact is obviously multi-faceted and impossible to capture statistically. However, Englebert has developed an indicator which records whether a country's past history of colonisation renders the state illegitimate or not. The process by which he decided a country was 'legitimate' or not is recorded in Table 2. Regression 1.6 includes this variable, but as can be seen, it is not a significant determinant of HIV prevalence and including it *increases* the size and significance of the African dummy variables. The African dummy variables must thus be capturing something else.

Other kinds of unmeasured African-specific factors which may be being captured by the African dummy variables could include social drivers such as African interpretations of disease causality (including witchcraft), approaches to death, awareness of personal risk, resistance to condoms, and problems relating to stigma and disclosure (see e.g. Caldwell 2002; Iliffe 2006). The dummy variables are probably also picking up the high prevalence of herpes simplex virus 2 (HSV-2) in Africa which is a known to heighten risk of HIV infection (Freeman 2006, Kapiga *et al.* 2007; Corey 2007). Oster (2005), in fact, argues

that the bulk of the difference in HIV transmission between the USA and Africa has to do with higher transmission rates associated with untreated STDs.[9] A recent study of Tanzanian women working in hotels found that becoming HIV positive over the study period was not associated with socio-demographic variables such as age, marital status, or religion but was strongly associated with being infected with HSV-2 (Kapiga *et al.* 2007). Unfortunately there is no cross-national data on HSV-2 prevalence, so this biological co-factor cannot be included directly in the regression and hence its effects are likely being picked up indirectly by the African dummy variables.

Table 2: Engelbert's construction of the state legitimacy dummy

		Legitimate = 1	Non-legitimate = 0
1	Was the country colonized in modern times	No	Yes (go to question 2)
2	When reaching independence, did the country recover its previous sovereignty, identity or effective existence?	Yes	No (go to question 3)
3	If the country was created by colonization was there a human settlement predating colonization?	No	Yes (go to question 4)
4	Did the colonizers (and/or their imported slaves) reduce the pre-existing societies to numerical insignificance (or assimilate them)?	Yes	No (go to question 5)
5	Does the post-colonial state do violence to pre-existing political institutions?	No	Yes

Source: Engelbert (2000).

Another, still very speculative, possibility is that the African dummy variables are picking up genetic differences that might make people of African descent more vulnerable to HIV infection. It is known that two chemokine receptors (CCR5 and CXCR4) facilitate HIV infection, that some people have genetic mutations which restrict their expression, and that they are thus less likely to get infected once exposed to HIV (O'Brien and Moore 2000). Europeans are more

[9] Oster argues that the most efficient way of combating HIV would therefore be to treat STDs. However, a recent clinical trial which used aciclovir to suppress HSV-2 found that it did not reduce the risk of HIV-infection (see conference report on http://www.aidsmap.com/en/news/AB30B830-260F-466D-A7FC-494D23C49FA0.asp.)

likely to have this restriction factor than other population groups – an outcome which is speculated to be an impact of the Black Death (*ibid*: 102). A recent study found that black people in the USA experience far greater virological failure when they are non-compliant with antiretroviral medication than their white counter-parts, and that this difference persists even after correcting for quality of life and socio-economic conditions (Schackman *et al.* 2007). The strong possibility thus exists that Africans are more vulnerable to HIV and hence that this could also be accounting in part for the significance of the African dummy.

It is, of course, likely that the dummy variables are also picking up factors specific to sexual culture/s within Africa. Stillwaggon, however, regards the very notion of a risky African sexual culture as a racist construction invented by ethnographers[10] (2006: 133-157). She argues that the cross-national evidence 'demonstrates that everywhere people have sex, everywhere some people have lots of sex, and everywhere most people do not' (2007: 26) and thus that sexual behaviour cannot account for differences in HIV prevalence (*ibid*: 17-27). But while it is true that survey based evidence on sexual behaviour (frequency of sex, number of sexual partners in a life-time) shows little significant variation across country (Wellings *et al.* 2006), such data sheds no light on the issue of *concurrent* sexual partnerships. As network modelling shows, holding the number of sexual partners constant whilst raising the degree of concurrency increases HIV prevalence substantially – whereas increasing the number of sexual partners whilst holding concurrency levels constant has little impact on HIV prevalence (Kretzschmar 2000). Given that concurrency and over-lapping sexual partnerships are common in Africa (see e.g. Caldwell *et al.* 1989, 1991; Poku 2005: 73-5; Epstein, 2007; Konde-Lule *et al.* 1997; Orubuloye *et al.* 1991; Lagarde *et al.* 2001; Iliffe, 2006; Hunter, 2002, 2006), it makes no sense to discard sexual partnership dynamics as a key driver of the AIDS epidemic. A recent study from South Africa found that concurrency was common amongst young people (Parker *et al.* 2007) and a detailed network investigation in Malawi found that 'half of all sexually active respondents were connected in a giant network component, and more than a quarter were linked through multiple independent chains of sexual relationships' (Helleringer and Kohler 2007: 2323). In the absence of cross-country data on degrees of concurrency, it may

[10] She is particularly critical of Caldwell, Caldwell and Quiggan (1989). In this regard, she is not particularly original – see Le Blanc *et al* (1991) for a critique of Caldwell et al, and Caldwell et al (1991) for a reply. Critiques of this kind of Caldwell tend to accuse him of painting an essentialist picture of African sexual culture which plugs too easily into inaccurate and exoticised notions of African sexual promiscuity. What they overlooked was his compilation of various forms of concurrency, i.e. the issue which is far more important for HIV epidemics than quantity of partnerships.

