SEXUALLY TRANSMITTED INFECTIONS, SEXUAL BEHAVIOR AND THE HIV/AIDS EPIDEMIC *

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Abstract

Forty million people are infected with HIV worldwide; twenty-five million of them are in Sub-Saharan Africa. This paper addresses the question of why Africa has been so heavily affected by HIV, and what explains the variation within Africa. I present a model that decomposes epidemic level into differences in sexual behavior and differences in viral transmission rates. I argue, using evidence drawn from the existing medical literature, that Africa has very high HIV transmission rates, likely due to high rates of other untreated sexually transmitted infections, while transmission rates in the United States are low. The difference in transmission rates is large enough to explain the observed difference in prevalence between the United States and Sub-Saharan Africa. The model also provides a good fit to cross-country data within Africa and suggests that, in contrast to the intra-continental results, differences within that continent can be attributed to differences in sexual behavior and epidemic timing. The results suggest that cost-effective policy interventions would focus on decreasing transmission rates within Africa, possibly by treating other untreated sexually transmitted infections.

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I. Introduction

In 2003, 2.3 million people in Sub-Saharan Africa died of AIDS. In the same year, there were 3.1 million new HIV infections, bringing the total number of HIV infections to 25.4 million [UNAIDS 2004]. Though AIDS is a worldwide problem, Sub-Saharan Africa has been much more heavily affected than elsewhere. The prevalence rate for pregnant women (the most widely tested group) is 0.15 percent in the United States, and ten to fifteen percent in Sub-Saharan Africa. There is also enormous variation in HIV infection rate within Africa – the prevalence in Madagascar is under one percent but it is over twenty percent in Botswana, Zimbabwe and elsewhere [UNAIDS 2004]. To this point, the reason for the enormous variation remains poorly understood.

There are an array of possible explanations. The most straightforward are differences in sexual behavior – either differences in the number of partners [Kapiga et al 1994; Hunter 1993] or differences in sexual mixing patterns [Morris and Kretzschmar 1997; Hudson 1996; Anderson et al 1990]. Others have suggested that differences in HIV transmission rates due to differences in the incidence of other sexually transmitted infections (STIs) are responsible for the variation [Over and Piot 1993; Grosskurth et al 1995; O'Farrell 2001]. Differences in circumcision rates across countries within Africa have also been cited, with the theory that transmission of HIV is more efficient for uncircumcised men [Ferry et al 2001]. Finally, some recent work has argued for a major role for unsafe blood transfusions and other medical errors [Gisselquist et al 2003].

Despite the variety of possible explanations, virtually no attempt has been made to quantify differences in the conditions discussed above or to understand whether these

differences are sufficient to explain the variation in HIV rates. Understanding which of these possibilities – if any – is important enough to explain the path of the epidemic across space is key to identifying what type of policy interventions are most cost-effective.

This paper addresses this puzzle using a simulation model of the heterosexual HIV/AIDS epidemic to calibrate the importance of different factors in explaining the difference between Sub-Saharan Africa and the United States, and the differences across Sub-Saharan Africa. I argue that the difference in HIV prevalence between Sub-Saharan Africa and the United States is attributable to differences in transmission rates of the virus, while differences across Africa are due to differences in sexual behavior and epidemic timing.

The paper begins by presenting a general model of HIV epidemic growth that will be simulated using data on sexual behavior, transmission rates and other epidemic factors. Several simplifying assumptions allow the model to provide intuition as to the importance of transmission rates and sexual behavior in epidemic growth. In particular, I argue that small differences in transmission rates can produce very large differences in infection rates. The intuition for this is straightforward: Higher transmission rates produce more infections this period, and each new infected person can infect people next period, so the result of a higher transmission rate is multiplied many times over.

