Awareness and AIDS: A Political Economy Perspective[*]

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Abstract

Across African countries, prevention policies are unrelated to HIV/AIDS prevalence. Even when successful, they are often unstable or reversed. To explain these puzzles, we propose a simple political economy model that examines joint determination of prevention policies and the epidemic dynamics. Prevention affects both behavior and the perception of the role of policies in fighting AIDS. Behavioral changes induced by the policy, in turn, reduce the infection risk for sexually active agents, which creates political support for future policies. The two-way relationship between prevention policy and awareness generates two stable steady-state equilibria. The low-prevalence equilibrium is fragile. Reduced transmission rates have an ambiguous impact on prevalence rates as they also imply less active prevention policies. An empirical analysis of the determinants of public support for HIV/AIDS policies using the 2005 Afrobarometer shows that high prevalence rates translate into public support for prevention policies only in countries which carried out active prevention campaigns in the past. Our framework extends to a large class of public health policies under which awareness partly follows from the policies themselves.

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1 Introduction

The HIV/AIDS epidemic has had devastating effects on the lives of millions of people, particularly in Sub-Saharan Africa, where prevalence and the associated mortality rates are extremely high. While scientists have now a reasonable understanding of the medical aspects of the epidemic, its transmission mechanisms and its consequences, the knowledge of how societies have been responding to the HIV/AIDS epidemic is much more limited.[1]

Countries have reacted very differently to the epidemic, particularly in terms of prevention policies. Surprisingly, countries with high prevalence rates do not respond more aggressively than countries that are much less affected by the disease. For instance, Senegal developed an early proactive education AIDS program despite being barely affected by the disease (with a prevalence rate below 1 per cent in 2003), while countries like Zimbabwe and Swaziland, characterized by extremely high prevalence rates (24.6 per cent in 2003 and 42.6 per cent in 2004, respectively), showed a very limited response to the epidemic (Patterson 2006). One measure of the policy actions to combat the disease spread is the AIDS Program Effort Index (API) constructed by the USAID, UNAIDS, and WHO (USAID et al. 2003). It is a survey-based measure of effort put by national governments to combat HIV/AIDS. Figure 1 plots the API score in 2003 against the HIV prevalence rate among adults aged 15-49 two years earlier, for the countries for which the API score is available.[2] We note that the countries with the highest prevalence rates (over 10 per cent) have, on average, a slightly lower API score than the countries with relatively low prevalence rates.

[**Figure 1 about here**]

Even more puzzling is the fact that public support for prioritizing HIV/AIDS policies is also not related to the prevalence of the disease (De Waal 2006). In many countries where HIV/AIDS is pervasive, combatting the disease does not seem to be a political priority of the electorate. Figure 2 uses the Afrobarometer 2005 data to depict the relationship between regional prevalence rates (proxied by the share of respondents that personally know someone who died of AIDS) and the percentage of respondents in favour of an increase in the share of

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[1]See Barnett and Whiteside (2006) for a comprehensive review.

[2]Unfortunately, these surveys were not conducted after 2003.

the national budget allocated to combatting AIDS.[3] The results are striking: public support is unrelated to perceived prevalence.

[**Figure 2 about here**]

The coexistence of high prevalence rates and a low priority given to the disease by citizens supports the idea of HIV/AIDS 'denial' that several observers have noted (Whiteside et al. 2004).[4] As de Waal emphasizes: "If African voters are not concerned over HIV/AIDS, it follows that the politicians they vote into office will not be impelled to make AIDS a priority" (De Waal 2006: 8). Similarly, Bor (2007) argues that: "The failure of elected leaders to respond to AIDS may reflect a rational response to the demands of their constituents." (Bor 2007: 1598). However, this state of affairs is surprising as countries which developed an active AIDS policy (Philippines, Brazil, and Uganda) have also experienced a sharp fall in their HIV prevalence rates.

A third stylized fact concerns the evolution of AIDS policies over time. We often observe large changes in policies over time, and the direction of the change appears inconsistent with a coherent long-run plan of disease reduction. For instance, Uganda adopted active prevention policies very early. In 1986, the President Museveni launched a successful prevention campaign focused around the so-called ABC message ("Abstain, Be faithful, use Condoms") which led to a substantial reduction in prevalence rates during the 1990s (see Alsan and Cutler, 2010).[5] In 2003, however, the policy changed drastically by focusing only on abstinence, under the pressure of the U.S. PEPFAR aid program and with the support of evangelical churches and the First Lady, Janet Museveni. This change resulted in a fall in condom use and frequent shortages in condom supply.[6] By 2006, prevalence rates in Uganda were again on the rise (Patterson 2006; AVERT 2012).

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[3]Section 3 gives a detailed description of the data used. A similar picture emerges if we use naming AIDS as one of the top three government priorities, a measure of political support for combatting the epidemic.

[4]Denial has taken several forms, such as Mbeki's controversial attitude to azidothymidine (AZT), the public (and counter-factual) claim by the King Mswati II of Swaziland that 'HIV/AIDS is promoted by an individual in the manner he or she goes about his or her life. Otherwise, polygamy is not a factor' (as quoted in Patterson 2006: 21) or Arap Moi's public statement about condoms: 'As president, I am shy that I am spending millions of shillings importing those things' (quoted in Whiteside et al. 2004: 146).

[5]Observers trace Museveni's active involvement in combatting the epidemic to his grassroot legitimacy derived from his guerilla struggle as well as to his strong concern about national security, which he thought could be jeopardized by the AIDS epidemic (Boone and Batsell 2001; Patterson 2006).

[6]Social scientists and observers largely agree about the negative role that the religious authorities have

Similar sharp changes in prevention policies occurred in South Africa. In 1994, the government launched the national anti-AIDS plan which, though promising, remained largely ineffective. The second half of the 1990s was characterized by a number of initiatives and controversies (both around prevention and treatment policies) that did not provide a response to the epidemic. Confusion culminated in the early 2000s when the newly elected President Thabo Mbeki and his health minister blamed the most effective antiretroviral drug at the time (AZT) to be toxic and ineffective to prevent mother-to-child HIV transmission. The arguments about other treatments evolved in the context of a global questioning about the causes of HIV/AIDS (exemplified by the infamous promotion of beetroot and garlic consumption as effective prevention means). A serious policy against HIV/AIDS was launched only in 2003 with the National Plan for access to Anti-Retroviral Treatment (for detailed descriptions of the evolution of HIV/AIDS policies in South Africa, see Fassin 2007; Patterson 2006; AVERT 2012).

Finally, the fourth fact is that despite decades of exposure to the epidemic and several massive interventions, accurate knowledge about the disease remains generally poor and strongly varies across and within developing countries. An examination of the 2003 National Demographic Health Surveys of 2003 reveals that while in urban Ghana or Zambia, the percentage of adults who knew that condom use reduces the transmission was above 80%, this percentage was much lower in Nigeria, where only 64% of urban and 48% of rural adults were correctly informed (with 55% of the rural respondents wrongly believing that sharing a meal with an HIV-infected person increased the risk of infection) (see Dinkelman et al. 2006 and Folayan 2004). More surprisingly, in a recent survey in South Africa in 2008, less than half of the respondents knew both about the preventive effect of condoms and of reducing the number of sexual partners, even though more than 80% of them have been exposed recently to a HIV/AIDS campaign. Moreover, their degree of knowledge about the disease has decreased significantly over time (HSRC 2010).

These facts call for a political economy perspective to HIV/AIDS policies. The role of

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played in reducing the effectiveness of prevention campaigns by insisting on abstinence and being strongly opposed to sexual education in middle schools, to specific actions in favor of sex workers or drug users, and to the promotion of condom use (see, for instance, Whiteside et al. 2004; Headley and Siplon 2006; Lieberman 2007; Folayan 2006; Low-Beer and Stoneburner 2004).

civil society, political leaders and religious authorities has been emphasized by social scientists in numerous case studies (see, e.g. Headley and Siplon 2006; De Waal 2006; Densham 2006; Boone and Batsell 2001). In public health literature, Nathanson (1996) has stressed the importance of public support for the implementation of health policies, as exemplified by the role of the political participation of the rural poor in Kerala or of the turn-of-the-century radicalized proletariat in Italy (Nathanson 2006: 613). That study also points out the existence of feedback mechanisms whereby the popular mobilization results from government health policies. In a similar vein, Patterson (2006) suggests the emergence of a low level equilibrium in AIDS policies whereby 'low public saliency on AIDS may enable governments to do the minimum to fight AIDS; government's inaction may fuel low public concern about the disease' (Patterson 2006: 65). However, to the best of our knowledge, there exist no macro-level analyses that unify these elements and investigate systematically the determinants of HIV/AIDS prevention policies.

In this paper, we propose a simple political economy model that examines how HIV/AIDS prevention policies and the dynamics of the epidemic are jointly determined. Our approach relies on two main mechanisms. First, public policies affect voters' awareness about the disease and the role of public authorities. By releasing more information about the issue, prevention policies also affect popular concern about the disease. Second, the behavioral changes induced by the policy reduce the risk of infection for sexually active agents. This positive externality motivates the demand of aware voters for public intervention. Because of the two-way relationship between prevention policy and awareness, our model exhibits two stable steady-state equilibria: one with a high share of aware citizens and a low HIV prevalence, and the other with a low awareness and a high prevalence. In the analysis of the short run dynamics of the economy, we find that the low prevalence equilibrium is fragile, as a sufficiently large exogenous drop in prevalence or in awareness leads to a fall in political support for further government spending on prevention, making the economy drift away towards the high prevalence equilibrium. Moreover, the low prevalence equilibrium fails to be stable when the equilibrium policies are too reactive to changes in the prevalence rates. We then investigate the role of NGOs and foreign donors in prevention. In order to have permanent reduction effects on HIV prevalence, these outside interventions should be

sufficiently large and stable to allow for a self-sustaining political support. Finally, we also find that falling transmission rates can increase prevalence rates as they also imply less active prevention policies in equilibrium.

