Socio-Economic Inequalities and HIV/AIDS Epidemic: Evidence from Sub-Saharan Africa

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Abstract

Throughout the developing world, HIV/AIDS epidemic has became a major cause of death and poverty. Its consequences are readily felt on social, demographic and economic fields. The African continent is bearing the brunt of the epidemic, with its consequences readily felt on social, demographic and economic fields there. If it is generally admitted that poor health is fuelled by poverty, the relation between HIV/AIDS epidemic and poverty appears somehow paradoxical. Indeed at the international level, the most affected regions are the poorest, among Sub-Saharan African countries however, the most affected are also the richest. Nevertheless, the fact that these African countries are also those with the least egalitarian income distributions leads to wonder whether there is a specific impact of income inequality on the course of the epidemic. In addition, the distribution of the epidemic across both sexes differs according to regions, with Sub-Saharan Africa being the most *gender-affected* region: more than half of infected people there are women. In this paper, we try to understand these particular patterns of the epidemic by assessing the importance of income and gender inequalities as determinants of the evolution of HIV/AIDS pandemic in Sub-Saharan Africa.

Using a panel data of 42 African countries from the 1997-2005 period, we examine the potential link between socio-economic inequalities (defined by income and gender inequalities) and HIV/AIDS epidemic by introducing variables representing income and gender inequalities among the traditional determinants of the epidemic. The results of our random effects model suggest that socio-economic inequalities are indeed specific determinants of the epidemic. Their impact is robust to alternative specifications of the model and to a dynamic analysis. This impact is further materialized when we simulate the effect of a reduction of inequalities on the course of the epidemic.

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Introduction

At the international level, it is generally admitted that HIV/AIDS epidemic is fuelled by poverty. Indeed, countries with the highest prevalence rate are also the poorest of the world. however, in Sub-Saharan Africa -the region most heavily affected by HIV/AIDS in the world- countries with the highest rate of prevalence are not necessarily the poorest but those with the less egalitarian income distribution. For instance, Botswana which had the second highest (after Swaziland) prevalence rate in the world and South Africa which had the highest absolute number of infected persons are in fact the two richest countries of the continent and belong to the group of the upper middle income countries. However, there is a high level of income inequality in these two austral African countries. In fact, six of the ten countries with the highest Gini Coefficient in the world belong to austral Africa region with most of them recording a Gini coefficient higher than 0.5. (UNDP, World Development Report 2003). Despite numerous papers documenting the link between income inequality and health, the nature of the link is still unclear. While for some authors, income inequality has an adverse impact on health (Wilkinson 1996 and 2000, Farmer, Lynch 2001), others abstain from making hasty conclusions due notably to non-consensual results (Deaton 2003, Wagstaff and Van Doorslaer, 2000). If the link between income inequality and health status in general is yet to reach any consensus among researchers, the question remains as to the relation between income inequality and the specific case of HIV/AIDS infection in Sub-Saharan Africa. In the case of HIV/AIDS in sub-Saharan Africa, there exist some specificities due to the mode of transmission of the virus and to some characteristics of the continent that further magnify the impact of income inequality on health. Indeed, commercial sex work and regional migrations, which are widely practiced among the poor and as income generating strategies, have become vehicles for transmission the epidemic. This is also the case for armed conflicts due to which population are put under financial and psychological precariousness and where rapes are usually used as one of the weapons of war. Finally from the mid 80s, African countries have experienced a deep economic crisis which did not spare health sectors. The consequence of this crisis of health sector has been a deepening of poverty and an increase in inequality. Whatever the causes of this crisis, it resulted in the proliferation of the virus.

In the early stages of the pandemic, HIV infection was predominantly among men in many industrialized and some developing countries. As of the end of 2005, however, almost 50% (17.5 million) of the 38.0 million adults living with HIV/AIDS globally are women (UNAIDS 2006). This trend is even more pronounced in sub-Saharan Africa, where women account for close to 60% of people living with HIV. Young women represent the group most susceptible to be infected in the world: they account for 67 per cent of all new cases of HIV among people aged 15 to 24 in developing countries. Today, according to UNAIDS, some 7,000 girls and women become infected with HIV every day. Thus, there is clearly a need to address the role of gender inequalities on women's susceptibility and vulnerability to HIV/AIDS. Transmission of HIV among women must be considered within the context of gender roles, access to social and economic capital and cultural values as it is now clear that most dimensions of economic and social life are characterised by a pattern of inequalities between women. Indeed, there are important differences between women and men in the underlying mechanisms of HIV infection and in the social and economic consequences of HIV/AIDS (Moss, 2002).These differences stem from biology, sexual behaviour and socially constructed 'gender' differences between women and men in roles and responsibilities, access to resources and decision-making power.

Using a panel data of 42 African countries for the period of 1997 to 2003, we test for the hypothesis of a specific link between socio-economic inequalities, defined by income and gender inequalities on the one hand and HIV/AIDS epidemic on the other hand, by including variables representing inequalities among the traditional determinants of the epidemic. The remainder of this paper is organized as follows. We will first present the obvious link between income inequality and HIV/AIDS epidemic. Afterward, we will study the link between gender inequality and the epidemic. In a next section, we present our model. Finally we simulate the impact of a reduction of inequalities on the course of the epidemic *ceteris paribus*, just before the concluding remarks.

1) Income Inequality and HIV/AIDS

For some authors (Wilkinson 1996, Marmot 2001), relative deprivation is of big concern for wellbeing, including health whereas other (Deaton 2003, Wagstaff and Van Doorslaer 2000) conclude that only individual poverty can affect people's health.

1.1) Income Inequality and health: a vague relationship

About two centuries ago, in his book "The Wealth of Nations", Adam Smith (1776) described the concept of relative deprivation, referring to the "necessaries" of daily life. Health status being an indisputable element of welfare, relative income is likely to have an impact on it through these « necessaries ». Lynch and *al* (1996) go as far as quantifying mortality attributable to income inequality as exceeding the combined deaths resulting from lung cancer, diabetes, motor accidents and HIV/AIDS 1995 in USA.

1.1.1) Relative Income and Income Inequality Hypothesis

Wagstaff & Van Doorslaer (2000) summarized the different theories that postulate a link between income inequality and health under the general form of two models: the income inequality hypothesis and the relative income hypothesis.

According to relative income hypothesis, individual's health depends on the differential between individual's income and average income of population.

$h_i = f_i\ (y_i - \bar{y}_p)$ where:

y_i is individual *i*'s income and $\bar{y}_p$ is average income of population P.

Thus, if all members of population P observe an increase in their income but individual *i* whose income does not decrease neither, his health will worsen.

By the same way health status in community c depends on the differential between average income in community c and the average of income of all communities:

$h_c = f_c\ (y_c - \bar{y}_c)$ where:

y_c is the average income in community *c* et $\bar{y}_c$ is the average of the incomes of all communities.

The equation relative to the income inequality hypothesis is the following:

$h_i = f_i\ (y_i, I_p)\ (f_I' \prec 0)$ where: I_p is income inequality in population P.

Thus, if income inequality increases in population, individual *i*'s health will worse, and if his income increases, his health status will improve.

According to Deaton (2003), the relationship between health, income and income inequality can take the form of the following equation:

$$h_{is} - \bar{h} = \alpha + \beta(y_{is} - \bar{y}) - \delta(y_{is} - \bar{y})^2 - \theta v_s$$

Where v denotes the pure effect of income inequality on individual *i*'s health.

$h_{is} - \bar{h}$ is the differential between individual i's health and average health in group *s*

$y_{is} - \bar{y}$ is the differential between individual i's income and group average income $\bar{y}$.

Thus, income surely promotes health, but less for the rich. Also, if average income is high, there is still need that everyone's income is high enough, at least beyond the point at which income distribution has much effect on health.

At the aggregated level, the same relationship translates as:

$$h_s - \bar{h} = \alpha + \beta(y_s - \bar{y}) - \delta(y_s - \bar{y})^2 - (\theta + \delta)\gamma_s$$

The impact of income on population's health is given by:

$$(h_s - \bar{h})'_{y_s} = \beta - 2\delta(y_s - \bar{y}) < 0. \qquad (1)$$

The impact of income inequality on state's health is given by:

$(h_s - \bar{h})_{\gamma_s} = \theta + \delta$, which is constant. *(2)*

The direct implication of *(1)* and *(2)* is that, as income increases, its impact on health decreases while income inequality's impact remains constant. That means the effect of income inequality becomes more important than the effect of income as countries become richer. Then, among rich countries, average income is less important and income inequality is relatively more important.

1.1.2) Transmission channels

Broadly, there are three interpretations as to the link between income inequality and health status.

- *Access to life opportunities*: When the gap between rich and poor widens, interests diverge, translating into reduced social spending, namely education and health (Kawachi, Kennedy and Wilkinson 1999). Kaplan and al (1996) using U.S data found a positive and significant correlation between the level of inequality at state level and support to human services. Richer people allocate a greater share of their income to « competitive spending » and try to escape from the neighbourhood. They are also more reluctant about spending that benefit

the whole community. Greater income inequality would thus affect health by reducing poor people's access to education and health services.

- *Social cohesion*: Kawachi and Kennedy (2002) found that when preferences are heterogeneous (low degree of social cohesion), the cost of consensus is high and it becomes more difficult to agree about the nature of expenditure to engage in. As the gap between rich and poor deepens, interests diverge, resulting in lower or inadequate social expenditure. In the case of health expenditure, decision-makers tend to give priority to expenditure related to the elite health's problems, neglecting health conditions that make up the bulk of epidemic profile in the country. Unlike poverty that affects only the health of the poor, income inequality is detrimental to everyone's health because it weakens social infrastructure and destroys social cohesion.

- *Psychosocial factors:* Income inequality affects heath status through the perception individuals have of their relative position in society[1]. According to Wilkinson (1996), beyond an income per capita of $10.000, what matters for health is no more income, but income differentials between the richest and the poorest citizens. This relation transits through psychosocial factors; economic insecurity and lack of control over one's life and work result in chronic stress and anxiety that weaken the immune system. These situations also contribute to the lack of self-confidence, development of depression anger and destructive behaviours with a deleterious impact on health and that are well spread in inegalitarian societies[2].