I use the simulation model to predict HIV rates in the United States and Sub-Saharan Africa. I present existing evidence (experimental and otherwise) drawn from the medical literature demonstrating that the HIV transmission rate (per unprotected sexual partnership) is much higher for individuals who have other untreated STIs, particularly those that cause open genital sores. Given that treatment levels for STIs are much lower in Sub-Saharan Africa than in the United States, this suggests that transmission rates may differ

between the two areas; indeed, I find that estimated transmission rates in Sub-Saharan Africa are much higher than those in the developed world. The transmission rate from men to women within one unprotected sexual partnership in Sub-Saharan Africa is around 30 percent; in the United States and Europe this figure is 10 percent.

In contrast, survey data shows little variation in sexual behavior between the United States and Sub-Saharan Africa. Cross-continent estimates of transmission rates and sexual behavior are embedded into the full simulation model. The model provides a good fit to the HIV rates in the two areas, and indicates that nearly all of the variation between the United States and Sub-Saharan Africa can be explained by differences in transmission rates. The model indicates that if Sub-Saharan Africa had the same transmission rates as the developed world, the HIV rate would be around 0.25 percent, rather than the estimated 12 percent with observed transmission rates.

I then turn to the differences within Africa and embed cross-country data on sexual behavior and other properties of the epidemic (including information on arrival time of the virus) into the simulation model. I find that the model is a good fit to the data on HIV rates across countries in Africa. It is worth noting here that the model is not fitted to the existing HIV rates; rather, sexual behavior parameters from survey data are taken as inputs and the predicted HIV rates from the model are compared with actual HIV prevalence. The results suggest that differences in sexual behavior and epidemic timing are sufficient to explain differences across countries in this region without any differences in transmission rates.

This result has two primary implications. First, the cross-country results from within Africa may be helpful for understanding the effect of other demographic variables on the HIV epidemic. Other literature has discussed the effect of variables such as income, education and

inequality on the HIV rate [Bonnel 2000; World Bank 1997; World Bank and Bonnel 2000]. The results here suggest that any variation arising from these variables must be due to their effect on sexual behavior, which makes calibrating the effect of changes in these underlying variables more tractable.

The second implication is that large gains may be possible in HIV prevention using readily available, off-patent drugs. Section VI uses the model in the paper to simulate the effect of two types of interventions and evaluate their effectiveness and cost-effectiveness. The results indicate that treating untreated bacterial STIs could prevent as many as 24 percent of new infections over the next decade, at a cost of less than $80 per infection, or around $3.67 per life year. Interventions to decrease sexual behavior are less effective, although they may also have a role.

Both types of interventions considered are more cost-effective than antiretroviral therapy, for which (generic) drug costs alone are $1 per day, or $365 per year, substantially higher than the estimated yearly cost of bacterial STI treatment. Although some policy-makers have focused on antiretroviral provision as the duty of rich countries and pharmaceutical companies (see, for example, Sachs [2000] and Sachs [2004]) the results here indicate that this type of treatment should not be the first line of defense.

II. HIV Epidemic Background

The Human Immunodeficiency Virus (HIV) is a retro-virus that attacks the immune system of the host individual, slowly invading and killing T-cells. As the disease progresses, individuals become increasingly susceptible to other illnesses. Eventually – usually within 7 to

10 years – the compromised immune system will lead to death through another proximate cause. An individual is said to have Acquired Immune Deficiency Syndrome (AIDS) once their immune system has been severely compromised.

The most common channels of transmission are sexual; the other major type of transmission is vertical – from mother to child – either in the womb, during birth, or while breastfeeding. HIV can also be spread through sharing needles (either by intravenous drug users, or poor hygiene in hospitals) and through transfusions with infected blood. The efficiency of these transmission mechanisms varies. Infection rates are higher for anal than vaginal sex, higher still for mother-to-child transmission, and extremely high (close 100 percent) for transfusion with infected blood.

Drugs that dramatically slow the progression of HIV have become available in recent years. Use of these regimens in the developing world is rare, due both to the cost of the drugs (even in generic form) and the difficulty of administering daily drug cocktails on a continent with few doctors. Interventions in Africa have focused more on prevention and treatment of opportunistic infections, including prevention of mother-to-child transmission, education about changes in sexual behavior, treatment of other sexually transmitted infections (STIs) and treatment of tuberculosis.