We then conduct an empirical investigation of the determinants of citizens' support for HIV/AIDS policies using the 2005 Afrobarometer opinion survey. In particular, we find that the perceived high prevalence rates of the disease in the region are not, by themselves, sufficient to create public support. It is only in those countries in which public prevention policies have been effectively implemented that higher prevalence rates translate into larger political support for public intervention. This evidence illustrates the crucial role of awareness in linking risk exposure, political opinions, and past prevention policies.

Taken together, these results may explain both the low correlation between HIV/AIDS prevalence and prevention policies across African countries, but also the observed instability of effective prevention policies over time. More generally, our model indicates a new approach to the political economy of public health policies, such as water and sanitation policies or deworming policies for which large externalities are present (see Miguel and Kremer 2004 and Baird et al. 2011). Our results also suggest that the cost-benefit analyses of policy interventions of several key HIV/AIDS microeconometric studies should be re-interpreted by explicitly taking into account the feedback effect of public support on equilibrium policies.

The existing economic literature on HIV/AIDS prevention is essentially empirical and focuses on the behavioral impact of information.[7] There is a large consensus that access to information has a major impact on risky sexual behavior. For instance, among HIV-infected individuals in Kenya, Goldstein et al. (2011) show that the impact of exposure to a full information program largely dominates that of access to a treatment on condom use by the patients. However, the impact of HIV/AIDS information strongly varies with individual characteristics, such as the level of education (Dupas 2011b, De Walque 2007) and gender, as women - even when fully informed - have a much weaker influence than their male partners over the choice of sexual practices and prevention methods (Varga 1997, Dinkelman et al.

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[7]There is also a more recent literature focussing on the effects of access to treatment for HIV-infected patients, which stresses the role of frequent reminders for continued administration of the medicine (Pop-Eleches et al. 2011) or the quality of the health center for health outcomes (Goldstein et al. 2012).

2006, Yamauchi 2008). The precise nature and the transmission channels of the information also matter. For instance, Dupas (2011a) shows that information on general prevalence is much less effective than information on relative risks ('older men are much more infectious') to generate behavioral change.[8] Similarly, Behrman et al. (2007) stress the role of social and family networks in providing detailed information about the level of HIV/AIDS in the local community, the infectious status of potential sexual partners or the acceptability of prevention practices, and thereby in shaping behavioral changes. In Malawi, Yamauchi et al. (2008) also find that an early exposure to prime-age deaths of a relative has a strong impact on sexual behavior (as measured by condom use or extra-marital affairs) and the resulting infectious status of younger women in the family.[9]

In an early attempt, Kremer proposes a simple behavioral model of AIDS epidemic where agents differ by their risk status and choose the intensity of their sexual activity. In this framework, high activity agents may respond to higher risk by further increasing their exposure, which allows for multiple equilibria. However, prevention policies are exogenous in this model. In a related paper, Oster (2005) proposes a simulation model of the epidemics and finds that small differences in transmission rates can lead to large differences in long run prevalence of the disease. Consequently, observed differences in prevalence rates across African countries are to a large extent explained by small differences in sexual behavior. In this paper, we argue that transmission rates and sexual behavior responds to prevention policies and, as a result, differences in prevalence rates can be traced back to endogenous political determinants. More recently, Greenwood, Kircher and Tertilt (2010) show that, in a general equilibrium model of matching, HIV/AIDS policies, if too moderate, may backfire and increase the level of the epidemic through the behavioral response they induce. More active policies, on the contrary, have the intended effects. Our paper also suggests that prevention policies should be large enough to have permanent effects; however, our mechanism is completely different from theirs, as they highlight the importance of general equilibrium (aggregation) effects, while we focus instead on the two-way relationship between prevention

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[8]Interestingly, Dupas (2011a) finds that, when provided with information on relative risk, female teenagers do not reduce their number of sexual partners, but adopt instead younger (less infectious) partners.

[9]For more on the role of social learning in the general context of health, see Dupas (2011b).

policies and endogenous political support.

This paper also relates to two strands of literature in political economy. Several papers (Piketty 1995; Alesina and Angeletos 2005; Benabou and Tirole 2006) have investigated the two-way interaction between redistributive taxation policies and voters' beliefs about the relative importance of effort and luck in determining individual outcomes and income inequality. These models, like ours, exhibit multiple steady-state equilibria because of the feedback effect of these policies on voters' beliefs. Our analysis differs in two respect. First, the nature of the policy we focus on, public health instead of redistributive taxation, is entirely different and the formation of beliefs is not at the heart of our analysis. Second, in our model, the policy affects both the individual (sexual) behavior and the individual perceptions about the effectiveness of the policy which determines public support in its favor. The second strand of literature to which our paper relates are the political-economic analyses of resistance to reforms (Fernandez and Rodrik 1991, Brainard and Verdier 1997, Coate and Morris 1999). These papers (in particular, Brainard and Verdier 1997 and Coate and Morris 1999) explain why certain economic policies (e.g. protectionist tariffs) are persistent: once in place, they create incentives for some economic agents to take actions that induce them to oppose future changes in the policies. This gives rise to long run inefficiencies in equilibrium outcomes. The main difference in our model is that the policy does not change the incentives of voters but instead informs them about the potential role and the consequences of policies.

The rest of the paper is organized as follows. Section 2 presents the theoretical model, and its main results. Section 3 presents some empirical evidence supporting the mechanisms highlighted by the model. Section 4 discusses some of the assumptions and suggests possible extensions. Section 5 concludes.

2　The model

2.1　The characteristics of the agents

We consider an overlapping-generations model. In each period $t = 0, 1, 2, ...,$ the population is composed of two types of atomistic agents: old (O) and young (Y). The size of each cohort is constant and equal to 1. Each individual lives for two periods, as a young agent in the

first and as an old agent in the second. At the end of the first period, each young agent has exactly one child, and the total population is therefore constant. Young agents work and get a fixed income which we normalize to 1. Old agents do not work.

Agents also differ in their attitudes regarding government prevention policies. A proportion $1 - \gamma$ of agents is conservative (c): these agents engage only in safe sex and are opposed to any government action towards HIV/AIDS. All conservative parents have conservative children. The remaining fraction of agents, $\gamma \in (0, 1)$, are either aware (a) about the existence of a simple costless technology effective in reducing the spread of the disease (i.e., safe sex practices), or are unaware (u) about it. In each period t, the proportion of aware young agents, denoted by q_t^y, is determined endogenously.

Three mechanisms determine the level of awareness among the young agents. First, a proportion of young agents become aware by inter-generational transmission, as aware parents and elders inform their children about the existence of prevention measures with respect to the disease. The probability of such transmission from the old aware population to their children is exogenous and given by $\chi \in (0, 1)$. Second, a fraction of young agents are made aware by the information campaigns carried out by NGOs or external donors, which is also exogenous in our model. We denote this proportion q_{Nt}. Third, a proportion q_{Pt} of young agents get informed through government campaigns.[10] As a result, the proportion of aware young agents in period t is:

$$q_t^y = \chi q_{t-1}^y + q_{Nt} + q_{Pt}. \tag{1}$$

The unit cost of the government information campaign is p. The information campaign is financed by a lump-sum tax, τ_t, on all working agents and the government budget constraint is given by:

$$\tau_t = p q_{Pt}. \tag{2}$$

2.2 The transmission of the epidemic

We now describe the transmission mechanisms of the epidemic (for a related approach, see Kremer 1996 and Oster 2005). At the end of each period, each agent is either healthy (H)

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[10]For simplicity, we are implicitly assuming that the NGO and government campaigns are perfectly targeted to unaware non-conservative agents.

or sick (S). The disease gets transmitted via sexual contact. In the absence of preventive measures, the transmission rate from a sick partner to a healthy one is equal to 1. Aware agents are willing to adopt the safe-sex technology (e.g., condom use). If both partners are aware, the risk of transmission is zero. If only one of the partners is aware, the transmission rate is $1 - \Phi$, with $\Phi \in (0, 1)$, as the choice of sexual practices is negotiated between the two partners and one partner cannot on her own fully protect herself from the risk of infection.

At the beginning of their life, agents are healthy.[11] The first source of infection for young agents first is through sexual contacts with the older generation. Young aware agents abstain from having sex with older partners. By contrast, young unaware agents have sex with a member of the old generation with probability $\theta \in (0, 1)$. The intergenerational transmission of the disease is given by the following equation:

$$\alpha_t^{y,a} = 0, \tag{3}$$

$$\alpha_t^{y,u} = \theta \left[q_{t-1}^y (1 - \Phi) \alpha_t^{o,a} + (1 - q_{t-1}^y) \alpha_t^{o,u} \right], \tag{4}$$

where $\alpha_t^{i,j}$ represents the probability of being sick for an agent of age $i \in \{y, o\}$ and awareness type $j \in \{a, u\}$. The expression in square brackets in (4) is the per-contact probability of disease transmission from the older generation. $\alpha_t^{o,a}$ is the prevalence of the disease among the old aware agents, the probability that a young unaware agent enters a match with such a partner is $q_t^o = q_{t-1}^y$, and the transmission rate in this match is $1 - \Phi$. Similarly, $\alpha_t^{o,u}$ is the prevalence rate among old unaware agents, and the probability of entering a match with this type of agent is $1 - q_{t-1}^y$, with a transmission rate equal to 1.

