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Poverty, Sex and HIV

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Abstract There is an ongoing debate about the relative importance of economic factors (notably poverty) and sexual behavior in driving the AIDS epidemic. This paper draws on relevant research and cross-country regression analysis to argue that the impact of economic determinants is dwarfed by contextual factors within Africa. The regression analysis suggests that controlling for per capita income, calories per capita and the ratio of female to male participation rates (none of which were statistically significant): being a Southern African country increases expected HIV prevalence 8.3 times; being in the rest of Sub-Saharan Africa 3 times; being a predominantly Protestant country 2.5 times; and being a predominantly Muslim country reduces expected HIV prevalence to 62% of the base case. Including the share of income going to the poor did not improve the model and was itself statistically insignificant. The analysis suggests that poverty may play a role in the HIV epidemic in some countries (and may well be a factor affecting the vulnerability of some people to HIV infection in all countries) but that its overall impact is dwarfed by social and behavioral factors.

Keywords HIV · Poverty · Sexual behavior · Africa · Cross-country regression

Introduction

There is an ongoing debate about the relative importance of economic factors (notably poverty) and sexual behaviour in driving the AIDS epidemic. Some accuse UNAIDS of paying too little attention to risky behaviour (e.g. Chin 2007; Epstein 2007; Pisani 2008) whilst others accuse of it overplaying sex and neglecting poverty (e.g. Stillwaggon 2006). UNAIDS has responded by defending its record and emphasising that the challenge is to 'know the local epidemic'—i.e., the specific socio-economic context and cultural factors that structure HIV risk in different settings—and craft interventions accordingly (De Lay and De Cock 2007; De Cock and De Lay 2008). This increasingly open-ended and pragmatic stance, however, does not resonate well with the literature stressing the centrality of poverty in driving the AIDS epidemic, particularly in Africa (e.g. Barnett and Whiteside 2002; Fenton 2004; Poku 2005, Stillwaggon 2006). For many people working and researching on AIDS, there is a strong presumption that socio-economic 'drivers' are fundamental.

This paper uses cross-country regression on HIV prevalence to explore the relative importance of poverty as a potential driver of the epidemic. The results suggest that contextual factors within Africa are probably more salient than economic factors and that the more nuanced and localised approach currently advocated by UNAIDS seems warranted.

Poverty and AIDS

The most extreme version of the 'poverty causes AIDS' argument is that by Stillwaggon (2000, 2002, 2006). She 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

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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 concludes 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 some evidence that people who are infected with parasites and other endemic diseases have an elevated immune response which may increase their susceptibility to HIV infection (e.g. Bentwich et al. 2000; Clerici et al. 2001; Borkow and Bentwich 2006; Fauci 2007; Sawers et al. 2008). But, while this points to the possible benefits of addressing co-infections, it is insufficient evidence to claim the *primacy* of poverty-related biological pathways in driving the HIV epidemic. As Borkow and Bentwich (2006: 608) observe in their review of the evidence, there have been no large-scale field studies to test the relationship between HIV susceptibility and helminth co-infection. The fact that Malawian patients infected with helminths (worms) were found in a subsequent study to be less, rather than more, likely to have HIV demonstrates the need for more caution in this regard (Hosseinipour et al. 2007).

On the issue of whether poor nutrition increases susceptibility to HIV infection, the evidence is thin and 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). But whiles this seemed suggestive of a poverty-related caloric shortfall as a determinant of HIV infection, no relationship was found between sero-conversion and socio-economic variables or blood vitamin levels.

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), but 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).

A recent study of malnourished children in Zambia has further undermined the 'malnutrition as cause of HIV disease' theory by showing that HIV-negative malnourished children had normal CD4 counts, whereas the HIV-positive malnourished children had below-normal CD4 counts and that these counts did not improve with better nutrition (Hughes et al. 2009).

In short, while it is possible that people living in poor conditions with inadequate public health services are at an elevated risk of HIV infection, the evidence is limited and contradictory. There is thus no good reason to assume that a relationship necessarily exists between measures of economic deprivation and HIV at an aggregate level.