HIV was first identified in the gay community in the United States in the early 1980s, but the origin of the virus is in Africa, likely around the Democratic Republic of the Congo (former Zaire) [Vangroenweghe 2001; Sharp et al 2001]. There are large differences in HIV prevalence across the world, with Africa sustaining much higher HIV rates than elsewhere. In the United States, current prevalence among young gay men is around 7 percent [Division of HIV/AIDS Prevention 2004], but the virus has been limited in its impact. HIV prevalence

among pregnant women, for example, is only around 0.15 percent [Davis et al. 1998]. Europe has experienced even lower rates – the average rate among new blood donors (data are not available for pregnant women) in Western Europe in 2002 was only 6.1 infections for 100,000 donors [EuroHIV 2003]. On the other side of the spectrum, Africa has an overall HIV rate of 10-15 percent among pregnant women. Several countries in Sub-Saharan Africa (Botswana, Zimbabwe, South Africa, Swaziland, Lesotho) have estimated HIV rates over 20 percent, and as high as 35 percent. Other countries (The Gambia, Madagascar, Senegal) have HIV rates not much higher than those in the United States.

III. Theory and Decomposition

This section presents a framework for analyzing HIV transmission patterns and epidemic growth.[1] I first present the general setup of the simulation model that will be used in Sections IV and V, and then make a number of simplifications that provide explicit expressions for the evolution and steady state of the epidemic.

Very generally, the model used here divides the population into groups and then maps information on sexual behavior and viral transmission rates for each group into a predicted HIV rate. This mapping relies on two equations: an equation determining viral incidence and one determining prevalence. HIV incidence is defined as the share of uninfected individuals who become infected in a given period; prevalence is the total share of infected individuals. In the full simulation, incidence and prevalence for each group is calculated, and

[1]The model here considers only heterosexual disease transmission, although the general framework could be extended to the homosexual epidemic. The model also does not address mother-to-child transmission; although this is an important part of the epidemic, individuals infected at birth will not live to sexual maturity, so there will not be interaction between the share of a cohort infected at birth and adult HIV rate.

then used to estimate the overall HIV rate.

To get a sense of the structure of the equations before moving onto the details of the groupings, consider the incidence and prevalence among a single group of individuals. All members of the group have n homogenous partners in each period and the existing HIV rate among their partners is h. Further, the viral transmission rate per partnership (the chance of becoming infected by each partner) is β. The probability of an uninfected individual becoming infected is Bernoulli in partnerships (this is supported by evidence on HIV spread – see Kaplan[1990]). Given this functional form, incidence I can be expressed:

$$I = 1 - (1 - \beta h)^n \tag{1}$$

Overall prevalence in this population is a function of the existing infection rate (denoted m), the incidence and deaths. Assume that a share μ of infected individuals die each period. The new prevalence (m^*) is therefore:

$$m^* = \frac{(1-\mu)m + (1-m)I}{1-\mu m} \tag{2}$$

These two equations are the central workhorses of the model; given these, the only remaining task is to define the groups in the model and their relationship to each other. Note that a group must be a set of individuals that have the same transmission rates (β) and the same sexual behavior (including the number of partners (n) and the average HIV rate among partners (h)).

Consistent with this requirement, individuals are grouped based on gender, age, marital status and types of sexual partnerships. For example, 18-year-old single women who have casual sexual partners are one group, as are 18-year-old single women who do not have any sexual partners and so on. The population HIV rate, or HIV rate for more aggregated

groups (women, men, etc), is calculated by aggregating the group HIV rates based on group share. For example, the HIV rate among 18-year-old women is a weighted average of the HIV rate among 18-year-old single women and the HIV rate among 18-year-old married women, with the weighting determined by the share who are married.