The second source of infection comes from sexual relations within the young generation. These relations occur after all sexual contacts with the older generation have been made and $\alpha_t^{y,u}$ young agents have been infected. We assume that non-conservative young agents have a sexual relation with one randomly chosen member of this generation. The probability of infection for a young agent is then given by:

$$\Pr(S_{t+1}^{o,a}) = \alpha_{t+1}^{o,a} = \alpha_t^{y,u}(1 - \Phi)(1 - q_t^y), \tag{5}$$

$$\Pr(S_{t+1}^{o,u}) = \alpha_{t+1}^{o,u} = \alpha_t^{y,u} + (1 - \alpha_t^{y,u})\alpha_t^{y,u}(1 - q_t^y), \tag{6}$$

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[11]We therefore abstract here from the possibility of mother to child transmission. We we will return to the implications of this particular mechanism in the discussion section.

where $\Pr(S_{t+1}^{o,u})$ is the probability of being sick in next period. The first equation describes the probability of infection for an aware young agents. This agent is healthy before having sex with a member of her generation. The only channel through which she can be infected is by entering into a relation with an unaware sick partner of the same age, which occurs with probability $(1 - q_t^y)\,\alpha_t^{y,u}$, the transmission rate being given by $1 - \Phi$. The second equation describes the same process for an unaware young agent. As described above, she can first be infected through inter-generational contact. Additionally, she can also become infected by having a sexual relation with a unaware sick partner of her generation, which occurs with probability $(1 - q_t^y)\alpha_t^{y,u}$ (and a transmission rate equal to 1).

The health status of an agent is revealed only when he becomes old. We assume that, compared to being sick, healthy agents, when old, enjoy a utility gain equal to $\Delta > 0$. The ex ante utility of an agent of type $i \in \{a, u, c\}$ is given by:

$$U_t^i = v(1 - \tau_t) + \delta[\left(1 - \Pr(S_{t+1}^{o,i})\right)(1 + \Delta)\Omega + \Pr(S_{t+1}^{o,i})\Omega], \tag{7}$$

where the first term represents the utility of a young agent and $(1 - \tau_t)$, the net income available for his consumption, $\delta < 1$ is the discount factor and the term in the square bracket is the expected utility of an old agent, with Ω being his (exogenous) utility level when sick.

Before describing the political equilibrium, it is useful to review the timing of the different events. For a young agent:

- The agent first inherits the conservativeness or awareness from the older generation;

- All young agents work and produce income.

- The government prevention policy is decided by majority voting, with only the young having the right to vote.

- NGOs and the government inform q_{Nt} and q_{Pt} of young agents, respectively.

- Unaware young agents engage in sex with the older generation (intergenerational transmission).

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- All young agents are randomly matched into couples and have a sexual relation (intra-generational transmission). Each young agent has one child.

The within-period timing of events for the old agent is as follows:

- With probability θ, the agent has a sexual relation with an unaware member of the young generation.

- The old agent learns her health status.

- Old agents neither work nor vote. If she is healthy, she enjoys a utility level of $(1+\Delta)\Omega$; if she is sick, her utility level is Ω.

2.3   Policy preferences and the political process

We now examine the policy preferences of the young agents. Conservative and unaware agents prefer no public policy and zero taxes, as the policy is viewed as bringing no benefits to them. By contrast, aware agents internalize part of the benefits generated by a public prevention policy. This is because the risk of being infected by an unaware sick partner is strictly positive and, therefore, increasing the proportion of aware agents in the population reduce the risks of transmission. Thus, aware young agents, who are initially healthy, are willing to pay taxes to reduce these risks.

More precisely, the ex ante utility of an aware agent is given by:

$$U_t^a = v(1 - pq_{Pt}) + \delta[(1 - \alpha_{t+1}^{o;a})(1 + \Delta)\Omega + \alpha_{t+1}^{o;a}\Omega], \tag{8}$$

where $\alpha_{t+1}^{o;a}$ is described by (5). Using (1) in (5), and maximizing (8) with respect to the policy variable q_{Pt}, the first-order condition yields the preferred level of prevention policy for the aware voters:[12]

$$pv'(1 - pq_{Pt}^*) = \delta\Delta\Omega\alpha_t^{y,u}(1 - \Phi). \tag{9}$$

The left-hand side of (9) describes the marginal cost of a larger government policy, which involves higher taxes. The right-hand side describes its marginal benefits, as $\alpha_t^{y,u}(1 - \Phi)$

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[12]The second-order condition is trivially satisfied. In the following, for the sake of notational simplicity, we focus on interior equilibria.

represents the reduction in the risk of infection. The marginal benefit of the government policy increases with the fraction of unaware sick agents in the young population, $\alpha_t^{y,u}$, and decreases with Φ, which measures the efficiency of unilateral prevention measures. In this sense, private and public solutions to prevent transmission act as substitutes.

Given these preferences, the policy outcome is fully determined by the type of agents constituting a majority in the electorate. In one case, unaware and conservative voters constitute the majority and enforce the zero-prevention policy. In the other case, the initially aware voters are the majority and support the prevention policy described by equation (9)). This policy is implemented if and only if:

$$\gamma \chi q_{t-1}^y \geq (1 - \gamma) + \gamma(1 - \chi q_{t-1}^y), \tag{10}$$

or iff:

$$q_{t-1}^y \geq \frac{1}{2\gamma\chi}. \tag{11}$$

A few important points should be noted about the mechanism underlying the equilibrium. First, awareness gets transmitted across generations through families and networks. Being an unaware young citizen implies two negative effects on the overall health conditions in the economy. On the one hand, it implies the transmission of ignorance to the next generation, thus reinforcing future public opposition to a prevention policy. On the other hand, it also increases inter-generational transmission of the disease, via the risk of early infection by the older generation. Second, government policies do not affect current voters: the political mechanism is such that the young voters made initially aware by their parents in period t transmit (imperfectly) this attitude to their children who then vote in period $t + 1$.

In the following, we assume that the inter-generational transmission of awareness is not strong enough to guarantee that, even with full awareness among non conservative agents in period $t - 1$, i.e. $q_{t-1}^y = 1$, the majority threshold will be satisfied in period $t + 1$ in the absence of a policy or of NGO interventions in period t. Using (1) and (11), this assumption can be written as:

$$\chi^2 \gamma < \frac{1}{2}. \tag{12}$$

2.4   Low and High prevalence equilibria

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We now investigate the existence of steady-state equilibria, which involve a constant level of prevalence, α, and awareness, q^y. For simplicity we assume that NGOs conduct campaigns of identical extent in each period:

$$q_{Nt} = q_N \quad \text{for all } t.$$

Using (5)-(6), we can rewrite (3)-(4) in period $t+1$ as

$$\alpha_{t+1}^{y,a} = 0, \tag{13}$$

$$\alpha_{t+1}^{y,u} = \theta \left[(1 - q_t^y)\alpha_t^{y,u} + (1 - q_t^y)^2(1 - \alpha_t^{y,u})\alpha_t^{y,u} + q_t^y(1 - q_t^y)(1 - \Phi)^2\alpha_t^{y,u} \right]. \tag{14}$$

Solving (14) for the steady-state interior values yields:

$$\alpha^{y,u} = 1 - \frac{1}{\theta(1 - q^y)^2} + \frac{1 + q^y(1 - \Phi)^2}{(1 - q^y)} = \alpha(\overset{-}{q^y}). \tag{15}$$

In this last expression, a higher fraction of young aware agents, q^y, reduces the steady state level of prevalence: first, aware agents are not infected by the older generation and, second, aware agents use preventive measures in their sexual relations which slows down transmission of the disease within the young generation.

In the following, we assume that the utility function $v(.)$ is logarithmic. The first-order condition in the preferred policy, (9), becomes:

$$q_P^y = \frac{1}{p} - \frac{1}{\delta \Delta \Omega (1 - \Phi)\alpha^{y,u}} = q(\overset{+}{\alpha^{y,u}}), \tag{16}$$

where q_P^y is a positive function of current prevalence among the young agents.

Finally, the majority needed to support this policy in the steady-state is given by (11):

$$q^y \geq \frac{1}{2\gamma\chi}. \tag{17}$$

These three last expressions, (15), (16), and (17), jointly determine the steady-state equilibria of the model and figure 3 represents these equilibria graphically. We thus have:

Proposition 1 *There are two steady-state equilibria in the economy: one with zero policy, zero awareness (in the absence of NGO activities), and high prevalence of the disease in*

the population, and the other with positive policy, high awareness, and low prevalence of the disease.[13]

[**Figure 3 about here**]

To the left of the majority support line (17), aware voters constitute a minority and the equilibrium policy reflects the preferences of conservative and unaware voters and is set to zero. The dynamics of the spread of the disease is determined by (15) and, in the absence of NGO interventions, the stationary equilibrium corresponds to the highest point on the "AIDS epidemics" line. The equilibrium level of prevalence is given by $\alpha^{y,u} = 2 - \frac{1}{\theta}$ (for $\theta > \frac{1}{2}$ and $q_N = 0$), and $q_P^y = q^y = 0$. In this equilibrium, the absence of a public prevention policy implies a strong inter- and intragenerational transmission of the disease, which leads to high prevalence rates. As awareness vanishes, it cannot be transmitted across generations and future public support for the policy is also non-existent.