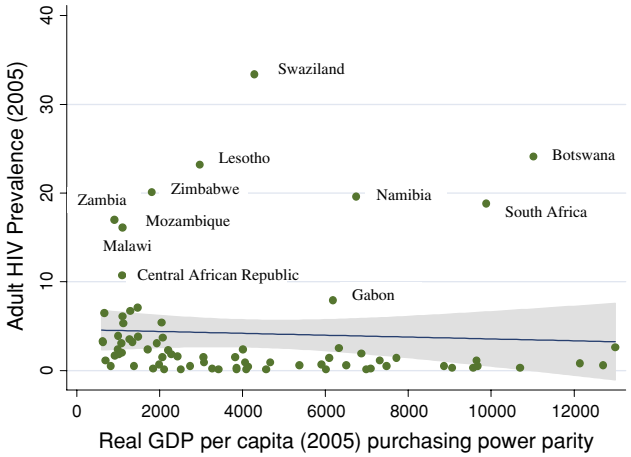

Fig. 1 HIV prevalence and GDP per capita (data from UNAIDS and the World Bank)

Figure 1 shows that there is no obvious relationship between the most basic development indicator—per capita income—and HIV prevalence. Subsequent regression analysis confirms this result (see Table 1 and also Zanakis et al. 2007). A quick inspection of the data rather 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 disproportionate effect on the estimated cross-country relationship between HIV prevalence and income inequality (Nattrass 2008).

Table 1 explores the relationship between HIV prevalence (as of 2005) and poverty more systematically, and in the context of other potential determinants, using ordinary least squares multiple regression analysis. HIV prevalence was logged in order to reduce the impact of outliers, to create a more normal distribution and to facilitate the interpretation of coefficients. The data were drawn from Englebert (2000) and: http://data.un.org/ (income share of the bottom quintile, labour force participation rates); http://ksghome.harvard.edu/~pnorris/Data/Data.htm (dominant religion (from CIA); http://faostat.fao.org (calories per capita); www.unaids.org (HIV prevalence, type of epidemic) and http://hdr.undp.org/en/statistics/ (GDP per capita).

Regression 1.1 shows that when controlling for per capita income and type of epidemic, average calories per capita are statistically significantly (and negatively) associated with HIV prevalence: a 1% increase in calories per capita is associated with a 2.9% decrease in HIV prevalence. However, the relationship between HIV prevalence

Table 1 Per capita GNP, inequality and adult HIV prevalence: seven regression models

Dependent variable: Log of Adult HIV Prevalence (2005)	1.1	1.2	1.3	1.4	1.5	1.6
Log of per capita GDP in 2005						
Coefficient (standard error)	0.344 (0.216)	0.061 (.257)	−0.867 (0.205)	−0.158 (0.188)	−0.232 (0.230)	−0.123 (0.194)
Log of calories per capita 2002/04	**					
Coefficient (standard error)	−2.852 (1.124)	−1.844 (1.342)	−1.056 (0.899)	−1.015 (0.898)	−0.206 (1.131)	−1.030 (0.969)
MSM/IDU epidemics	***	***	***	***	***	***
Coefficient (standard error)	−1.873 (0.309)	−1.881 (0.350)	−1.306 (0.291)	−1.093 (0.265)	−1.257 (0.312)	−1.131 (0.264)
Southern Africa			***	***	***	***
Coefficient (standard error)			2.575 (0.529)	2.114 (0.490)	1.729 (0.612)	2.092 (0.495)
Rest of Sub-Saharan Africa			***	**	**	**
Coefficient (standard error)			1.518 (0.538)	1.105 (0.483)	1.406 (0.558)	1.056 (0.487)
Predominantly muslim				*	*	
Coefficient (standard error)				−0.480 (0.264)	−0.551 (0.309)	−0.410 (0.283)
Predominantly protestant				***	***	***
Coefficient (standard error)				0.914 (0.276)	0.920 (0.325)	0.887 (0.275)
Log of the ratio of female to male participation rate						
Coefficient (standard error)				0.617 (0.425)	0.601 (0.447)	0.674 (0.423)
Log of income share bottom 20%		***				
Coefficient (standard error)		−1.115 (0.328)			−0.408 (0.301)	
State legitimacy (Englebert)						
Coefficient (standard error)						−0.086 (0.244)
Adjusted R^2	0.4305	0.5406	0.5805	0.6734	0.7173	0.6932
Number of observations	80	65	80	80	65	78