1.1.3) Critics of the income inequality hypothesis

To date, the link between income inequality and health, though largely documented is far from being consensual. Critics about this link are based on theoretical grounds as well as on empirical demonstrations.

From a theoretical point of view, some authors criticized the income inequality hypothesis because of its conceptual vagueness. First, the mechanisms through which income inequality impacts health are poorly understood (Wagstaff &Van Doorslaer, 2000); it is not obvious why an individual with a decent income, sufficient to afford his basic

[1] The awareness of one's subordinated position in social or professional hierarchy causes chronic stress, hypercholesterolemia and an abnormally high cortisol rate in blood, with all their negative effects on health.

[2] This is the case for addiction to smoking, drug and the practice of violent hobbies. The level of stress undergone by each individual also depends on the differential between his or her income and that of the other group members.

needs could have poor health outcomes because he compares himself to his richer peers. Second, though it is adequately proved that inequality poses a hazard to health, it is not certain that it is precisely income inequality that is in cause, especially when it is comforted by other forms of inequalities. In that view, Deaton (2003) warns about a misinterpretation of income inequality coefficient in econometric regressions. In studies conducted in some rich and non-poor countries, namely USA, Brazil and South Africa the proportion of the population who live in poverty or at least in relative poverty is quite big. As a result, the income inequality's effect in these countries would in reality represent the impact of poverty. By the same way, women, rural people and minorities are generally poorer. In this way, income inequality' effect captures the impact of these other forms of inequality.

In addition to these theoretical critics, there are also some empirical problems that have been raised in relation to the link between inequality and health. Thus, Gravelle (1998) showed that the relationship found at aggregated level simply reflects the concavity of the function linking health and income level. Besides, the conclusions of the studies establishing a link between income inequality and health have a limited validity because results found with aggregated data are not always reproducible at individual level. In other words, the observed relation between income inequality and health could simply reflect the fact that inequality rates are inadequately measured at individual level and yield an "ecological fallacy" (Fiscella and Franks, 1997).

1.2) Peculiarities associated with HIV/AIDS and with SSA

The relationship between poverty and HIV/AIDS transmission is more complex that it first appears. Although it is generally admitted that it is poverty that causes AIDS, it is also worthy to notify that there are rich countries that are badly affected by the epidemic and within a particular country, the most affected people are not always the poorest (Lachaud, 2007). HIV/AIDS infection is not randomly distributed in the population. Indeed, though it seems to affect more people from higher socio-professional categories at the onset of the epidemic, it is thereafter fuelled by poverty like any other infectious disease[3]. In addition to the broad links between poverty, inequality and health, HIV/AIDS epidemic carries a set of specificities that magnify these links. Furthermore, Sub-Saharan Africa exhibits some peculiarities that intensify the relation between HIV/AIDS, poverty and income inequality.

[3] Even among infected people, there is inequality between rich and poor; infected poor people are susceptible to develop AIDS earlier than the rich ones due to malnutrition and poor access to treatment for opportunistic infections and even to antiretroviral therapy.

In this way commercial sex work and regional migrations, two income generating activities to cope with poverty acted as catalysts of the epidemic. Lastly, health services crisis in the region favoured the transmission of the virus.

1.2.1) Commercial sex work

Heterosexual intercourse is the main mode of transmission of HIV/AIDS in Africa. Commercial sex work is usually the only income generating activity available to women in a setting where unemployment rates especially among women are high. Two basic requirements for the onset of commercial sex work are fulfilled in Sub-Saharan Africa: a concentration of a sexually active population and, more importantly, the socioeconomic disparities that make sex work affordable by the client and an economic opportunity for the worker. Transactional sex, which is exchange of sexual services for money, gifts and protection, is even more common than sex work and people engaged in this kind of transaction are not always aware of the situation. In almost all countries with a generalized epidemic, the principal high-risk group identified is that of sex workers. Sex work has a big dissemination power in total population because the clients do not usually use condom during sexual intercourse with their life partner, regardless whether they use it or not in their commercial relationships. Thus prevention efforts among that group would be the most efficient, as they would prevent the infection of a big part of population (Ainsworth and Over, 1997).

1.2.2.) Regional migrations and armed conflicts

Even in countries where HIV/AIDS prevalence rate is low, this rate is relatively high among male migrant workers. In general, accelerated development of a region relative to its neighbors will lead to an increase income inequality between them and contribute to regional migration. Some aspects of their lifestyle make migrant workers more vulnerable to HIV/AIDS infection; accommodation with little or no rooms for privacy, relatively few number of women thus implying multiple partners, alcohol consumption and conception of manhood that induce risky behaviours[4]. Wars impoverish populations and displace them, making them more vulnerable to infection as they lack access to prevention services as well as STI diagnosis and treatment. Armed conflicts can also have an affect on the

[4] Within a country, income inequality between rural and urban areas will generate rural exodus especially of young and unmarried people in search for a better future to the cities. This displacement of rural dwellers is partly responsible for higher infection rates in the cities relatively to the villages as these rural newcomers are exposed to the same risks as migrant workers.

epidemic especially through the use of rapes as weapon of war. Evidence in SSA, especially in Uganda however fails to confirm that internally displaced persons and refugees are more likely to be HIV-infected than people in more stable settings (UNAIDS, 2006). A recent review of HIV literature on displaced persons in eight countries (including Uganda) also failed to find evidence that conflict increases HIV transmission (Spiegel and Harroff-Tavel, 2006).

1.2.3) Health sector crisis

From the mid-80s, health sectors in most African countries experienced a long and deep crisis, translating into the degradation of health outcomes which where improving from their independence level. The causes of this crisis are multiple; they can be attributed to Bamako initiative as well as to structural adjustment or more simply to overall economic crisis prevailing in the continent at that moment and which was the justification of structural adjustment. Authors are not unanimous about the effects of structural adjustment on health indicators in Africa. Whatever the actual causes of the crisis, it did favor the epidemic because: (1) mother-to-child-transmission could not be reduced in Sub-Saharan Africa as successfully as it was in other parts of the world[5], (2) health workers were directly exposed to the risk of contamination due to direct contact with blood and patients secretions.[6], (3) health services did not automatically treat patients for STIs and opportunistic infections, thus favouring the dissemination of the virus[7] and (4) Voluntary Counseling and Testing services, an important component of the fight against HIV/AIDS were not always available and when they did, the conditions required for them to be efficient were not fully fulfilled (confidentiality, tests kits, skilled workers).

[5] While some combinations of medicines (for example a combination of zidovudine and lamivudine) or single medicines (nevirapine) which are proved to be efficient in reducing vertical transmission were available and subsidized in these countries, infrastructures problems and poor quality of staff training rendered the implementation of these interventions impossible.

[6] Post-exposure prophylaxy measures that can reduce the risk of infection in medical field were not implemented early enough at the onset of the epidemic.

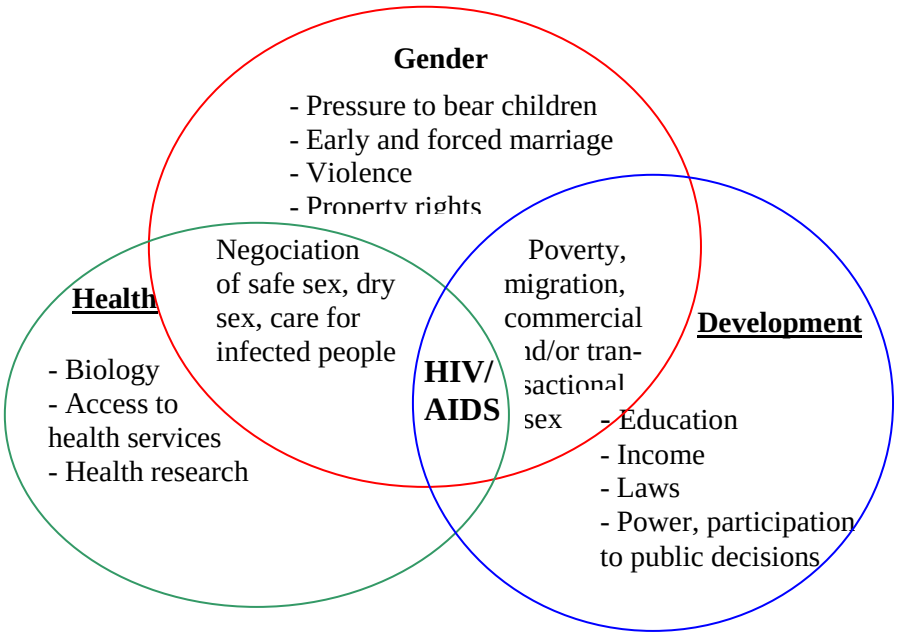
[7] These two kinds of infections are associated with higher rates of transmission, the former as a catalyst and the latter as an enemy of immune system that reinforces infectivity of sick people and accelerates the onset of full-blown AIDS, thus increasing fear and stigma associated with the disease.

2) Gender inequality and HIV/AIDS epidemic

2.1) Gender, development and health

Men and women are differentiated by social -or gender- characteristics on the one hand and by biological (or sex) characteristics on the other. Gender gaps are widespread in access to and control of resources, in economic opportunities, in power, and political voice. Women and girls bear the largest and most direct costs of these inequalities, but the costs cut more broadly across society ultimately harming everyone. For these reasons, gender equality is a core development issue-a development objective in its own right. In many societies, women have fewer educational opportunities than men and receive unequal access to resources such as food and income (Sen 1988), all of which are strong predictors of health status. In that sense, HIV/AIDS epidemic lies at the intersection of gender, development and health issues, as represented in the Figure 1.