To the right of the majority support line (17), the steady-state political economic equilibrium must satisfy two conditions. Since the aware voters constitute a majority, the size of the government policy must reflect the preferences of the aware voters, for any level of the disease in the population. Thus, the equilibrium point must lie on the green ("preferred-policy") line, described by the equation (16). Additionally, the dynamics of the epidemic must converge towards its steady-state level described by the equation (15). Graphically, the equilibrium must also lie on the blue ("AIDS epidemics") line. Given that the relations (15) and (16) are both monotonic, there is only one intersection point, which simultaneously satisfies both relations, and corresponds to the low prevalence stationary equilibrium. Intuitively, as aware voters are sufficiently numerous to constitute a majority, the government implements active prevention campaigns, which reduce both the inter- and the intragenerational transmission of the disease in the population but also increase the proportion of aware agents in the population. The inter-generational transmission of awareness guarantees the existence of a strong public support for the policy in the future.

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[13]While the existence of the high prevalence equilibrium is always guaranteed, the low prevalence equilibrium requires a number of restrictions on the parameter values (in particular, one needs $\gamma\chi > \frac{1}{2}$) that we omit here for the sake of expositional simplicity. There also exists a third steady-state equilibrium with zero prevalence. However, it is always unstable, so we do not consider it in the discussion.

The co-existence of these two equilibria can help explaining why countries have reacted so differently when confronted to the HIV/AIDS epidemic. It also helps to understand why many African countries with high prevalence rates have failed to implement active prevention policies. We now show that the low prevalence equilibrium is particularly fragile.

2.5 The fragility of the low prevalence equilibrium

In the following, we assume that the utility function $v(.)$ is logarithmic and that NGOs are absent, so that $q_{Nt} = 0$. First, the low prevalence equilibrium is not always locally stable (the formal details are given in the Appendix). The stability of the equilibrium critically depends on the responsiveness of the preferred policy to changes in prevalence rates, $\frac{\partial q_P^y}{\partial \alpha^{y,u}}$. If this responsiveness is moderate enough, the economy smoothly converges towards the long run low prevalence equilibrium following a small exogenous change in awareness or prevalence. (This holds for a large set of parameter values.) However, when responsiveness is large, public support may over-react to a small increase in prevalence rates by enforcing large increases in prevention policies. This itself may entail an even larger (in absolute value) reduction in next period prevalence rates, and the economy quickly spirals away from the low prevalence equilibrium to a zero policy equilibrium, through increasing fluctuations in policies and prevalence rates. This instability is illustrated in Figure 4 below.

[**Figure 4 about here**]

We now show that the low prevalence equilibrium is also not globally stable:

Proposition 2 *Under assumption (12), the low prevalence equilibrium is not globally stable, as a sufficiently large drop in prevalence or in awareness diverts the economy toward the high prevalence equilibrium.*

Proof. *Using equations (1), (11) and (9), the majority threshold in period $t + 1$ is not satisfied if:*

$$q_t^y = \chi q_{t-1}^y + \frac{1}{p} - \frac{1}{\delta \Delta \Omega (1-\Phi) \alpha_t^{y,u}} < \frac{1}{2\gamma\chi} \quad or \quad \left(\chi q_{t-1}^y - \frac{1}{2\gamma\chi} \right) + \left(\frac{1}{p} - \frac{1}{\delta \Delta \Omega (1-\Phi) \alpha_t^{y,u}} \right) < 0.$$

In this last expression, the first bracketed term is strictly negative by assumption (12), while the second term under brackets can be set arbitrarily low, while still positive, with low enough

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values of $\alpha_t^{y,u}$. For such values, the majority threshold will not be satisfied in the following period, and prevention policies will not be adopted by a majority in all future periods. If awareness in period t drops exogenously such that (11) is not satisfied, the second part of the proposition trivially follows. ∎

A large exogenous fall in prevalence rates induces a large drop in the preferred policy which, if large enough, implies in the next period that the fraction of aware voters is too low to constitute a political majority. The economy is then trapped in a trajectory of zero-policy equilibria towards the large prevalence steady-state equilibrium. A similar phenomenon occurs for large falls in awareness levels.

Finally, when the equilibrium level of awareness is closer to the majority threshold, the low prevalence equilibrium may cease to exist altogether for small enough changes in the parameters:

Proposition 3 *Under assumption (12), the low prevalence equilibrium fails to exist (i) for a large enough decrease in the transmission of the disease (lower θ or higher Φ), in the transmission of awareness, χ, or in the utility premium associated with health, Δ, or (ii) for a large enough increase in the share of conservative agents (lower γ) or in the unit price of the policy, p.*

The proof is omitted as it is similar to the proof of Proposition 2. Using Figure 3, the changes described in the proposition correspond to either the rightward shift in "Majority support" line (lower χ or γ), the leftward shift in the "Preferred policy" curve (higher p, lower Δ, or higher Φ), or the downward shift in the "AIDS epidemics" curve (lower θ or higher Φ). Under assumption (12), any of these changes causes the disappearance of the low prevalence equilibrium.[14]

The economic intuition for this result is relatively straightforward. For instance, lower transmission of awareness across generations or more conservatives in the economy imply that

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[14]In a recent paper, Deaton et al (2010) finds, on the basis of opinion surveys, that the subjective well-being losses attached to the consequences of AIDS in Africa are low. If confirmed by future studies, these results suggest little priority in public opinion to develop AIDS policies, and therefore that the 'low prevalence' equilibrium may not always exist. As discussed below, this may call for an increasing role of NGOs and external donors in prevention policies.

the proportion of aware members of the old generation should be correspondingly higher in order to generate enough aware voters in the next generation. If, the preferred policy is such that the number of aware voters is close to the majority threshold, the number of aware voters in the next generation becomes too small and the low prevalence equilibrium disappears. Similarly, a higher unit price of the policy, a lower utility premium associated with health or lower transmission rates of the disease reduce the marginal benefits of the policy. The resulting fall in the policy may reduce the number of aware voters of the next generation below the required threshold.

To sum up, the low prevalence-active policies equilibrium is fragile in three respects: (i) it may not be locally stable if policies over-react to changes in prevalence, (ii) it is not globally stable, so that large enough changes in the environment move the economy towards the zero-policy equilibrium and (iii) the existence of the equilibrium is not guaranteed, as large enough changes in the relevant parameters may cause the low-prevalence equilibrium to disappear. These results provide some light on the third stylized fact discussed in the introduction, where we described the variability of policies and prevalence over time in some countries. In the case of Uganda, the decline in HIV prevalence in the early 2000s is partly due to the success of the prevention policies (in particular the widespread use of condoms and the reduction in the number of partners), but also, as Oster (2010) shows, to the reduction in the mobility of truck drivers caused by the fall in exports.[15] Our model predicts that if this drop in prevalence is sufficiently high, the next-period awareness campaigns will be too small to guarantee the majority needed to keep the economy in the prevention policy equilibrium.

In fact, the subsequent rise in HIV prevalence in Uganda can also be explained by this reduction in the political support for awareness campaigns. Mutabaazi (2006) argues that the reason why the prevalence is picking up again is the current reduction in the emphasis on condom use in the ABC campaign, in favor of the first two components (see also Cohen et al. (2005) and Cohen and Tate (2006)). Moreover, the author cites the Uganda AIDS com-

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[15]There isn't a systematic relationship between HIV infections and income growth, however. For instance, Dupas and Robinson (2011) in the case of Kenya shows how income falls have increased occasional prostitution and the adoption of risky practices, which led to increases in infection rates.

mission report that notes the decreasing concern in the population towards AIDS epidemic. One interpretation of this sequence of events is therefore that the complacency caused by the early reductions in the disease prevalence (due to drops in truck driver mobility) has allowed ABC policy opponents to obtain the *de facto* majority and remove a key component of the policy. This change led to increases in new infections. Unless NGOs and foreign donors put additional pressure to re-balance the ABC campaign, Uganda is likely to draw back to the high prevalence, zero-policy equilibrium.

Another implication of the above results is that, if the economy is in the high prevalence equilibrium, NGOs and external donors can play two distinct and important roles through their prevention campaigns. The first one is to divert the economy away from the high-prevalence towards the low-prevalence equilibrium, if it exists. These external interventions must be large enough to have a permanent effect, by letting the low prevalence equilibrium to emerge and prevention policies to become self-sustaining. If too limited, the awareness created by these interventions does not allow a sufficiently strong public support, and the economy will converge back towards the high prevalence equilibrium. The second role of external interventions is to guarantee a permanent minimal level of prevention policy which allows for both the existence of a low prevalence equilibrium (where it fails to exist) and its stability. This calls for campaigns that are stable over time, even if more limited in size.

2.6 The impact of transmission rates

In this section, we focus on the low prevalence equilibrium and investigate the role played by transmission rates.[16] As stated above, infections can occur either through sexual contact with the older generation, which occurs with probability θ, or through sexual contact with an infected member of the same generation, for which private prevention measures reduce the risks of transmission with probability Φ. These two avenues of disease transmission have a radically different impact on long run prevalence rates:

Proposition 4 *An increase in the inter-generational transmission of the disease, θ, increases both prevalence and awareness in equilibrium. An decrease in the effectiveness of*

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[16]The corresponding comparative statics on the high prevalence equilibrium are straightforward and therefore omitted here.

private prevention, Φ, increases the equilibrium awareness but has an ambiguous impact on prevalence.