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

and per capita income is statistically insignificant—and remains so across all specifications.

Type of epidemic is captured by a dummy variable taking a value of one if the country has a predominantly concentrated HIV epidemic amongst men who have sex with men (MSM) or injecting drug users (IDUs) and zero if the country has a heterosexual epidemic. This is to account for cross-country variation arising from the fact that countries with more 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, MSM/IDU epidemics are expected to have HIV prevalence rates 15% of that found in countries with heterosexual epidemics. (The estimate of 15% is derived by taking the anti-log of the coefficient on the MSM/IDU dummy variable).

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, Grieg and Koopman 2003, Hallman 2004, 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, Potts et al. 2008). 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 Kretzchmar 1997a, b; Epstein 2007). As Shelton et al. observe:

> 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 (2005: 1058).

As the relationship between wealth and HIV is even stronger for women than men, Shelton et al. note that this

undermines the oft-made claim that it is poverty that primarily drives women to engage in risky sex: "it appears that both wealth and economic disadvantage, or at least the desire for economic advancement) play pivotal roles in HIV transmission (2005: 1058). Their suggestion has subsequently been supported by a 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, contextual factors probably shape whether it is the poor, or the not-so-poor, who are the most vulnerable to HIV.

According to the simple regression model 1.2, a 1% increase in the share of income going to the bottom 20% is associated with a decrease of 1.1% in HIV prevalence. Unfortunately, adding the share of income going to the lowest quintile to the model reduces the number of observations to 65 because of missing data. Even so, the regression suggests that across a large sample of countries both inside and outside of sub-Saharan Africa, greater relative poverty is associated with higher HIV prevalence (see also Drain et al. 2004). However, as shown in regression 1.5, this relationship disappears once regional and cultural variables are included as controls. (It is also worth noting that interacting income per capita and share of income to the poor suggests that the biggest impact of increasing income to the poor occurs in low income countries. However, the standard errors on the point estimates are enormous and including the interaction term in the model reduces the adjusted R-squared).

Regression 1.3 includes broadens the analysis to include two dummy variables; one for being a Southern African country (i.e. South Africa, Botswana, Lesotho, Namibia and Swaziland) 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: 113–114). 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 three cultural variables. A dummy variable for whether the country is predominantly Muslim and a dummy variable for whether the country is predominantly Protestant were included to capture the potential impact of religion on sexual behaviour given that Muslim societies are less tolerant of sexual freedom than other religions, whereas Protestant countries tolerate a greater degree of sexual freedom than other countries. According to Iliffe (2006: 48–56), the Muslim social order was a key factor contributing to the lower HIV prevalence in West Africa. However, there is also a biological reason for including these religious dummy variables because Muslims 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 (Langerhans' 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 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). A third cultural variable, the ratio of the female to male economic participation rate, was added as a proxy for the relative empowerment of women.