Figure 1: HIV/AIDS in gender, development and health nexus



2.1.1) Gender and Health: the gender gap in health

There are concretely no societies in which women are treated as equals with men (World Bank 2001), and this inevitably affects both men's and women's health. Girls face disproportionately privation, lack of opportunity and lower levels of investment in their health, nutrition and education. Women receive unequal distribution of land and access to

resources such as food and health care. Women and men may also respond differently to treatment, have different access to health care and are treated differently by health providers. Thus, being male or being female has a major effect on an individual's health and well-being. Gender inequality and discrimination harm girls' and women's health directly and indirectly, throughout the life cycle; and neglect of their health needs prevents many women from taking a full part in society. Moreover, better access of women to these resources would allow to improve not only women' health, but also children's and the entire community's health.

2.1.2) Gender inequality and development

Given the fundamentally different social and economic roles of men and women, their different economic behaviours, including different patterns of consumption and expenditure, and their differential access to income and resources, it is no longer effective to construct an analysis of an economy or a society, at micro or macro levels which is not gender-sensitive. In addition to these personal costs, gender inequalities reduce productivity in farms and enterprises and thus lower prospects for reducing poverty and ensuring economic progress. Gender disparities are inextricably linked to poverty. On one level, poverty exacerbates gender disparities; inequalities between girls and boys in access to schooling or adequate health care are more acute among the poor than among those with higher incomes (World Bank 2001). On another level, gender inequalities hinder development. Finally, gender inequality undermines the effectiveness of development policies in fundamental ways, acting through three powerful tools for development: education, income and participation to public life.

2.1.3) Health and development

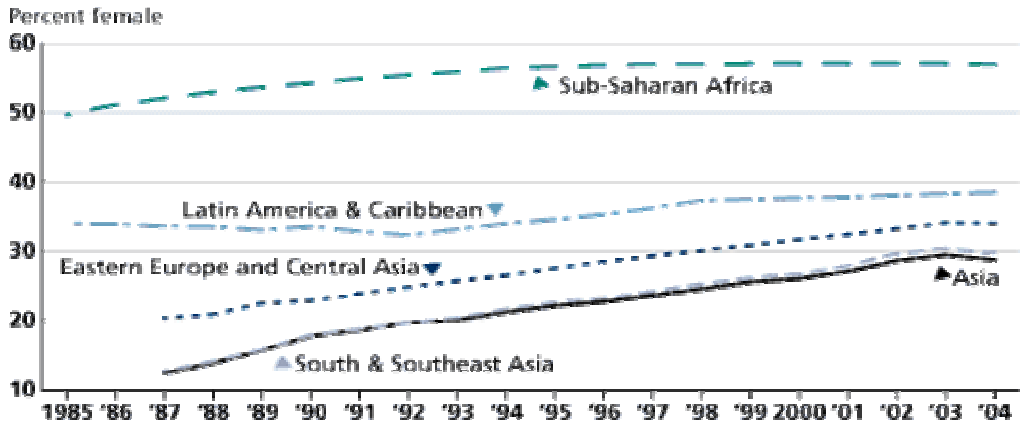
In *Development as Freedom*, Sen recalls that improving the quality of people's lives is development's ultimate goal. While there is a connection between opulence, on the one hand, and health, longevity and other achievements, on the other, the linkage may or may not be very strong and may well be extremely contingent on other circumstances. The issue is the capability to live really long and to have a good life while alive, exempt of misery and *unfreedom*, things that would be strongly valued and desired by nearly everyone (Sen, 2001). And among the most important freedoms that we can have is the freedom from avoidable ill-health and from escapable mortality. The usefulness of wealth lies in the things that it allows us to do, the substantive freedoms it helps us to achieve, including the freedom to

live long and to live well. But this relation is neither exclusive, since there are significant other influences on our lives other than wealth nor uniform, since the impact of wealth on our lives varies with other influences.

2.2) Gender discrimination and susceptibility to HIV/AIDS epidemic

Though the epidemic in the world initially affected mostly men, today approximately half of the 40 million people living with HIV are women. This is due to the fact that it spread rapidly in Sub-Saharan Africa where transmission is primarily heterosexual, often in the context of marriage.

Figure 2: Percent of Adults (15-49) Living With HIV Who Are Female, 1985-2004



Source: UNAIDS and WHO, *Women and AIDS* (2004).

The economic vulnerability of women makes it more likely that they will exchange sex for money or favours, less likely that they will succeed in negotiating protection, and less likely that they will leave a relationship that they perceive to be risky. Women have greater susceptibility than men to infection due to biological, social, cultural and physiological reasons. The pattern of women's and men's roles and relationships in Africa puts women at greater risk. In this sense, polygamy, sexual coercion and violence against women all contribute to the distressing gender gap in HIV/AIDS.

2.2.1) Direct exposure to the virus

Biologically, the risk of HIV infection during unprotected vaginal intercourse is two to four times higher for women than for men[8]. Second, there are some traditional sexual practices that need to be mentioned as especially dangerous for women's health. The practice of dry sex for example, very common in Southern Africa is harmful to women and put them at greater risk of infection. Finally, while this route is very rare, there is little evidence of contamination during daily life with an infected person (CDC 1999); indeed the vast majority of women and girls who care for infected family members are at risk, doing so with very little material support and this might result in their own infection with the virus (Ogden and Esim, 2003).

2.2.2) Patterns of marital life

The first pattern of marital life that puts women at greater risk of infection in Sub-Saharan Africa is polygamy. Polygamy is inherently discriminatory, and, because the man may be having unprotected sex with multiple partners, exacerbates the risk of HIV transmission. Another characteristic of marital life in African countries is early marriage for girls. In many countries, including several with high rates of HIV infection, girls are married in their teens, often as a poverty reduction strategy. Husbands and families also apply considerable pressure on young wives to have a child soon after marriage, increasing their risk of maternal death or injury and hampering efforts to prevent STIs and HIV through regular condom use. There is evidence that the age gap between partners affects the chances that young women will become infected (Kelly et al., 2003).

2.2.3) Low socioeconomic and sociocultural status

Low socio-economic and socio-cultural statuses experienced by women translate into commercial sex work, violence, inability to negotiate safe sex and to access adequate information.

Commercial sex work and transactional sex: The poverty experienced by women and men in developing countries has been deepened by increasing global economic inequalities. But unequal gender relations and unequal access to economic resources have made women poorer than men. Poverty and wealth inequality between men and women can fuel HIV

[8] This is because women have a bigger surface area of mucosa exposed to their partner's sexual secretions during intercourse and semen can stay in the vagina for hours after intercourse. In addition, women are also more likely than men to have asymptomatic STIs, which can increase the risk of HIV infection by three to four times.

transmission as women engage in unsafe sex in exchange for money, housing, food or education. Although many women do not view themselves as sex workers, most of them have at one moment in their life resort to transactional sex, that is using sex as a commodity in exchange for goods, services, money, accommodation, or other basic necessities—often with older men (Halperin and Epstein, 2004). The dependencies built into these relationships limit women's abilities to protect themselves from HIV infection, especially when the perception of younger women as "pure" encourages men to avoid using condoms (Gregson et al. 2002).

Violence: As well as economic and social insecurity, many women also have to face the threat of physical violence if they are not sufficiently responsive to a partner's desires. Violence in the form of coerced sex or rape may also result in the acquisition of HIV, especially as coerced sex may lead to the tearing of sensitive tissues and increase the risk of contracting the HIV virus. Young women and girls are at greater risk of rape and sexual coercion because they are perceived to be more likely to be free from infection, or because of the erroneous but widespread belief in some regions that sex with a virgin can cleanse a man from infection. Fear of violence prevents women from accessing HIV/AIDS information, being tested, disclosing their HIV status, accessing services for the prevention of HIV transmission to infants, and receiving treatment and counselling, even when they know they have been infected, especially in settings where HIV-related stigma remains high.

Negotiation of safe sex: When women and girls have sexual relations, it tends to be with older men, increasing the likelihood that their partners are already infected. Social norms frequently hold that it is the male's responsibility to acquire condoms and for a young woman, to carry condoms suggests that she intends to have sex (Childhope 1997). At the same time, the prevailing norms in many settings dictate that since reproductive and sexual health are "female" concerns, women must be the ones to suggest contraceptive use[9]. It is especially difficult, if not impossible to negotiate condom use in the following settings: adolescent girls married with older men, women who experience physical or sexual violence, women who are economically dependant on their partner, newly wed women pressured to produce a child in order to be accepted by husband and in-laws, sex worker with clients paying more the usual rate for unprotected sex.

[9] While it is estimated that perfect use of the female condom may reduce the annual risk of acquiring HIV by more than 90% among women who have intercourse twice weekly with an infected male, the price of the female condom (4–10 times that of male condoms) makes it inaccessible to most women.

Lack of information (failure of HIV/AIDS prevention services): Because sex is a taboo topic in many countries, large numbers of young people do not get sufficient information or the skills to refuse sex or negotiate safer sex practices. That lack of knowledge magnifies their risk of HIV infection. While most young people have heard about HIV/AIDS, few know enough to protect themselves against infection[10]. In many countries HIV/AIDS information and services are provided primarily through family planning, prenatal and child health clinics, which are typically not designed to reach men or meet men's needs. As a result, men may be less likely than women to receive HIV/AIDS information, counselling and treatment services. Moreover, girls and women may be reluctant to seek advice, for fear of stigmatization.

2.3) Gender discrimination and vulnerability to HIV/AIDS epidemic

In addition to an increased physiological susceptibility to HIV infection, violations of women's rights heighten their vulnerability to AIDS. Women's subordinate position in society, discriminatory property and legal rights, are just some of the factors that sustain the escalation of the pandemic, and make women particularly vulnerable to HIV infection. If a woman does become infected with HIV or with any other STD, gender inequalities may affect the progression of the illness and possibly her survival chances. The impact of HIV/AIDS differs markedly along gender lines, reflecting men's and women's different roles and responsibilities in household and in the whole society.