Proof. *An increase in θ directly affects equation (15), and therefore leads to higher $\alpha^{y,u}$, and through equation (16) to a higher q^y. A decrease in Φ directly affects both equation (15) and (16), which leads to an ambiguous impact on the equilibrium level of $\alpha^{y,u}$.* ∎

An increase in sexual relations between generations increases the prevalence of the disease for all levels of awareness, as the disease gets transmitted more frequently from the old to the new generation. The equilibrium policy (which reflects the preferences of the aware citizens) increases in response to these changes in prevalence. This effect is however never large enough to offset the rise in prevalence so that, at the new equilibrium, prevalence and awareness are larger. These changes can be easily illustrated with the help of Figure 3, as an upward shift in the 'AIDS epidemic' curve. The impact of private prevention is different. A fall in private protection (a lower Φ) increases the risks of transmission both inter- and intra-generationally, which corresponds, as above, to an upward shift in the 'AIDS epidemic' curve. However, it also leads to an increase in the preferred prevention policy, as the marginal benefit of the policy increases (resulting in a rightward shift of the 'Preferred policy' curve). Equilibrium awareness unambiguously increases. The effect on prevalence is unclear however, as the two effects, the higher transmission rates and the stronger prevention efforts, go in opposite directions. This result calls into question the expected success of policies exclusively targeting transmission rates, such as programs for male circumcision, as the potential feedback effect on the desired level of prevention policies may, in equilibrium, result in a net increase in prevalence rates.

3 Empirical analysis

In this section, we use our theoretical model as a guide for an empirical investigation of the determinants of political support for anti-HIV/AIDS policies in Sub-Saharan African countries. The absence of good time-varying measures of prevention policies for a sufficiently large set of countries does not permit to test our model directly, and the correlations that we document cannot be interpreted as causal ones. Nevertheless, we are still able to investigate

whether the empirical patterns of political support are in line with the main predictions of our theory.

3.1   Data and descriptive statistics

Our principal data source is the Afrobarometer data, a large-scale individual-level cross-national survey of public attitudes in a broad set of political, economic and social issues, conducted jointly by the Department of Political Science at Michigan State University, the Institute for Democracy in South Africa, and the Ghana Center for Democratic Development (see Bratton et al. 2006, for a detailed description). For reasons explained below, we use Round 3 of this survey which covered over twenty-five thousand respondents in eighteen countries (Benin, Botswana, Cape Verde, Ghana, Kenya, Lesotho, Madagascar, Malawi, Mali, Mozambique, Namibia, Nigeria, Senegal, South Africa, Tanzania, Uganda, Zambia, and Zimbabwe).

To measure individual policy support for anti-HIV policies, we use three different measures. The first is a binary variable which takes value 1 if the respondent prefers government spending on HIV/AIDS to be increased (and 0 otherwise). The second is a binary variable taking value 1 if the respondent names AIDS among the top three priority issues for the government (out of twenty-one possible ones). The third is a more refined version of the second measure and takes into account the intensity of those preference. This measure takes value 3 if the respondent named AIDS among the highest priority issue for the government, 2 if she named AIDS as the second-highest priority issue, 1 if she named AIDS as the third-highest priority issue, and 0 otherwise. To conduct a falsification test, we also exploit a measure of policy support for general public health policies which is unrelated to HIV/AIDS. To this end, we use a binary variable that takes the value 1 if the respondent names public health but not HIV/AIDS among the top three priority issues for the government. Afrobarometer surveys also contain several questions that specifically address the HIV epidemics. The key measure that we exploit in our analysis is the perceived prevalence of the disease, captured by whether the respondent personally knows someone who died of AIDS.

To measure the quality of early prevention policies, we use the AIDS Program Effort Indices (API) developed by the UNAIDS (see UNAIDS 2003 for a detailed description).

These indices provide a measure of national governmental efforts to fight the HIV epidemics along several dimensions. For the purposes of our study, we use the API Prevention Index which provides a quantitative measure of the intensity of national HIV/AIDS prevention policies in 2003.[17]

Second, the latest round of the Afrobarometer surveys was collected in 2008. However, by 2005, large-scale international interventions to combat HIV/AIDS accelerated (e.g. in 2009, about one-half of the total global funding to fight the epidemic came from developed countries' governments[18]). Therefore, it is likely that the individual responses concerning policy support for HIV/AIDS in 2008 are more likely to be influenced by these international initiatives (either positively through raising awareness, or negatively by crowding out the support for domestic HIV programs). We therefore chose to use the earlier round, conducted in 2005, which is less sensitive to this problem.

[**Table 1 about here**]

Table 1 presents the summary statistics for the main variables. Even though about one-half of the respondents express their preference for increasing national spending on AIDS, they do not rank AIDS highly in terms of political priorities: barely 7 per cent of them consider AIDS to be among their governments' top three priorities. By contrast, 23 per cent of them consider public health (excluding AIDS) to be among those top three priority issues. This in itself is surprising, since almost forty per cent of the respondents know someone who died of AIDS. As suggested by our theoretical model, we will show below that this puzzle is explained by the intensity of earlier prevention policies. Finally, we find very large variations in the intensity of past policies: on the 0 to 1 scale, the API Prevention Index in 2003 is on average equal to 0.7, with a minimum of 0.22 (for Lesotho) and a maximum of 0.86 (for Nigeria).

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[17]As explained by the designers of the API project (UNAIDS 2003: 4), API indices were initially constructed in 2000, but this first round contained some inconsistencies, and a new survey was administered in 2003 to address them. To the best of our knowledge, this represents the only internationally comparable quantitative measure of the intensity of public policies against HIV/AIDS, but there is no data available measuring properly the intensity of early anti-HIV/AIDS policies (i.e. before 2003).

[18]See www.avert.org/aids-funding.htm.

3.2 Empirical strategy

We analyze the relationship between individual support for HIV/AIDS policies and HIV/AIDS prevalence. An approach which would simply correlate policy support by an individual and the prevalence she perceives would yield biased results, because of the unobservable individual characteristics (e.g. attitude towards risky sexual behavior) that drives both her policy support and her exposure to prevalence. We avoid this shortcoming by aggregating the measure of perceived prevalence at the regional level. Given that our dataset contains a large number of individual observations in each region (there are over 25 thousand individual respondents living in 202 regions), we use this average as a measure of the regional prevalence of HIV/AIDS.

The following equation describes empirically the individual policy support as a function of the prevalence rate:

$$q_{ijk} = b_0 + b_1 \alpha_{jk} + c' \mathbf{X}_{ijk} + \varepsilon_{ijk}, \tag{18}$$

where, q_{ijk} represents the support for national HIV/AIDS policies by respondent i in region j of country k, α_{jk} is the HIV prevalence rate in region j, $\mathbf{X}_{ijk}$ is a vector of individual characteristics of citizen i that influence her policy support, and ε_{ijk} is the error term. Our theoretical model, however, qualifies this relationship as it predicts that prevention policies are path-dependent. As a result, for a given prevalence rate, the individual support for HIV/AIDS policies should be more important in countries that have actively implemented large-scale prevention policies in the past. We therefore modify the empirical relationship as follows:

$$q_{ijk} = b_0 + b_1 \alpha_{jk} + b_2 Q_k + b_3 (\alpha_{jk} Q_k) + c' \mathbf{X}_{ijk} + \varepsilon_{ijk}, \tag{19}$$

where Q_k represents the importance of HIV/AIDS prevention policies in country k in the past, and $\alpha_{jk} Q_k$ is the interaction between the regional prevalence rate and these past prevention policies. Our model predicts that, ceteris paribus: (i) individual policy support is higher in countries where past prevention policies were more extensive and (ii) in these countries, individual support reacts more strongly to a given variation in HIV prevalence. We therefore expect positive signs for both b_2 and b_3.

3.3   Econometric results

Our results are presented in Table 2. In all specifications, standard errors are corrected for the possible presence of heteroskedasticity and are clustered at the region level. The first three columns of Table 2 present policy support as a function of past prevention policies. In 2005, citizens express stronger political support for HIV/AIDS policies in precisely those countries where prevention policies were more extensive in 2003 (as captured by the API Prevention Index). The pattern is similar (although the correlation is somewhat weaker) when the alternative measures of the policy support are used. This suggests some path dependence in HIV/AIDS policies, as predicted by our theoretical model.

[**Table 2 about here**]

Given that prevention policies were conducted at the national level, there is no within-country variation in past policies (which is why we cannot include country fixed effects in the specifications adopted in columns 1-3). Our findings could therefore simply capture variations across countries in attitudes towards HIV/AIDS, which could drive both prevention policies in 2003 and public support 2005, without implying any path dependence. In columns 4-8 we therefore investigate within-country variations in (perceived) HIV prevalence rates and address our second testable prediction.

In column 4, we regress the individual policy support measured by the citizen's preference for increasing government spending on AIDS, as a function of perceived regional HIV prevalence. Citizens in regions with higher perceived prevalence do not seem to provide stronger support for HIV/AIDS policies. However, this lack of correlation hides very different responses to variations in perceived prevalence, depending on the importance of past prevention policies. In columns 5-7 we interact regional HIV prevalence rates and the 2003 API Prevention Index. For all measures of policy support, we find a positive and significant coefficient on the interaction term. Citizens in countries which carried out more extensive prevention policies in 2003 respond much more strongly to variations in perceived prevalence, as compared to those living in countries with less active 2003 prevention policies. This

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supports our second testable hypothesis.[19]

In column 8, we proceed to a simple falsification test. Instead of using HIV policy support, we use individual support for public health policies (excluding HIV/AIDS). We do not expect more extensive past HIV prevention policies to have an impact on public support for more general health policies. In fact, we find no correlation between individual support for these policies and regional prevalence (interacted or not).