Note that including the two religious dummy variables and the female empowerment proxy increases the explanatory power of the model (indeed, this particular model has the best 'fit' of all—see Table 1) whilst rendering the economic explanatory variables statistically insignificant. More specifically, regression 1.4 shows that, controlling for per capita income, calories per capita and the ratio of female to male participation rates (none of which were statistically significant determinants): being a Southern African country increases expected HIV prevalence 8.3 times; being in the rest of Sub-Saharan Africa 3 times; being a predominantly Protestant country 2.5 times; and being a predominantly Muslim country reduces expected HIV prevalence to 62% of the base case. Regression 1.5 shows that including the proxy for degree of poverty makes no difference to the sign and statistical significance 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 (and may well be a factor affecting the vulnerability of some people to HIV infection in all countries) but that its overall impact at a country level appears to be 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 Potts et al. 2008) and Iliffe's assessment that poverty was not a major driver of the African AIDS epidemic. As Noreen Kaleeba (the founder of the AIDS support organisation (TASO) in Uganda) puts it, 'AIDS affects ordinary people'—i.e. not just the poor (quoted in Iliffe 2006: 63). 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 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 also makes 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 (2006), 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 systematic or reliable evidence to indicate that reducing poverty has the same effect (Bärnighausen et al. 2007; Gillespie et al. 2007). A recent randomised trial of the impact of providing small loans to poor women in South Africa found no effect on sexual behaviour or HIV incidence (Pronk et al. 2006).

Note that the above regression analysis does not attempt a full exploration of all potential determinants of HIV prevalence (as in Zanakis et al. 2007; Drain et al. 2004). The purpose was rather to interrogate the 'poverty causes HIV' claim and to suggest that we need to be open to the ways in which regionally specific cultural issues (including religion and sex) shape epidemics at the national and local levels—and to the ways in which HIV risk and vulnerability is present across the income distribution albeit to different degrees in different contexts.

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, chap. 5). This impact is obviously multifaceted and impossible to capture statistically. The only available measure is that by Englebert who developed a dummy variable indicating whether a country's past history of colonisation rendered the state 'legitimate' or not (see Table 2). regression 1.6 includes this variable, but the coefficient is small (being classified as 'legitimate' reduces HIV prevalence to 92% of the base case) and, in any event, is statistically insignificant.

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 et al. 2006, Kapiga et al. 2007; Corey 2007; Drain et al. 2004; Sawers et al. 2008) and has argued to be a key driver of the HIV epidemic in Africa (Smith and Robinson 2002; Oster 2005; Abu-Raddad et al. 2008). However, as the available data on HSV-2 varies greatly by coverage and year of assessment (Smith and Robinson 2002), it was not included here. Hence, any impact of higher HSV-2 in Africa on HIV prevalence would be picked up indirectly in the regressions by the African dummy variables.

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

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 Englebert (2000)

2000). Europeans are more 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 African-Americans 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 (2006: 133–157), however, regards the very notion of a risky African sexual culture as a racist construction invented by ethnographers. 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 tells us nothing about long-term *concurrent* sexual partnerships. As network modelling has demonstrated, and as is increasingly being recognised, concurrency drives HIV prevalence to a far greater degree than the number of sexual partners per se (Morris and Kretzchmar 1997a b; Kretzschmar 2000; Epstein 2008, Potts et al. 2008). Given that long-term concurrent sexual partnerships are common in Africa (see e.g. Caldwell et al. 1989, 1991; Poku 2005: 73–75; Epstein 2007; Konde-Lule et al. 1997; Orubuloye et al. 1991; Lagarde et al. 2001; Iliffe 2006; Hunter 2002, 2006; Parker et al. 2007; Helleringer and Kohler 2007; Mah and Halperin 2008; Mah 2008), it may 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.

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). Uganda's President Museveni campaign for 'zero-grazing' has been widely cited as a feasible African solution in that, as Epstein puts it, its real message was: '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' (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. But the fight remains an uphill struggle, not least because of resistance to behaviour change (Caldwell 2002) and failure of political leadership (de Waal 2006).

Conclusion

The evidence suggests that there is no systematic relationship between poverty and HIV, although malnutrition and economic vulnerability may well increase the risk of HIV infection in some contexts. Strategies to alleviate poverty, whilst valuable in themselves, are unlikely to be effective in combating the HIV epidemic. Rather, the evidence suggests that pragmatic, context-specific approaches which prioritise knowing the local epidemic and involving civil society organisations and people living with HIV are warranted.

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