2.3.1) Stigma, fear of abandonment and violence

Both women and men living with HIV/AIDS experience discrimination and stigma. However, there are gender differences in the way stigma affects women and men. Women may be more affected by stigma and discrimination than men because of social norms concerning acceptable sexual behaviour in women, and because women are often more economically vulnerable than men. The shame associated with AIDS is a major obstacle to its prevention, and the stigma that surrounds people living with HIV is compounded by discrimination against women. Many HIV-positive women avoid testing and treatment

[10] Surveys from 40 countries indicate that more than half of the young people have misconceptions about how HIV is transmitted. In Botswana, where one in three people is living with HIV/AIDS, virtually all young people have heard of AIDS and more than 75 per cent know the three primary means of protection. Still, 62 per cent of girls had at least one major misconception about how HIV is spread. Surveys in Cameroon, Lesotho, Mali and Senegal indicated that two thirds or more of young women (aged 15 to 24 years) did not know three HIV prevention methods.

services for fear of abandonment and other repercussions from husbands, families, communities and health providers. Both HIV negative and HIV positive women face violence in day-to-day life, but HIV positive women are at greater risk because of their HIV-positive status[11].

2.3.2) Widow inheritance and ritual cleansing

Wife inheritance and cleansing rituals are customary practices common in some communities in sub-Saharan Africa. The deeply rooted tradition of widow inheritance is widely practiced by Luo groups in Uganda, Tanzania, Zaire, and Sudan. A study conducted by Luke (2002) in the Luo community of Kenya points out that this practice of sexual networking whereby men who inherit widows have multiple sex partners, high frequency of exchange between widows, and low levels of condom use, however, encourages the spread of HIV. In some forms of ritual cleansing, a widow has to have sex with a social outcast who is paid by the dead husband's family, supposedly to cleanse the woman of her dead husband's evil spirits. In both practices, condoms are seldom used. These practices are allegedly protective for women; the true story is that these practices are predatory and exploitive and contribute to the spread of HIV/AIDS.

2.3.3) Pregnancy and childbearing

Pregnancy and childbearing raise specific issues for women. Studies from industrialized countries indicate that pregnancy does not affect the progress of infection in HIV-positive women who show no symptoms, or in those in the early stages of infection. Care should be taken, however, not to generalize these results to the developing world, since there has been as yet little research on this topic in such settings. As HIV can be transmitted through breast milk, breastfeeding presents a dilemma for many women. Those who decide to discontinue breastfeeding in favour of infant formula may reduce the risk of HIV transmission to their child, yet may expose the infant to diseases resulting from an unclean water supply, as well as to malnutrition. The use of infant formula can also alert others to the mother's HIV status and lead to stigma and discrimination.

[11] Gugu Dlamini, the first women to disclose her HIV positive status in South Africa was beaten to death in December 1998. Yet she lived in Kwa-Zulu Natal, a region where HIV prevalence rate at that time was estimated at 20-30% of adult population.

2.3.4) Denial of women's property rights and inheritance laws

Women's vulnerability to HIV is further exacerbated by unequal property and inheritance rights. In much of sub-Saharan Africa, property is usually owned by men, with women occasionally acquiring rights mainly by virtue of marriage. Multiple legal regimes overlap in many African countries, incorporating old colonial laws, more recent constitutional law, and ongoing customary law. The payment of bride-price upon marriage tightens men's control over women and property; in some countries women remain legal minors even after marriage (Human Rights Watch, 2003). The outcome is a status quo that often fails to recognize or uphold women's property rights, that reduces women's economic security and can lead to women having to endure abusive relationships or resort to sex for economic survival[12]. Impoverishment may force them to send some of their children away, engage in occasional sex for money or earn a living as commercial sex workers.

3) Macro-econometric evidence

Socio-economic inequalities are only some among many other determinants of HIV/AIDS epidemic. Other determinants include poverty, national income, income inequality, education, prevalence of STIs, etc… These determinants can be classified into socio-economic socio-cultural and epidemiological, as showed in Figure 3 below.

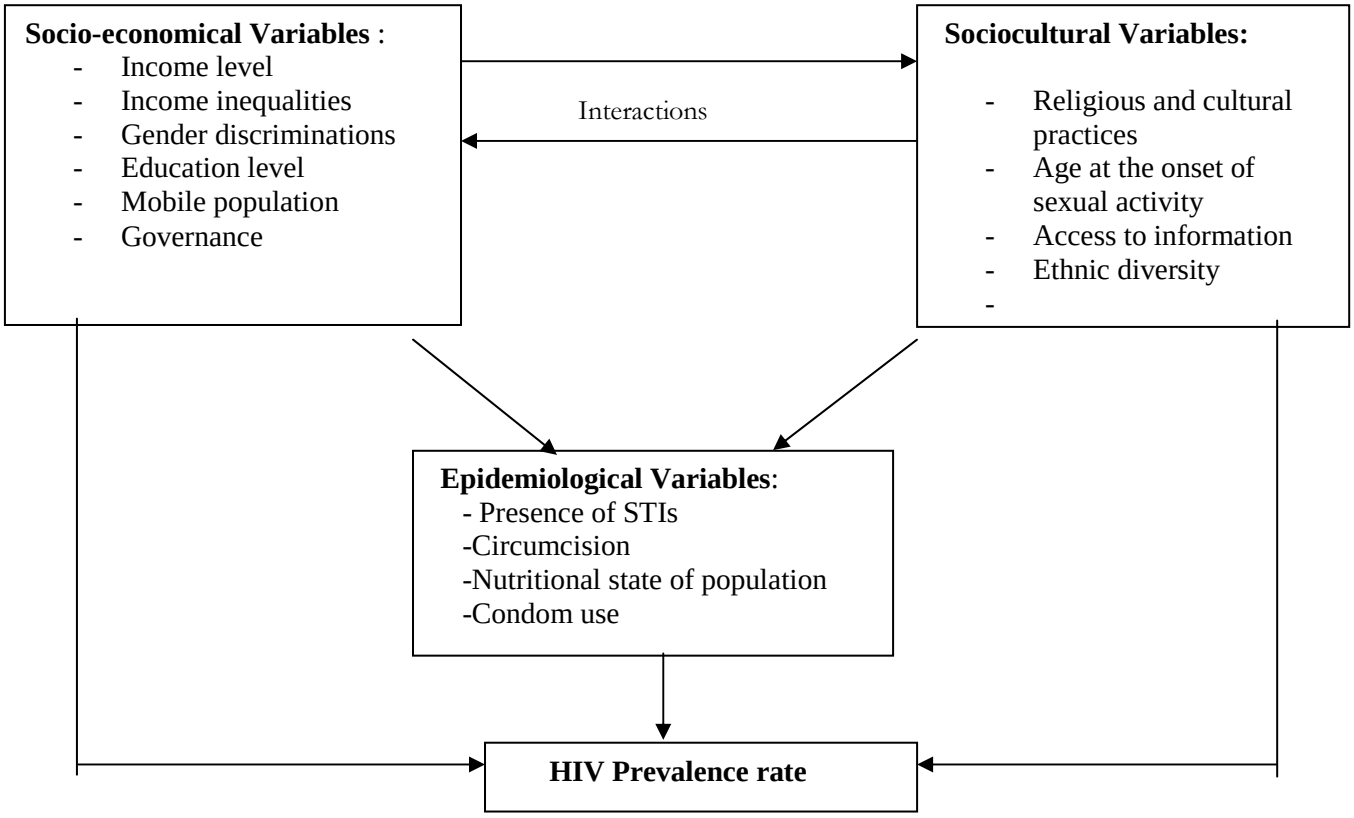
3.1) Data and descriptive statistics

3.1.1) Data

Before addressing the main issue of this paper, which is to assess the impact of socio-economic inequality on HIV/AIDS epidemic, we present a brief description of our data and justify the choice of the indicators for our variables of interest. Our analysis is performed using panel data for 42 Sub-Saharan African countries. These data come from various sources that we briefly present before taking a close look to data characteristics.

[12] In some countries, women whose male partners die of AIDS (many of whom are already infected with HIV) are subjected to property stripping by their spouses' relatives, they may have no legal rights to land and property (due to customary inheritance laws or the difficulties of enforcing existing remedial legislation) after their husbands' death.

Figure 3: The determinants of HIV/AIDS transmission at macro level



Gini coefficient will be our indicator for income inequality at country level. Since there is no equivalent of Gini coefficient for gender inequality, this variable will be captured through inequality in education, inequality in participation to economic life and maternal leaves benefits[13].

Table A.1 in the appendix provides a detailed list of indicators for our variables, as well as their source, with the first column reporting the signs coefficients are expected to take in our regressions.

[13] We tried here to capture the phenomenon in the fullest possible extent, without confining it to only one dimension. The different dimensions of gender discriminations do not always point in the same direction, so that there can be no or only little variation in the value of the index, while its constituents (especially women's education and economic activity/income, since length of maternal leave and percent of income perceived are time-invariant) have varied greatly in the same time. Moreover, there is no reason *a priori* that gender inequality's components affect the epidemic the same manner. In particular, within the same society, there can be a low level of women formal employment, but fairly good compensation for women during maternal leave, or even a long duration of maternal leave, but without preservation of wage. There can also be a case where women's education (both enrolment and literacy rates) are high, but do not necessarily mean that women have greater access to labour market. Thus, for this reason, we address the dimensions of gender inequality separately.

Table 4.1: Data and source

Variable	Sign	Indicator	Source
HIV prevalence rate		HIV prevalence rate in adult population (15-49)	AIDS Epidemic Update UNAIDS (1998-2006)
Income inequality	+	Gini coefficient	WIDER II, UNDP
Poverty	+	Malnutrition prevalence rate	Human Development Report (HDR) data (UNDP) 1995-2006
National income	-	Gross National Income, GNI (in Purchasing Power Parity US)	World Development Indicators database (World Bank) 2006
Access to information	-	Number of television sets for 1000 people	WDI (World Bank) 2006
Access to health care	-	Health expenditure per capita ($), Private health expenditure (% of total), Public health expenditure (% of total),	HDR(UNDP)1998-2006
Contraceptive use	-	Contraceptive prevalence	HDR(UNDP)1998-2006
Religious and traditional practices (circumcision)	-	Percentage of Muslim in total population	ACT (AIDS Campaign Team), World Bank
Ethno fragmentation	?	Ethnolinguistic fractionalization index	World Bank (Eastely and Levine, 1998)
Female income	-	Female estimated earned income (as % of male)	HDR (UNDP) 1995-2006
Female participation in economy	-	Percentage of women in total labor force, Female economic activity rate	HDR (UNDP) 1995-2006
Female literacy	-	Female literacy rate (> 15 years)	HDR (UNDP) 1995-2006
Maternal leave	-	Length of maternal leave (weeks), percent of wage perceived	Women's Indicators and Statistics Data base (Wistat), UN
Sea regional dummy	?	Dummy variable that takes the value 1 for countries located in southern and eastern Africa	Author

Source: Author

3.1.2) Descriptive statistics

Table 1 below provides a summary of our descriptive statistics.