4   Discussion

Our model is built on several basic premises. First, the inter-generational transmission of the disease is via sexual contact between the old and the unaware young. The alternative assumption would be to consider mother-to-child transmission (MTCT), which can be avoided if the mother is aware. The timing of event then needs to be changed slightly, but the qualitative results of the model are not altered. The reason why we have opted for our specification is that the relative importance of the MTCT as compared to the inter-generational sexual contacts is limited as compared to other modes, the most important being heterosexual contact (see Beyrer et al 2005, and also Barnett and Whiteside 2006: 354-355, for a discussion of MTCT prevention policies). Second, the role of conservative citizens is limited to voting against the policy (and avoiding risky sexual contacts). More realistically, they could also dilute the effectiveness of government policies if campaign targeting is imperfect (as some of the campaign messages would be 'wasted' on conservatives). They could also play a role opposite to that of NGOs, for instance, by organizing campaigns against the use of condoms (as occurred in Kenya in the 1990s, see Kwena 2004). Both of these alternative assumptions would raise the majority-rule threshold, without affecting the qualitative properties of our model.

Similarly, old citizens do not play an active role in the model. Without altruism, the old generation always votes against prevention policies (which are of no benefit for the elderly).

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[19]The strong relation between past policies and curent policy support calls into question alternative explanations for the lack of response of public policies to prevalence. One such alternative is the malfunctioning of the political systems in most of these countries (despite many well known exceptions of direct relevance here, such as Botswana or South Africa), and the lack of political accountability is a well-known malaise of Sub-Saharan African countries (Van de Walle, 2001). Another alternative (Deaton et al. 2010) suggests that the value of life in Africa is too low to provide public support for such policies.

This simply adds an extra conservative weight in the political process. Allowing for filial altruism (as is highly plausible in traditional societies, see Banfield 1958, and Platteau 2000), the issue of strategic inter-temporal voting would arise. This additional complication would again make our results less clear cut, adding however no further insights. We instead opted for a simpler political economy approach without altruism, which allowed us to derive well-defined results.

A key point is the link between voter preferences and equilibrium policies, which one might question, given the poor functioning of democratic institutions in many of the African countries. Our assumption of aggregation of policy preferences through voting can we relaxed substantially, without losing any of the main insights of the model, if we allow for both more sophisticated political rules (based on lobbying, protest or legislative bargaining) or even to encompass non-democratic societies (as far as there exist *some* channel of influence by aware and unaware/conservative citizens on the decisions of the autocratic leader).[20] Similarly, the main results survive even when one allows for the higher weight given to the aware voters when the prevalence rates are high (e.g. because of civil society pro-awareness activism); in that case, the majority support line on Figures 3 and 4 will tilt counter-clockwise, but the model would still feature multiple equilibria and the fragility of the low-awareness equilibrium.

We have adopted a very simple awareness technology based on a one-to-one correspondence between policy efforts and the proportion of aware agents. Alternatively, one can assume a more complex technology, where the proportion of aware citizens depends non-linearly on prevention policies and the weight of conservative forces in the society (e.g. $q_t^y = f(q_{Pt}, \gamma)$). These changes, while possibly closer to the spirit of a more general model of public health, again do not alter our main results. What is essential here is the dual role played by awareness: it both affects individual behavior directly and the agents' perception of the role of prevention policies.

We do not treat two other important issues. One is the choice in the number of partners.

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[20]We abstract from the role of ethnic fractionalization, which has been shown to play a role for the importance of internal prevention policies (Lieberman 2007), the quality of the local health centers (Bjorkman and Svensson 2011) and HIV transmission rates (Pongou and Serrano 2009).

Oster (2005) shows that the combination of multiple partners and risky sexual practices is the main determinant of the differences in prevalence across African countries (see also Morris 1997). Our model instead assumes only one partner within the young generation. Introducing multiple partners and endogenizing the number of partners is the natural next step in understanding the political-economic dynamics of the epidemics. The other issue is the multiple dimensions of HIV/AIDS policies. Most government policies currently combine prevention and treatment. In this paper, we focussed on prevention policies, as this was the major policy carried out by government before 2005. In future work, we intend to explicitly address the issue of treatment versus prevention, as these two policies differ in many different dimensions, and exhibit some degree of political substitutability. For instance, while treatment policies can be better targeted to its potential beneficiaries than prevention policies, their effects can also be more easily measured and can therefore be more politically rewarding. In the context of our model, the increased availability of inexpensive antiretroviral drugs has the effect of reducing the utility differential between the sick and the healthy. As the demand from aware voters would be accordingly reduced, these changes imply less active government prevention programs and therefore may lead to higher prevalence in equilibrium.

5 Conclusion

Over the past two decades, government policies against the HIV/AIDS epidemic in developing countries raise two challenging issues. First, these policies, particularly in terms of prevention, vary a lot, are often inconsistent over time and are not related to the prevalence of the disease. Second, and perhaps more surprisingly, public opinion in favor of HIV/AIDS policies is also not related to strength of the epidemic. In this paper, we adopt a political economy approach and develop a model of HIV/AIDS prevention policies in which the policies and the political support in their favor are simultaneously determined.

Two stable steady state equilibria emerge: one with high prevalence, low awareness and no policies and the other with low prevalence, active policies and high awareness. The low prevalence equilibrium is fragile on several counts as the public support needed for its persistence gets easily eroded by external changes. We re-interpret the first stylized fact above

as arising from the coexistence of two very different equilibria and the inherent fragility of the active policy equilibrium. We also stress the crucial role of NGOs and external donors in providing a stable source of prevention campaigns that help to consolidate popular support in their favor. Moreover, reduced transmission rates have an ambiguous impact on prevalence rates as they also imply less active prevention policies in equilibrium. In the model proposed, past policies have a direct influence on the current levels of awareness that determine public support for such policies in the future. We re-examine public opinion surveys on the issue and show that public support for HIV/AIDS policies is strongly related to prevalence rates in precisely those regions that had an active prevention policies in the past, but not in regions that had no such policies.

The model and the empirical investigation proposed in this paper also have a much wider applicability than the analysis of HIV/AIDS policies, as they suggest a new approach to the political economy of public health policies for which externalities are present (such as sanitation, misuse of antibiotics or deworming policies). There also, the sustainability of current policies crucially depend on public support in their favor, which itself results from previous policies and information campaigns. We believe that pursuing this approach can provide new insights on the political determinants of public health.

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6 Appendix: Local stability of the low prevalence equilibrium

We derive here sufficient conditions for the local stability of the interior equilibrium $(q^y, \alpha^{y,u})$. The equilibrium value for q_t^y is:

$$q^y = \frac{1}{p} - \frac{1}{\delta \Delta \Omega (1 - \Phi) \alpha^{y,u}} = \frac{1}{p} - \frac{1}{K \alpha^{y,u}},$$

where $K = \delta \Delta \Omega (1 - \Phi)$. The dynamics in $\alpha_t^{y,u}$ is given by:

$$\alpha_{t+1}^{y,u} = \theta \left[(1 - q_t^y) \alpha_t^{y,u} + (1 - q_t^y)^2 (1 - \alpha_t^{y,u}) \alpha_t^{y,u} + q_t^y (1 - q_t^y)(1 - \Phi)^2 \alpha_t^{y,u} \right].$$

Differentiating this last equation around the equilibrium value $\alpha^{y,u}$ yields the following expression:

$$\alpha_{t+1}^{y,u} \simeq \theta \left[\begin{array}{c} (1-q^y)(\alpha^{y,u}+d\alpha) + (1-q^y)^2(1-2\alpha^{y,u})(\alpha^{y,u}+d\alpha) + q^y(1-q^y)(1-\Phi)^2(\alpha^{y,u}+d\alpha) \\ -\alpha^{y,u}\frac{\partial q_t^y}{\partial \alpha}d\alpha - 2(1-q^y)(1-\alpha^{y,u})\alpha^{y,u}\frac{\partial q_t^y}{\partial \alpha}d\alpha + (1-2q^y)(1-\Phi)^2\alpha^{y,u}\frac{\partial q_t^y}{\partial \alpha}d\alpha \end{array} \right]$$

where $\frac{\partial q_t^y}{\partial \alpha} = \frac{1}{K(\alpha^{y,u})^2}$. Using this and rearranging the last expression, we have:

$$\alpha_{t+1}^{y,u} \simeq \alpha^{y,u} + \theta \left[\begin{array}{c} (1-q^y) + (1-q^y)^2(1-2\alpha^{y,u}) + q^y(1-q^y)(1-\Phi)^2 \\ -\alpha^{y,u}\frac{1}{K\alpha^{y,u2}} - 2(1-q^y)(1-\alpha^{y,u})\alpha^{y,u}\frac{1}{K\alpha^{y,u2}} + (1-2q^y)(1-\Phi)^2\alpha^{y,u}\frac{1}{K\alpha^{y,u2}} \end{array} \right] d\alpha$$

$\Leftrightarrow$

$$\alpha_{t+1}^{y,u} \simeq \alpha^{y,u} + \theta \left[\left[1/\theta - (1-q^y)^2\alpha^{y,u}\right] + \left[-1 - 2(1-q^y)(1-\alpha^{y,u}) + (1-2q^y)(1-\Phi)^2\right] \frac{1}{K\alpha^{y,u}} \right] d\alpha.$$

In the bracketed term, the first term, which represents the inter-temporal transmission of prevalence for a given prevention policy, is always positive, while the second, which captures the policy response to a small change in prevalence, is always negative. A sufficient condition for local stability is:

$$\left| \theta \left[\left[1/\theta - (1-q^y)^2\alpha^{y,u}\right] + \left[-1 - 2(1-q^y)(1-\alpha^{y,u}) + (1-2q^y)(1-\Phi)^2\right] \frac{1}{K\alpha^{y,u}} \right] \right| < 1.$$

To simplify notations, we define A and B as follows:

$$A = \left[1/\theta - (1-q^y)^2\alpha^{y,u}\right] ; B = \left[-1 - 2(1-q^y)(1-\alpha^{y,u}) + (1-2q^y)(1-\Phi)^2\right] \frac{1}{K\alpha^{y,u}}.$$

First note that $\theta A < 1$, and since B is always negative, we know that $\theta(A+B) < 1$ for all relevant parameter values.