well be that the African dummy is picking up factors specific to sexual concurrency in Africa and the heightened risk that this poses for the spread of HIV.[11]

Sexual behaviour change is clearly crucial – and in this regard, the Ugandan example has been portrayed as something of a beacon of light. Uganda is well known for its sexual behaviour change and dramatic declines in HIV infection rates in the 1990s (Stoneburner and Low-Beer 2004; Green *et al.* 2006). Echoing this literature, Epstein argues that Uganda's President Museveni got it right in the late 1980s when he campaigned for 'zero grazing'. This, she says, was a feasible African solution because it recognised the reality of polygamous marriages – and the reality that men are expected to have, and do have, long-term girlfriends in different places. She argues that 'Zero grazing was a compromise, and its real message was this: "Try to stick to one partner, but if you have to keep your long-term mistresses, concubines and extra wives, at least avoid short-term casual encounters with bar girls and prostitutes. Also, you mustn't casually seduce and exploit young women who may be susceptible to your charms and wealth"' (2007: 196).

Both Iliffe (2006) and Epstein (2007) argue that African societies are responding to the AIDS epidemic by mobilising what Epstein somewhat romantically calls 'collective social energy' to promote the necessary behaviour change. Both highlight the role of women (especially those providing leadership through Uganda's AIDS support organisation, TASO). But whether this faith is justified, or whether the more appropriate response should be to demand better policies from national governments and international organisations, is moot.

Conclusion

Ultimately one cannot avoid the cultural and political contexts which stymied HIV prevention efforts probably more emphatically than program design. As Caldwell (2002) argues, one of the key reasons why HIV prevention floundered in Africa was because of direct resistance on the part of those who did not want

[11] Neither the Malawian study (Helleringer and Kohler, 2007), nor a study of concurrency in four African cities Lagarde *et al* (2001), found a significant association between degree of concurrency and HIV prevalence. Lagarde *et al* attributed it to the fact that the study was done on a 12-month recall basis in the midst of the epidemic (so it is not an adequate basis for looking at the spread of HIV). The fact that their study was limited to four cities with greatly differing HIV rates is probably also a factor. Helleringer and Kohler attribute their lack of correlation between network density and HIV prevalence to confounding factors, but point to the serious risk for an epidemic in the area.

to change their sexual culture, and because most national leaders failed to show the necessary leadership in facilitating behaviour change (see also de Waal 2006).

Changing sexual behaviour has to come 'from below'. The transformative power of organic grass-roots organisation and mobilisation in the gay community in the North is widely recognised to have been the key ingredient in facilitating behaviour change. Certainly from its inception, UNAIDS recognised the importance of obtaining greater local 'ownership' of programs, of involving civil society and of tailoring interventions to suite local socio-economic and political realities (Caraël 2006). Both Norieen Kaleeba's TASO and the South African Treatment Action Campaign have been supported by WHO/UNAIDS. But this bold agenda had its own dangers – such as the difficulty of forging coherent policies through broad consultative processes and the fall-out from the inevitable failure to live up to the expectations of those participating in UN sponsored get-togethers.

Probably the most disappointing aspect of the unfolding story of the African AIDS epidemic is the lukewarm approach to addressing it adopted by most national governments. As Caldwell (2002), De Waal (2006), and Iliffe (2006) have argued, denial and political incentives not to challenge prevailing sexual cultures have created a deadly combination which allowed the AIDS pandemic to continue, largely unchecked. UNAIDS thus found itself devoting more time and energy than expected trying to advocate for greater national involvement. Perhaps the downward correction of HIV estimates that took place in November 2007 should, and could, have been done earlier (as charged by Chin).[12] But if there was a politically-inspired delay, it was most likely to have been caused by concerns that this would take the pressure off national governments – rather than by any venal concern to grab a greater share of existing development resources. After all the United Nations agencies and donors were well aware of the capacity constraints faced by existing interventions (see e.g. WHO 2006; JLI 2004, UNAIDS 2007)[13]. It is now common cause that unless more resources are allocated by donors and national governments to enhancing general capacity in domestic health care systems, the economies of scale with regard to further AIDS funding will be very low.

[12] This argument was picked up by others, see e.g. African Press International, 21 November 2007 "UNAIDS… Watch Media Information Note' Available on: http://africanpress.wordpress.com/2007/11/26/accused-of-misleading-international-community-unaids-director/

[13] See also UN General Assembly, Sixtieth session, Agenda item 45: Follow-up to the outcome of the twenty-sixth special session: Implementation of the Declaration of Commitment on HIV/AIDS (available on: http://data.unaids.org/pub/InformationNote/2006/20060324_HLM_GA_A60737_en.pdf

The challenge is to use local, national and international resources creatively and effectively to forge responses which make sense at grass-roots level. The standard universalising approach taken by international organisations – such as the ‘three ones’ demand that there be one national AIDS co-ordinating body, one national plan and one evaluation and monitoring system – should perhaps be re-examined. Both in terms of the analysis and recommended policy prescriptions, the lesson of recent literature is to know your local epidemic and to beware of strong claims about the primacy of any single approach.

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