Table 4.1: Summary Statistics

	Mean	Min	Max	Standard Total	Deviaton Within Country
HIV prevalence rate	8.14	0.07	38.8	8.56	2.83
Income Inequality (Gini)	47.79	28.9	76	10.84	4.87
Share of female labor (as ratio of male ratio)	73.15	33	102	14.78	3.34
Female income (as ratio of male)	58.56	23.7	100	14.13	6.18
Female literacy rate (as ratio of male)	50.70	7.2	93.9	22.76	5.04
Female combined enrolment rate (as ratio of male)	42.94	11	96	18.68	6.44
Wage during maternal leave (%)	80.35	0	100	30.45	0
Length of maternal leave (weeks)	12.25	100	16	2.50	0
GNI per capita (PPP)	1860	430	12450	2166.14	1248.84
Contraceptive prevalence	19.14	2	75	14.96	4.00
Malnutrition prevalence (%)	28.44	6	73	11.37	6.12
Adult Literacy rate (percent)	57.13	12.84	90	20.00	8.10
Health Care expenditure(per capita)	51.29	3	669	79.45	45.82
Telephone lines (for 1000 people)	20.44	0.18	287	42.37	6.41
Television sets (for 1000 people)	51.30	0.10	385.58	63.20	20.76
Ethnic fractionalization	65.96	4	93	23.01	0
Percentage of total population which is Muslim	29.75	0	100	31.33	0

3.13) Specification

The specification of a dependant variable depends on various parameters, including its natural evolution. Prevalence rates are percentages and as such, they are bounded to one or 100%. In addition, as prevalence represents the number of people infected with a disease at a given moment, it follows the typical evolution depicted by the epidemiological curve. Such a variable can be adequately analyzed only if the appropriate transformation is done. Thus, in order be able to apply any linear regression analysis to our nonlinear variable, need to transform the HIV prevalence rate, following Over (1998). The *logit*(*hiv*) is then defined as:

$$Logit(hiv) = \ln\left(\frac{hiv}{100 - hiv}\right)$$

Since there is no zero-value of the HIV prevalence rate in our sample, all observations can be transformed as *'logits*[14]*'*. These *'logits'* of prevalence rates are the variables we explain in our statistical analysis and this transformation renders it possible to use a linear functional form.

The random effects and the fixed effects models carry their own specificities and are relevant for different data. When performed on our data, the Hausman test does not favour the fixed effect model. Due to the specificities of our data, we prefer using random effects for two reasons; first, the time dimension of our panel is weak with only five periods and second, there are critical time-invariant variables in our series. The importance of cultural factors (ethnic fragmentation, traditional and religious practices) has been assessed in a previous study (Bonnel 2000). These cultural factors are the slowest to change over time and for our period of study (1997-2005), they are not likely to have changed.

In analyzing the impact of inequality on HIV/AIDS epidemic, our basic specification is given by:

$$logit(hiv_{i,t}) = \alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \varepsilon_{i,t} \qquad (1)$$

Where $i = 1, ..., N$ denotes countries,

$t = 1, ..., T$ denotes time periods;

hiv denotes HIV/AIDS prevalence rate,

I_{it} is the matrix of socio-economic inequality variables,

X_{it} is the matrix of traditional determinants covariates that always include period dummies in order to account for common shocks that affect all observations in a given period and

ε_{it} is a disturbance term *iid.*

3.2) Basic results

Table 2 below presents the results of equation (1), using Generalized Least Squares. Column (1) reports the results with all the variables, while column (2) adds a regional dummy to these variables.

[14] 100 is the maximum value for the prevalence rate.

Table 2: Socio-economic inequalities as determinants of HIV/AIDS epidemic

Dependant Variable	**Logit of HIV prevalence rate**		
Estimator	GLS	GLS	TSLS[15]
	(1)	(2)	(3)
Female participation to economic life	**0.000****	**0.000**	**0.003**
	(2.01)	**(1.57)**	**(0.39)**
Female enrolment rate (percent)	**-0.011****	**-0.012****	**-0.013***
	(2.19)	**(2.53)**	**(1.93)**
Maternal leave benefits	**-0.001***	**0.283**	**-0.002**
	(1.76)	**(1.43)**	**(0.67)**
Income Inequality (Gini)	***0.012****	***0.013****	***0.014****
	(2.26)	***(2.09)***	***(1.75)***
Log of GNI per capita (PPP)	0.009	0.010	0.004
	(0.35)	(0.39)	(0.02)
Malnutrition prevalence	-0.007	-0.008	-0.012
	(1.38)	(1.61)	(1.56)
Access to health care	0.006***	0.006**	0.007**
	(5.24)	(4.76)	(2.54)
Adult Literacy rate (percent)	0.017***	0.015**	0.012**
	(2.64)	(2.29)	(2.05)
Percentage of total population which is muslim	-.023***	-0.018***	-0.016***
	(4.07)	(4.30)	(3.52)
Ethnic fractionalization	0.017*	0.025***	0.020***
	(1.86)	(3.43)	(4.51)
Log of Television sets (for 1000 people)	-0.076	-0.108	-0.167*
	(0.85)	(1.35)	(1.84)
Contraceptive prevalence	-0.038***	-0.042***	-0.037***
	(3.62)	(4.51)	(4.55)
		1.478***	1.529***
Regional dummy 'Sea'		(4.34)	(5.77)
	-3.140***	-6.676***	-3.811***
Constant	(2.85)	(3.83)	(3.28)
R-squared (Overall)	0.57	0.77	0.751
R-squared (Between)	0.61	0.82	0.803
Hausman test p-value	(0.99)	(0.99)	
Rho (Fraction of variance due to *u_i*)	0.94	0.72	
Sargan-Hansen test	(0.05)[16]		
Observations	115	115	115
Number of pays	29	29	29

[15] In equation (3), the log of the lagged prevalence rate, the log of GNI and health care expenditure per capita are our endogenous variables. Weakly exogenous variables are female enrolment rate and the share of female labor (as percent of male) times income. The other variables are considered exogenous.

[16] The Sargan-Hansen test is not programmed on *stata* for the random effects model, so we had to perform it step by step. We first regressed the TSLS equation with the instruments and predict the residuals. Then, these residuals are regressed on the explicative variables, including the instruments. The R^2 of this late regression is the multiplied with the number of observations. The value obtained is the test statistics that follows a $\chi^2(F)$, with F, the degrees of freedom being the number of explicative variables. Then, the reading of the test for the pooled sample differs from that of the split sample. The test statistic (7.749) follows a chi-square with 2 degrees of freedom. So the probability to reject the null hypothesis (validity of the instruments) is the value in parentheses, which lies between 0.05 and 0.025.

(1) GLS regression with control variables only.
(2) GLS regression with inequality variables added to control variables.
(3) Two Sample Least Squares Estimation on the pooled sample (1997-2005)
* Significant at 10% confidence level
** Significant at 5% confidence level
*** Significant at 1% confidence level
All regressions used year-dummies. Absolute value of *t*-statistics in parentheses

As shown by our results, income and gender inequality variables are critical determinants of the epidemic. Gender inequality in education, maternal leave benefits and income inequality are relevant in explaining the epidemic.

Our results are quite contra-intuitive, since literacy rate, female participation to economic life and access to health care appear to favour the epidemic, but this upward bias in the epidemic has already been found using Sub-Saharan data (Lachaud, 2007, Nattrass 2004).

3.2.1) Spatial correlation

Moreover, when a regional dummy is added to the other variables, it helps us better understand the epidemic because it accounts for the specificities of Southern and Eastern Africa that are not captured accurately by our model. So, our model gains in explicative power, as suggested by the increase in the R-squared. Moreover, female participation to economic life does no longer appear to favour the epidemic. Among the covariates, income inequality, ethnic fragmentation, the percentage of the population which is Muslim, contraceptive prevalence, adult literacy rate and access to health care are also strong predictors of the epidemic.

3.2.2) Endogeneity issues

In column 3, we address the issue of potential endogeneity. In our analysis, there are some good reasons to suspect national income and access to health care of endogeneity in our model. First, the relationship between income and health is very often one of reverse causation[17] (Fuchs, 2004). Unless we correct for the potential endogeneity by using instrumental variables method, we can consider all the previous results biased. We used logarithm of lagged (5 years) values of GNI and the number of fixed telephone lines as instruments for the logarithm of the GNI. The GNI levels five years ago are strongly

[17] National income is basically defined as the overall wealth produced by all factors in the country. Yet, those factors, be it labour force or capital are affected by HIV/AIDS epidemic itself. In addition national income is also determined by some of covariates in our model such as education, ethno-linguistic fragmentation and the quality of governance.

corrected with the current GNI value (through growth rate), but not with the current HIV prevalence rate. The second instrument is the log of fixed telephones lines for 1000 persons. This variable is not likely to be correlated with the dependant variable, while it is obviously the case with country's GNI[18] .

Because AIDS-infected people are likely to experience more illness episodes, we assume that they will be in contact with health services more often. Meanwhile, access to health services increases the likelihood that sexually active people could seek care for STIs or in order to get tested[19]. The values of the Sargan-Hansen tests associated with our regressions can be read at the bottom of Table 2 and confirm the instruments utilized valid for the endogenous variables, national income and access to health care indicators.

3.3) Robustness

We tried to challenge the robustness of our previous result, first changing the specification of the dependant variable, second, performing a dynamic analysis of our model and finally, estimating our equation on incidence data[20].