To ensure stability, we therefore need to provide sufficient conditions such that $\theta(A+B) > -1$. Term A is strictly increasing in q^y, so that $A \geq 1/\theta - \alpha^{y,u}$. Term B is increasing or decreasing in q^y depending on whether $2(1-\alpha^{y,u} - (1-\Phi)^2)$ is greater or smaller than 0. In the first case, a lower bound for B is obtained by taking the lowest admissible value of q^y, which is $\frac{1}{2} : B \geq (-1 - (1-\alpha^{y,u}))\frac{1}{K\alpha^{y,u}}$. A sufficient condition for stability is then:

$$\theta(A+B) > -1 \; if \; -\alpha^{y,u} + \frac{\alpha^{y,u}-2}{K\alpha^{y,u}} > \frac{-2}{\theta}.$$

In the other case, a lower bound for B is obtained by taking the largest admissible value of q, which is $1 : B \geq (-1 - (1 - \Phi)^2)\frac{1}{K\alpha^{y,u}}$. A sufficient condition for stability is then:

$$\theta(A + B) > -1 \; if \; -\alpha^{y,u} - \frac{1 + (1 - \Phi)^2}{K\alpha^{y,u}} > \frac{-2}{\theta}.$$

A sufficient condition for these two conditions to hold simultaneously is that: $-\alpha^{y,u} - \frac{2}{K\alpha^{y,u}} > \frac{-2}{\theta} \Leftrightarrow \frac{1}{K\alpha^{y,u}} < \frac{1}{\theta} - \frac{\alpha^{y,u}}{2}$ which holds if $\frac{1}{K\alpha^{y,u}} < \frac{2 - \theta}{2\theta}$. In words, if the policy response to the increased prevalence (remember that $\frac{\partial q_t^y}{\partial \alpha} = \frac{1}{K(\alpha^{y,u})^2}$) is not too large, the stationary equilibrium is locally stable.

Using the expression above, we can also find a condition for the equilibrium to be locally unstable. In order to do this, we look at the conditions under which, for the largest values of A and $B, \theta(A + B) < -1$. An upper bound on A is given by $\frac{1}{\theta}$, while an upper bound on B is given by $-\frac{(1 + (1 - \Phi)^2)}{K\alpha^{y,u}}$ or $-\frac{2 - \alpha^{y,u}}{K\alpha^{y,u}}$ depending on whether B is increasing or decreasing in q^y, respectively. Note that those two values are both smaller than $-\frac{1}{K\alpha^{y,u}}$. As a result, a sufficient condition for $\theta(A + B) < -1$ is given by the following condition:

$$\theta\frac{1}{\theta} - \theta\frac{1}{K\alpha^{y,u}} \leq -1 \Leftrightarrow \frac{1}{K\alpha^{y,u}} \geq \frac{2}{\theta}.$$

The interior equilibrium is not locally stable if policies are sufficiently responsive to changes in prevalence.

Figure 1

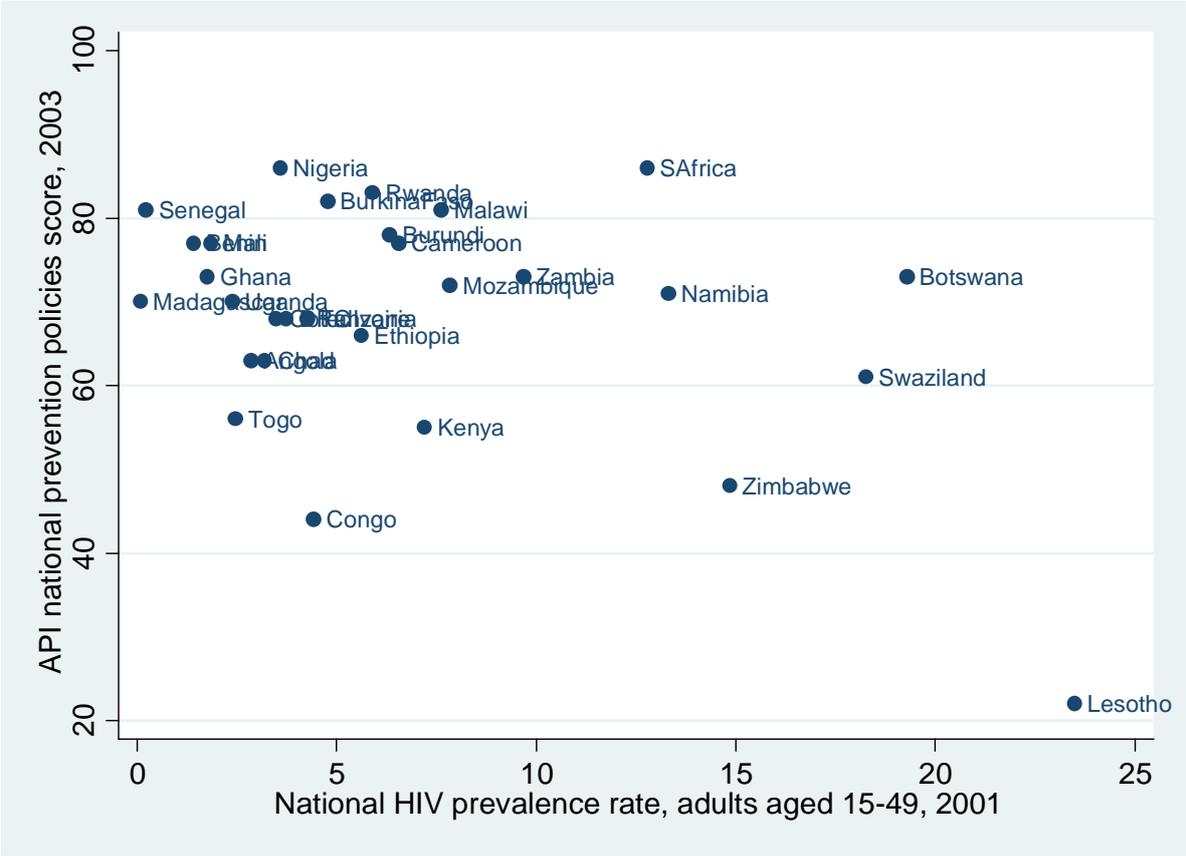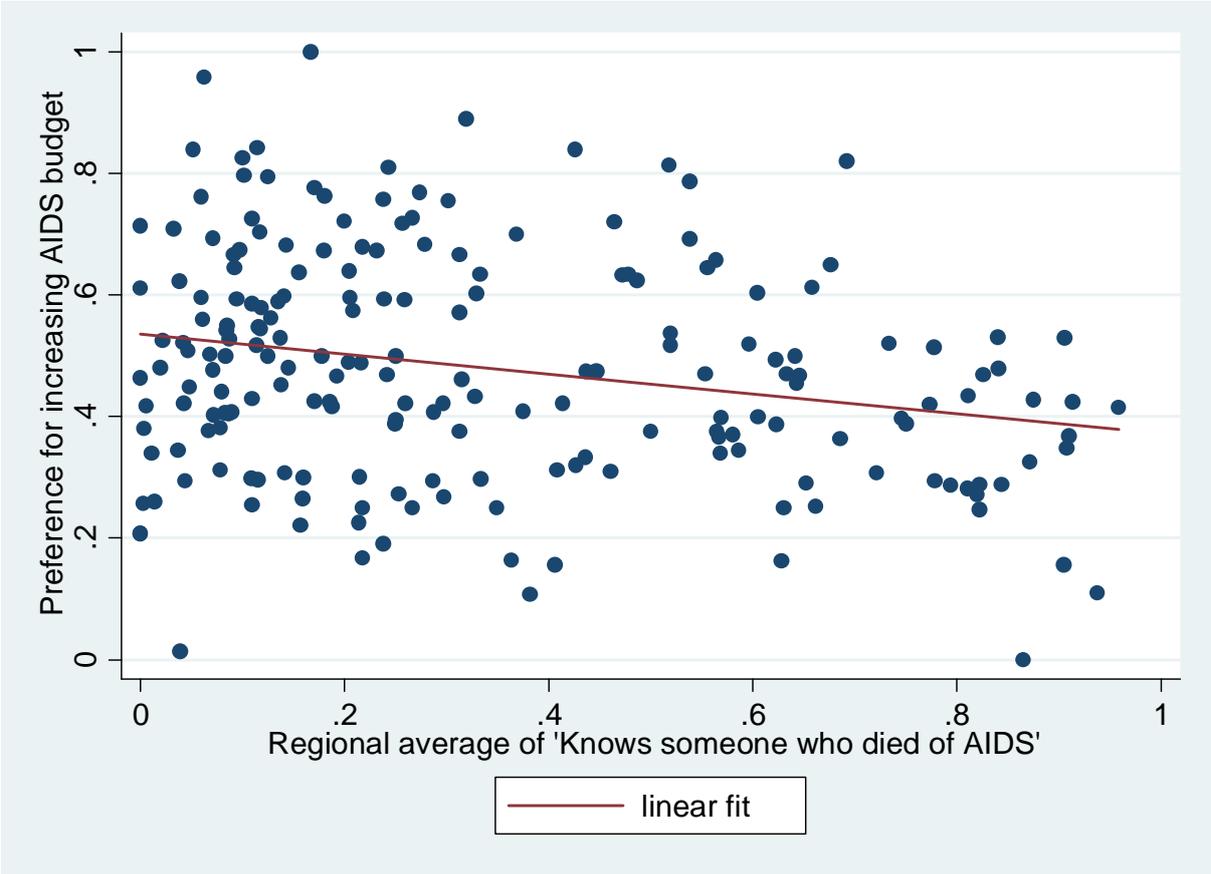## API score in 2003 versus HIV prevalence rate among adults (15-49) in 2001
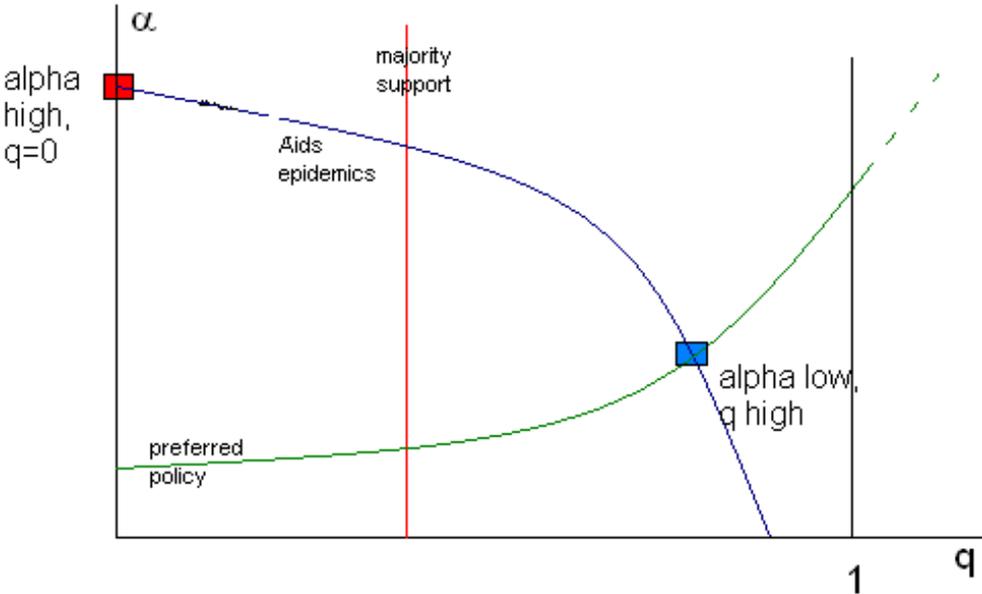
Figure 2