3.3.1) Alternative specifications

In analyzing how our basic results might change according to the different specifications, we first use the fixed effects Hausman-Taylor estimator, keeping using the continuous *logit* as the specification of our dependant variable. Then, in order to make sure that our results are not sensitive to the linear functional form, we transform HIV prevalence rate, making it a binary variable. We chose the threshold of 10% for prevalence rate (as this is slightly higher than the average prevalence rate in our sample, 8.14%) for marker of a severe

[18] Unlike cell phones lines that depend on personal income, fixed telephone lines is part of a country's infrastructure and a country's ability to deliver adequate infrastructure directly influenced by its national income.

[19] A valid instrument for this variable would represent access to health care at best, without being correlated with HIV prevalence rate. We used immunization rate as a first instrument, since immunization occurs only at health facilities, but on babies so that it has no correlation with adult prevalence rate. A second instrument we used was the ratio of private to public health expenditure. The sum of the denominator and the numerator of this ratio is the value of our endogenous variable (health expenditure per capita), so they are strongly correlated. However, the structure of health expenditure in countries composing our sample is unlikely to have changed due to the epidemic.

[20] We used HIV/AIDS prevalence rate in adult population as our dependant variable. Nonetheless, this measure of infected people at a given moment is not the most relevant indicator for the evolution of an epidemic. The best way to measure it is to use incidence rates or new infections among the population. Regrettably, this indicator is very difficult to observe in general and more especially in the case of HIV/AIDS because of stigma associated with the infection. Fortunately, prevalence rate among young population can be used as a proxy for prevalence rate, since this age group entering into their active sexual life evolves over time. We thus estimate our model on two distinct sample, a big one made off 42 countries and five years (1997, 1999, 2001, 2003 and 2005) and a smaller one made off 20 countries and three years (2001, 2003 and 2005). The countries composing each sample are presented in Appendix 4.1 and Appendix 4.2.

epidemic. This binary variable is then estimated using a *probit* model first and then linear probability.

Table 3: Socio-economic Inequalities and HIV/AIDS epidemic: Alternative specifications of the model

	Logit of HIV Prevalence rate		
Dependant Variable			
Estimator	Fixed effects	Probit	Linear probability
	(1)	(2)	(3)
Female participation to economic life	***-0.000***	**-0.001*** **	**0.000**
	(0.24)	**(2.74)**	**(0.94)**
Female enrolment rate (percent)	***-0.014*** **	**-0.006**	**-0.010***
	(2.38)	**(0.24)**	**(1.76)**
Maternal leave benefits	***-0.000***	**0.000**	**-0.000**
	(0.53)	**(0.14)**	**(0.92)**
Income Inequality (Gini)	***0.014****	***0.122*** **	***0.018*** **
	(1.75)	***(3.66)***	***(2.43)***
Log of GNI per capita (PPP)	0.073***	0.336*	0.007
	(2.87)	(1.72)	(0.25)
Malnutrition prevalence	-0.006	0.006	-0.007
	(0.97)	(0.14)	(1.19)
Access to health care	0.006***	-0.004	0.005***
	(4.20)	(0.55)	(4.07)
Adult Literacy rate (percent)	0.009*	-0.005	0.014***
	(1.93)	(0.18)	(3.15)
Percentage of total population which is Muslim	-0.014**	-0.051*	-0.011**
	(2.23)	(1.85)	(2.20)
Ethnic fractionalization	0.018**	0.012	0.015***
	(2.64)	(0.88)	(2.60)
Log of Television sets (for 1000 people)	-0.198*	-0.757***	-0.114
	(1.68)	(2.77)	(1.18)
Contraceptive prevalence	-0.019**	0.004	-0.026***
	(1.99)	(0.17)	(3.12)
Sea (regional dummy)	1.385***	1.478***	1.165***
	(3.28)	(4.34)	(3.42)
Constant	-4.108***	2.649***	-4.434***
	(3.93)	(2.76)	(5.19)
R-squared (Overall)	0.50	0.50	0.65
R-squared (Between)			0.67
Rho (Fraction of variance due to *ui*)	0.79		0.76
Observations	116	116	133
Number of pays	29		33

(1) Fixed effects estimation using Hausman-Taylor estimator.
(2) Probit estimation, using 10% as marker for a severe epidemic.

(3) Linear probability, dependant variable = *logit(hiv)* with 100% as ceiling.

3.3.2) Dynamic analysis of the epidemic

Our period of study (eight years) is not a long period in regard of macroeconomic analysis. Nevertheless, it is a long period in the history of HIV/AIDS epidemic (25 years) and indeed, it covers the whole period for which routine data on the epidemic has been collected. Given the evolution of the epidemic, it is also a period during which significant changes are likely to have occurred in the impact of determinants or prevalence rate's acceleration. The epidemic was not certainly sensible to its determinants in 1997 in the same way it was in 2005. Moreover, among determinants, there can be a shift in the importance, so that determinants important at the beginning of the period of study are not the same at the end. To explore the potential difference in the way the epidemic is determined throughout the period of study, we split this period into two shorter sub-periods. Another way to perform a time analysis of our model is to allow past value of the dependant variable to be a determinant of the epidemic, a hypothesis we explore in a model of dynamic panel.

Evolution of the epidemic over the two sub-periods: The curve of the epidemic, which is not linear, implies that the impact of different variables is not constant over time. In fact, our period of study corresponds to the part of the curve from when the epidemic accelerates through hopefully the beginning of its plateau. The Chow test validates this intuition, being unable to reject the null hypothesis of difference in coefficients when our sample is broken into two sub-samples.

The breaking point for our sample is arbitrary, since the only valid reason we have to choose another date leads to two very disproportionate sub-samples[21] (a sample of one year and the other made off the other four years), which is not suitable for econometric analysis[22].

Dynamic evolution of the dependant variable: The curve of the epidemic follows an S-shaped curve and is not totally randomly determined. In particular, the growth rate of the number of new infections is positive, though decreasing. That means countries with already high

[21] We first thought of the date of creation of the Global Fund as breaking point for our sample, but the first grants were awarded in 2003 and are likely to influence prevalence rates for 2005 only.
[22] Furthermore, due to the small size of the second sub-sample, equation (1) was estimated using pooling (ordinary least squares that do not account for the random effect) instead of panel data on both sub-samples.

prevalence levels are likely to reach their plateau sooner, but that also means that these countries could have a higher number of new infections, since their initial prevalence rate is high. The sign of the lagged value of the dependant variable in the regression cannot be anticipated and depends on the magnitude of those two effects; the potential for new infection which is positive and the decreasing growth rate of prevalence that is negative.

The lagged value of the dependant variable is by construction an endogenous variable. If estimated with the GLS estimator, the subsequent estimation would yield correct, but not robust coefficients. The model that allows estimating lagged value of the dependant variable while correcting for this intrinsic endogeneity is the Generalized Method of Moments.

We use the GMM-system estimator for our dynamic estimation, where current values are used as instruments for exogenous variables; lagged values for one period are used as instruments for weakly exogenous variables[23] and lagged values for at least two periods are used as instruments for endogenous variables.

Leaving from our initial equation:

$$logit(hiv_{i,t}) = \alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \varepsilon_{i,t},$$

We now estimate the model:

$$logit(hiv_{i,t}) - logit(hiv_{i,t-1}) = \alpha_i^{'} + \beta_1^{'}(I_{i,t}) + \beta_2^{'}(X_{i,t}) + (\beta_{3i,t} - 1) logit(hiv_{i,t-1}) + \varepsilon_{i,t}^{'}$$

(2)

Where *t-1* denotes the previous period and $\varepsilon_{i,t}^{'} = u_i + \upsilon_t + e_{i,t}$.

This dynamic equation can also be written as:

$$logit(hiv_{i,t}) = \alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \beta_{3i,t} logit(hiv_{i,t-1}) X + \varepsilon_{i,t}$$

(3)

Table 4 summarizes the result s of the dynamic analysis of HIV/AIDS epidemic over the period of study[24]. Column (1) presents the results of equation (2) and the last two columns presents the results of equation (1) performed on the two sub-samples.

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[23] Weakly exogenous variables are defined here as variables that are predetermined or that can be influenced by past values of dependant variables, but which are not correlated with future realizations of error term.

[24] The Sargan-test associated with our estimation validates our choice for instruments in this model; there is also no second order autocorrelation in this model, as showed by the p-value (0.192) of Arellano and Bond test for autocorrelation.

The value of coefficient $\beta_{3i,t}$ is 0.625 and represents the impact of previous prevalence rate on current prevalence rate. But this is different from the value of previous prevalence rate on the evolution of prevalence rate, which is given by $\beta_{3i,t} - 1$. The command *lincom* programmed under *stata* allows us to have the value of this coefficient, which is -

Again, income and gender inequalities appear to significantly favour HIV/AIDS epidemic. In addition, poverty, that has had not been indicated as a significant variable so far, appears as a strong predictor of the epidemic. Among control variables, only the regional dummy is significative in addition to malnutrition prevalence, the indicator for poverty.

The results of the estimation on the split samples are also coherent with what has been previously found; socio-economic inequality increases the prevalence rate. However, gender inequality does not appear as a strong determinant of the epidemic in the first sub-sample. The significative covariates are the same for both sub-samples, except access to information, which is a strong predictor of the epidemic only for the first period 1997-2001.

0.262, significant at the 5% confidence level[24]. This negative sign translates into a convergence in prevalence rates, where high prevalence countries tend to reach their plateau before low prevalence countries, which also situates where countries in our sample are located on the epidemiological curve.

Table 4: Socio-economic inequalities and HIV/AIDS epidemic: Dynamic Analysis

Dependant Variable	**Logit of HIV prevalence rate**		
Estimator	Generalized Method of Moments	Two Sample Least Squares[25]	
	(1)	(2)	(3)
Female participation to economic life	**-0.000****	**-0.001**	**-0.039***
	(2.40)	**(0.14)**	**(1.71)**
Female enrolment rate (percent)	**-0.019**	**-0.025**	**0.002**
	(1.32)	**(1.21)**	**(0.09)**
Maternal leave benefits	**-0.000**	**-0.003**	**0.012**
	(1.21)	**(0.67)**	**(1.21)**
Income Inequality (Gini)	***0.015****	***0.033****	***0.070****
	(2.12)	***(2.43)***	***(2.73)***
Log of GNI per capita (PPP)	-0.027	-0.639	-0.360
	(0.74)	(0.60)	(1.02)
Malnutrition prevalence	-0.016*	-0.042*	0.000
	(1.80)	(1.74)	(0.01)
Access to health care	-0.001	0.018	0.001
	(0.36)	(1.05)	(0.11)
Adult Literacy rate (percent)	-0.017*	-0.242*	-0.007
	(1.78)	(1.93)	(0.40)
Percentage of total population which is Muslim	-0.008	0.023	-0.003
	(1.57)	(0.93)	(0.20)
Ethnic fractionalization	0.003	-0.013*	-0.004
	(1.24)	(1.71)	(0.26)
Log of Television sets (for 1000 people)	0.114	-0.242*	0.039
	(1.17)	(1.93)	(0.11)
Contraceptive prevalence	0.003	-0.053***	-0.030
	(0.31)	(5.09)	(1.36)
Sea (regional dummy)	1.034***	1.318*	2.697**
	(3.23)	(1.92)	(2.59)
Logit of lagged HIV prevalence rate	0.625***		
	(5.43)		
Constant	0.688	1.706	-2.793
	(0.79)	(0.21)	(1.04)
R-squared		0.75	0.40
A-R (1) *p*-value	(0.061)		
A-R(2) *p*-value	(0.192)		
Sargan-Hansen test *p*-value	(0.511)	1.417	0.737
		(0.492)	(0.691)
Observations	95(29)	81	45

(1) Estimation on the pooled sample using the Generalized Method of Moments.
(2) Two Sample Least Squares Estimation for the period 1997-2001
(3) Two Sample Least Squares Estimation for the period 2001- 2005

[25] Instruments used for these two equations are the same as those used in Table 2 Column 3.

3.3.3) Estimation with incidence data

As mentioned earlier, the indicator relevant for the evolution of an epidemic is incidence rate. However, since the number of new infections is very difficult to observe (if not impossible at all in the particular case of HIV/AIDS), the prevalence rate among young people is used as a proxy for the incidence rate.

Due to the small size of our incidence dataset, (20 countries and 3 periods), we perform bootstrap estimations in order to yield robust standard-errors[26]. McKinnon (2002) showed that bootstrap estimations works better than asymptotic estimations, except in three cases: serial autocorrelation, heterosckedasticity and simultaneous equations model. This is not our case, so we can confidently perform bootstrap estimations on our data.

Table 5 summarizes the results of the estimation of incidence rates on the same determinants used earlier for prevalence rates, using the Two Sample Least Squares in order to correct for potential endogeneity.

[26] Bootstrap is a method for estimating the sampling distribution of an estimator by resampling a number of times (in our case 500) with replacement from the original sample. It yields estimates of standard errors and confidence intervals of a population parameter like a mean, median, proportion, odds ratio, correlation coefficient or regression coefficient on small samples.

Table 5: Inequalities and HIV/AIDS epidemic: estimation with incidence rates

Dependant Variable	**Logit of HIV prevalence rate**	
Estimator	Two ample Least Squares	
	(3)	(4)
Income Inequality (Gini)	**0.022**	**0.022**
	(1.85)* ***(0.02)***	**(2.01)*** ***(0.81)***
Female participation to	**-0.031**	**-0.031**
economic life	**(6.03)*** ***(0.17)***	**(6.52)** ***(2.02)***
Female enrolment rate (%)	**0.043**	**0.039**
	(1.44) ***(0.06)***	**(1.46)** ***(0.69)***
Maternal leave benefits	0.382	0.330
	(1.30) *(0.01)*	(1.24) *(0.47)*
Log of GNI per capita (PPP)	-0.188	-0.183
	(0.60) *(0.01)*	(0.63) *(0.22)*
Malnutrition prevalence	0.058	0.056
	(2.84)****(0.58)*	(2.89)**** *(0.51)*
Access to health care	-0.276	-0.231
	(1.50) *(0.00)*	(1.38) *(0.54)*
Adult Literacy rate (percent)	0.009	0.010
	(1.04) *(0.01)*	(1.23) *(0.42)*
Percentage of total population which	0.007	0.007
is Muslim	(0.68) *(0.00)*	(0.71) *(0.15)*
Ethnic fractionalization	-0.005	-0.007
	(0.75) *(0.01)*	(1.02) *(0.29)*
	-0.084	-0.082
Log of Television sets (for 1000	(0.77) *(0.00)*	(0.78) *(0.20)*
people)		
Contraceptive prevalence	0.027	0.026
	(2.15)***(0.01)*	(2.19)***(0.49)*
	0.514	0.477
Sea (regional dummy)	(1.42) *(000)*	(1.40) *(0.43)*
	-5.804	-5.444
Constant	*(2.46)***(0.01)*	(2.42)***(0.49)*
R-squared (Overall)	0.929	0.93
R-squared (Between)	0.976	
Hausman test p-value		
Sargan-Hansen test p-value	(0.05)	(0.05)[27]
Observations	34(14)	34(14)

*Significant at 10% confidence level **Significant at 5% confidence level ***Significant at 1% confidence level

In columns (2) and (4), instruments used are the logs of lagged values of GNI (5 years ago) and the logs of the number of fixed telephones lines (for log of the GNI per capita) on the one hand and immunization rate and the ratio of private to public health care expenditure for health care expenditure per capita on the other hand.

(1) Generalized Least Squares, with a ceiling of 100 for hiv prevalence rate.

[27] The Sargan-Hansen test is not programmed on *stata* for the random effects model, so we had to perform it step by step. We first regressed the TSLS equation with the instruments and predict the residuals. Then, these residuals are regressed on the explicative variables, including the instruments. The R^2 of this late regression is the multiplied with the number of observations. The value obtained is the test statistics that follows a $\chi^2(F)$, with ***F,*** the degrees of freedom being the number of explicative variables. Then, the reading of the test for the pooled sample differs from that of the split sample. The test statistic (7.749) follows a chi-square with 2 degrees of freedom. So the probability to reject the null hypothesis (validity of the instruments) is the value in parentheses, which is 0.05.

(2) Generalized Least Squares, with a ceiling of 60 for hiv prevalence rate.
(3) Two sample Least Squares, with a ceiling of 100 for hiv prevalence rate.
(4) Two sample Least Squares, with a ceiling of 60 for hiv prevalence rate.

The results of the estimations with incidence rates are not far from those with prevalence rates. In particular, income and gender inequalities remain strong determinants of HIV/AIDS epidemic. Income inequality is also a significant variable with young people prevalence rate. The notable difference however with the results of the two dependant variables is the component of gender inequality that is important. In the adult prevalence rate, this component is women's education as showed by the coefficient and significative sign of female enrolment rate, whereas the pertinent variable with incidence data is women's economic power, which is robust even with the bootstrap estimation. Among the covariates, the only pertinent determinants assessed by incidence data are poverty, contraception use and the regional dummy. These variables carry the expected sign and are robust.

4) Impact of a reduction of inequalities on the epidemic

In the previous sections, we attempted to assert the importance of income and gender inequalities as determinants of HIV/AIDS epidemic. This impact is significant and robust to the alternative specifications of the model, of the dependant variables, and to the inclusion of lagged prevalence rate as a new variable in the model. The interpretation of the coefficients in the *logit* specification is not straightforward, so to be able to visualize the impact of inequality on the epidemic, we perform simulations based on our estimations, trying to assess the effect of the reduction of inequality on the curse of the epidemic[28].

Since the simulations are based on the predictions of our dependant variables, we first estimate the ability of these predictions to match the actual data, and then we assess the effect of a reduction in inequality on the dependant variables, HIV/AIDS prevalence rates.

4.1) Quality of the predictions

The quality of our predictions is not uniform across the whole sample. For some countries, the predictions are a close match of the actual data. In the most cases where the predictions

[28] The rationale behind our simulations is to estimate the model, with new values of inequality variables, but leaving all other variables unchanged. So the predictions are performed using the coefficients yielded by the initial regression.

are not accurate, there is an overestimation of the value of the dependant variable. Zimbabwe is practically the only case in which the value of the dependant variable is patently underestimated by our predictions. Examples of good predictions include Nigeria, Niger, Mali, Malawi, Madagascar, Central African Republic, Burundi and Cote d'Ivoire. The value of the dependant variable is overestimated in countries like Burkina-Faso, Cameroon, Ethiopia, The Gambia, Gabon, Guinea, Kenya, Botswana, Rwanda, Mauritius, Tanzania, Uganda and Zambia. Surprisingly enough, the quality of predictions is better with incidence data; the gap between actual and predicted data is on average 6.5%., with a minimum of 1.7% and a maximum of 13%. South Africa, Zambia, Zimbabwe and Botswana also perform well in this model.

4.2) Results and comments

When making simulations, it is important to make realistic hypotheses. Since income and gender inequality are shown to favour a tragic epidemic, our natural tendency is to sharply reduce them in order to maximize their impact on the epidemic's evolution. However, reducing income inequality is hard to implement in practice. This arduous task requires the implementation of many types of policy (redistribution, fight against poverty). In this section, we assign a reduction of 5% in income inequality, an objective which is ambitious, but not impossible in our context if political will is present.

The task is much easier with the reduction of our variables of gender inequality. Although increasing women's income or enrolment rates will not immediately translate into women being treated better in the societies in which they leave, these actions would nevertheless reduce HIV/AIDS prevalence rate[29]. We simulate an increase of girls' enrolment rate of successively 5%, 10% and 20%. For simulations on the first sample, we first simulate the reduction in gender inequality in education, and then we combine it to the reduction of 5% in income inequality.

Since the only inequality variable robust on incidence data after bootstrap estimations is females' income relative to males, only the change in this variable will be simulated on our second sample.