HIV prevalence and the preference for increasing AIDS budget

Figure 3

Steady state equilibria

Notes: Figure 3 plots the share of aware individuals among non-conservative voters (*q*) against the HIV prevalence among the young unaware (*α*). Blue and red squares denote, respectively, the low-prevalence and the high-prevalence steady-state equilibria.

Figure 4

Short run dynamics: local instability
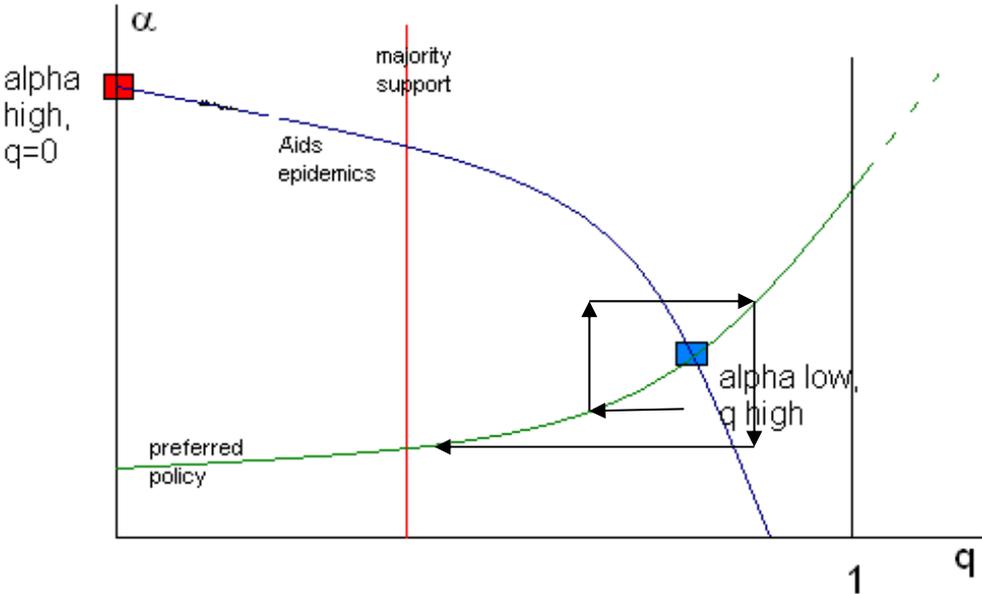
Notes: Same as in Figure 3. Arrows show the short-run dynamics of the system to an exogenous drop in HIV prevalence, when the economy is initially at the low-prevalence steady-state equilibrium.

Table 1. Summary statistics

Variable	Obs	Mean	Std Dev	Min	Max
Individual-level					
Preference for increasing gov't spending on AIDS	24602	0.466	0.499	0	1
Named AIDS as one of gov't top 3 priorities	25397	0.072	0.258	0	1
Priority score of AIDS for gov't intervention	25397	0.131	0.516	0	3
Named health (but not AIDS) as one of gov't top 3 priorities	25397	0.232	0.422	0	1
Know someone close who died of AIDS	24618	0.384	0.486	0	1
Region-level					
Perceived prevalence of HIV	202	0.338	0.271	0	0.958
Preference for increasing gov't spending on AIDS	202	0.480	0.186	0	1
Regional mean of naming AIDS as one of gov't top 3 priorities	202	0.070	0.099	0	0.531
Country-level					
API Prevention Index, 2003	17	0.696	0.156	0.22	0.86

Table 2. Regression results

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Preference for increasing gov't spending on AIDS	Named AIDS as one of gov't top 3 priorities	Priority score of AIDS for gov't intervention	Preference for increasing gov't spending on AIDS	Preference for increasing gov't spending on AIDS	Named AIDS as one of gov't top 3 priorities	Priority score of AIDS for gov't intervention	Named health (but not AIDS) as a gov't top 3 priority
API Prevention Index 2003	0.175	0.067	0.139					
	(0.071)**	(0.046)	(0.084)*					
Regional HIV prevalence				0.115	-1.969	-0.286	-0.542	0.272
				(0.124)	(0.535)***	(0.229)	(0.433)	(0.212)
API Prev. '03 x Reg. HIV prevalence					2.935	0.614	1.222	-0.230
					(0.770)***	(0.365)*	(0.684)*	(0.295)
Gov't effectivess in improving health servi	0.015	0.018	0.036	0.012	0.014	0.010	0.019	-0.045
	(0.009)	(0.004)***	(0.008)***	(0.008)	(0.009)*	(0.003)***	(0.006)***	(0.005)***
Age	0.000	-0.001	-0.002	0.000	0.000	-0.001	-0.002	0.005
	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)*	(0.001)***
Age, squared /1000	-0.010	0.011	0.029	-0.011	-0.009	0.005	0.016	-0.048
	(0.016)	(0.008)	(0.016)*	(0.015)	(0.015)	(0.007)	(0.014)	(0.011)***
Gender = female	0.017	0.011	0.020	0.022	0.023	0.003	0.006	-0.008
	(0.007)**	(0.003)***	(0.007)***	(0.006)***	(0.007)***	(0.003)	(0.006)	(0.006)
Household head	0.004	-0.007	-0.017	0.015	0.016	-0.004	-0.008	-0.004
	(0.011)	(0.006)	(0.011)	(0.009)	(0.009)*	(0.004)	(0.008)	(0.007)
Religious	0.013	-0.014	-0.021	0.026	0.029	-0.002	0.006	0.023
	(0.015)	(0.007)*	(0.015)	(0.012)**	(0.012)**	(0.004)	(0.009)	(0.008)***
Education	-0.016	0.016	0.028	-0.010	-0.011	0.005	0.007	0.001
	(0.004)***	(0.002)***	(0.005)***	(0.003)***	(0.004)***	(0.001)***	(0.003)***	(0.002)
Wealth	-0.004	-0.000	-0.000	-0.006	-0.006	-0.000	-0.000	-0.000
	(0.005)	(0.000)***	(0.000)***	(0.005)	(0.005)	(0.000)	(0.000)	(0.000)***
Constant	0.344	-0.038	-0.066	0.418	0.426	0.015	0.025	0.137
	(0.061)***	(0.041)	(0.073)	(0.059)***	(0.053)***	(0.027)	(0.056)	(0.029)***
Country fixed effects	N	N	N	Y	Y	Y	Y	Y
Observations	22523	23078	23078	23518	22523	23078	23078	23078
R-squared	0.01	0.02	0.02	0.04	0.04	0.12	0.12	0.08

*** $p<0.01$, ** $p<0.05$, * $p<0.1$. Robust standard errors in parentheses, clustered at region level.