[29] Moreover, increasing women's income or enrolment rates is more feasible than reducing income inequality. The reduction of gender inequality in education is one of the MDGs and countries are allocated the means they need in order to meet the target assigned by the UN. Concerning women's income and participation to economic life, the microfinance institutions are already doing a great job in helping them to acquire a form of economic independence.

The effect of the different *scenarii* about the reduction of inequality on average HIV/AIDS prevalence rate is reported on Table 6. The range of this impact is a reduction of prevalence rate varying from 2% to 10.43% of the baseline value. The reduction of income inequality alone causes the least impact on prevalence rate, and this impact is at least doubled when combined with any postulated reduction in gender inequality in education. The most noticeable effect if obtained when increasing girls' enrolment rates by 20%, whether this measure is accompanied by a reduction in income inequality or not. Reducing income inequality alone lowers average prevalence rate by 2%, but when this measure is combined with the increase in girls' enrolment rates, its impact tends to disappear. Increasing girls' enrolment rates by 5% or 10% produces the same impact on average prevalence rate.

The reduction of gender inequality through an increase in women's income strongly affects the evolution of the epidemic among young people, as suggested by the results of our simulations. The proportionality factor here is almost of one to one, with 5% of increase in women's income translating into a reduction of average prevalence rate by 5%, as an increase of 10%, then of 20% translates into a reduction of 10% and 20% respectively in the average prevalence rate.

Table 6 below reports the effects of a reduction in inequality on the average prevalence rates in the two samples. The top part of the table represents the impact on adult prevalence rate and the other, the effect on prevalence rate among young population (15-24).

Table 6: Effect of the reduction of inequalities on HIV/AIDS epidemic (Average prevalence rates)

Change in income inequality(%)	Change in gender inequality (%)	Effect on average adult HIV prevalence rate (%)	Effect on young people prevalence rate (%)
ΔGini = 0	ΔGirls's enrolment rate = 0	Baseline	
ΔGini = 0	ΔGirls' enrolment rate = +5	- 6,25	
ΔGini = 0	ΔGirls' enrolment rate = +10	- 6,25	
ΔGini = 0	ΔGirls' enrolment rate =+20	- 10,34	
ΔGini = -5	ΔGirls's enrolment rate = 0	- 2,00	
ΔGini = -5	ΔGirls's enrolment rate = +5	- 4,15	
ΔGini = -5	ΔGirls' enrolment rate = +10	- 6,25	
ΔGini = -5	ΔGirls'enrolment rate = +20	-10,34	
ΔGini = 0	ΔWomen's income = 0		Baseline
ΔGini = 0	ΔWomen's income = +5		- 5,36
ΔGini = 0	ΔWomen's income = +10		- 10,43
ΔGini = 0	ΔWomen's income = +20		- 19,74

Source: Simulations based on author's calculations

This average effect, though important, masks significant differences across the sample. Indeed, as what could be logically expected, the impact is more striking among country with high initial inequality levels and high prevalence rates[30]. When looking at prevalence rate among adult population, in Botswana where the prediction is accurate for 2003, a reduction in inequality would have lead to a decrease in prevalence rate by 2 to 12%, according to the *scenarii* used to reduce inequality, whereas the reduction would range from

[30] The effect of the increase in women's relative income is a reduction on average prevalence rate among young population that ranges from 10% for Botswana in 2005 to 35% in Ghana in 2003. In Zimbabwe where our prediction almost matches the actual figure in 2005, an increase of women's income would have reduce prevalence rate among young population by 4.75% to 18%, according to the amount of this increase. Meanwhile, the same reduction gender inequality in income would have translated into a decrease of only 2.4% to 10% in Cote d'Ivoire.

1.6 to 7.5% in Burundi for 2001, a year when the model is also fit to predict the prevalence rate.

The interpretation of these results needs to be cautious, as there are large benefits in prevalence rates that do not necessarily translate into large benefits in terms of infected population, and similarly small benefits in terms of prevalence rates that correspond to large benefits in terms of infected population[31]. It is worth noting that the effect we computed considers the reduction in inequalities, holding everything else given, but precisely in this case, everything else is unlikely to remain unchanged. A reduction of income and gender inequalities is likely to have an impact on other determinants[32].

Concluding remarks

The debate on the link between inequality and health is not a new one. On the one hand, there seems to be some reasons to believe that people's health is affected not only by their absolute income, but also by the overall income gap in the society in which they live. This is obvious in rich countries if we consider that there are psychosocial factors that harm people's health when they are on a low level of the socioeconomic ladder. The explanation is less convincing in poor countries, as what should be important for people there for their health is only their absolute income or poverty level. Yet, there is comprehensive evidence that high levels of income inequality lead to less public spending, which affects the quantity of public resources that promote health. In poor countries, these are clean water and sanitation, well-functioning health facilities and skilled health staff. Thus in these countries, income inequalities also has a detrimental effect on health, in addition to the impact of poverty. Moreover, this detrimental effect will be perceived on everybody's health and not only on poor's health as is the case with poverty. It then seems that poor countries, which are until now excluded from the debate on income inequality and health are also concerned with the question. As for HIV/AIDS in Sub-Saharan Africa, it is unlikely that the impact of income inequality transits through psychosocial factors. It is more obviously the result of a mix between specificities due to the sample and those linked with the transmission of the

[31] This is precisely the case with the decrease in prevalence rate in Mauritius that ranges from 2 to 17%. Since population and prevalence rate in this country are low, the benefit in terms of infected people is not significant. On the other hand, Nigeria offers an example of much larger benefit in terms of infected population with a decrease in prevalence rate that ranges from 3.8 to 12.8%.

[32] As an example, throughout our analysis, contraceptive prevalence appears always significant. Enhancing girl's education and raising women's income would increase their awareness about the infection, preventive measures and make contraception more affordable. In this sense, the effect we predicted is a minimum effect.

virus, both of which magnify the link between income inequality and health. Regional migration, persistence of conflicts in Africa and health sector' crisis in the 90s further undermined the environment, allowing HIV transmission to occur even more easily.

On the other hand, there is evidence that women's health is affected not only by the average income and education in the society, but also by the distribution of wealth and education across both sexes. Thus, gender inequality and moreover gender discriminations have a detrimental effect on health, in addition to the impact of poverty. In this sense, commercial sex work, a coping mechanism for women struggling with poverty and lack of economic opportunities is also the vehicles of the epidemic. The weakness of our analysis lies in the lack of adequate data to measure what we indeed intend to. In that sense, some important determinants, namely those expressing behaviour could not be included in this analysis. Moreover, among variables studied, mobility, traditional and religious practices and most of our variables of interest do not adequately measure the variables. Despite this methodological weakness, our results are robust and suggest that there is indeed a specific link between income and gender inequalities and HIV/AIDS epidemic. The robustness of our results is ascertained by the use of alternative specifications for the model and when we perform a dynamic analysis of the epidemic.

These results suggest a new orientation in the fight against HIV/AIDS. As for income, instead of focussing only on poverty reduction, the strategies should also be directed toward narrowing income and education gaps between rich and poor, but moreover between men and women. Furthermore, tackling socio-economic inequalities would have a leverage effect, as these variables have a leading effect on most of the other determinants of HIV/AIDS.

Appendix

Table A.1: Country list (sample1)

Angola	Lesotho
Benin	Madagascar
Botswana	Malawi
Burkina-faso	Mali
Burundi	Mauritius
Cameroon	Mauritania
Central African Republic	Mozambique
Chad	Namibia
Congo, Rep	Niger
Congo, Dem Rep	Nigeria
Côte d'Ivoire	Rwanda
Djibouti	Senegal
Equatorial guinea	Sierra-Leone
Erithrea	South Africa
Ethiopia	Sudan
Gabon	Swaziland
Gambia	Tanzania
Ghana	Togo
Guinea	Uganda
Guinea-Bissau	Zambia
Kenya	Zimbabwe

Table A.2: Country list (sample 2)

Angola	Lesotho
Benin	Malawi
Botswana	Mozambique
Burkina-Faso	Rwanda
Burundi	South Africa
Chad	Swaziland
Cote d'Ivoire	Tanzania
Djibouti	Uganda
Ethiopia	Zambia
Ghana	Zimbabwe

Table A.3: Effect of the reduction of inequalities on HIV/AIDS epidemic in selected countries

Country (year)	Prevalence rate (actual value)	Prevalence rate (predicted value)	New value of prevalence rate*	Change in preva -lence rate** (%)
Botswana (2003)	37.3	37.25	[36.5 - 32.74]	[2.07 - 12.12]
Burundi (2001)	6.2	6.24	(6.13 - 5.77]	[1.63 - 7.5]
Centr. Af Rep(2001)	13.5	14.51	[13.09 - 13.5]	[2.88 - 6.71]
Madagascar (2001)	1.3	1.62	[1.6 - 1.44]	[2.21 - 11.4]
Malawi (2001)	14.3	14.3	[13.98 -12.13]	[2.20 - 15.14]
Nigeria (2001)	5.5	5.6	[5.46 - 4.98]	[2.46 - 10.91]
Nigeria (2003)	5.4	5.01	[4.88 - 4.30]	[2.5 - 14.11]
South Africa (2003)	21.5	22.85	[22.31- 19.32]	[2.33 - 15.43]
Botswana (2001)	33.6	31.7	[30.5 - 27]	[3.92 - 15.14]
Botswana (2005)	33.5	34.6	[33.7 - 31.3]	[2.41 - 9.47]
Burkina-Faso (2003)	2.3	2.5	[2,27 - 1.72]	[8.78 - 30.84]
Burundi (2003)	13.6	14.5	[13.37 - 10.3]	[8.13 - 29.23]
South Africa (2005)	25.2	25.1	[24.33 -22.14]	[3.05 - 11.73]
Tanzania (2001)	7.5	6.8	[6.17 - 4.62]	[9.06 - 31.84]
Togo (2005)	9.3	9	[8.84 - 7.9]	[3.32 - 12.67]
Uganda (2003)	10	8.6	[7.4 - 5.71]	[8.14 - 29]
Zimbabwe (2001)	29.8	29.6	[28.18 - 24.22]	[4.75 - 18.14]

Source: Simulations based on author's calculations

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