Gender and martial status alone divide the population of each age into four groups; within each of these groups there are further subgroups based on sexual behavior. The types of partnerships allowed for in the model differ by marital status and gender. Single women (of each age group) are divided into two subgroups: Those with no partners and those with casual partners. Casual partners are drawn at random from the general population of men, and all those with casual partners have the same number. This is a good approximation a more general case in which some single women have one casual partner, some have two, etc, and it simplifies the analysis considerably since it implies that there are only two groups of single women in each age category.

Married women are also divided into two subgroups: Those who partner only with their spouse and those who also have extramarital partners. A spouse is assumed to be a random draw from the population of married men between 1 and 10 years older.[2] Again, as for single women, I assume that all married women with extramarital partners have the same number.

There are four subgroups of single men for each age category: Those with no partners, those with casual partners, those with female sex worker partners, and those with both casual and female sex worker partners. Casual partners are drawn at random from the

[2]In the countries used, 63 percent of women report their partner's age between 1 and 10 years older, with a roughly equal distribution across the ages. Fully 80 percent of women report that their husband is between 1 and 15 years older. Modifying the model to allow for this makes little difference and, in general, the simulation is not particularly sensitive to this assumption.

female population and female sex worker partners are drawn at random from the sex worker population. In parallel, married men of any age are in one of four subgroups: Spouse only, spouse and casual partners, spouse and female sex worker partners or spouse, casual and female sex worker partners.[3] The spouse is drawn from the population of married women 1 to 10 years younger.

Finally, female sex workers (FSWs) are modelled as a separate group. Consistent with anthropological evidence from Sub-Saharan Africa, the model has two types of FSWs: those engaged in casual sex work who have relatively few partners per year and those who work in brothels and have many partners [Wojcicki 2002; Nagot et al 2002]. Incidence and prevalence for the two groups are calculated separately, and overall prevalence is estimated by aggregating based on shares in each group.

The discussion above outlines the sexual behavior patterns in the model by defining groups and their relationship to each other. Turning to the evolution of the model: Gender-age cohorts are followed over time, with a period length of one year. The aging of cohorts over time has implications for the evolution of marital status and sexual behavior within a cohort. Actual data on share of people married by each single year of age are used to calculate the evolution of marital status; as the cohort ages, more individuals get married, but there is no divorce. To take a simple example, imagine that all 19-year-old women are single and have premarital sex and all 20-year-old women are married but only half of them have extramarital sex. This implies that when a cohort of women ages from 19 to 20 they will all acquire a spouse, half of them will be mapped into a group that has extramarital sex and the

[3]It is worth noting here that the number of casual partners that women have must be equal to the number of casual partners that men have. The fact that men generally report more risky sexual behavior means that the model is closed by the presence of sex workers.

other half will be mapped into a group that does not.

Deaths in this model are handled relatively simply: Individuals die ten years after contracting the virus. So deaths in any group this period are equal to the number of new infections in that group ten periods ago. There is also natural death, captured by exit from the model at age 60, consistent with pre-HIV life expectancy in the region. In addition, in each period a new group of uninfected 15-year-old men and women enter the sexually active population.

The final important factor in the model is group-specific viral transmission rates, which differ by gender and partnership type. Here, the transmission rate (β in equation (1) above) is defined as the chance that an uninfected individual becomes infected during a period-long partnership with an infected individual when there is no condom use. As is discussed in more detail in Section IV.A., viral transmission rates from men to women are higher (by a factor of 2) than from women to men. In addition, due to the extremely short length of partnerships with female sex workers, I assume the transmission rate is lower in these partnerships than in casual or spousal partnerships.

Although the per-partnership transmission rate as defined above is unchanged over time, the effective transmission rate will change with the introduction of condoms. In the simulation, information on the date of condom introduction and the growth of condom use over time is used to adjust transmission rates in each period. Condom use differs by gender, marital status and partnership type. There is assumed to be no condom use in spousal partnerships, and higher condom use in partnerships with female sex workers than in other premarital or extramarital sex.

A fuller description of the equations simulated can be found in Appendix A and in

the web appendix to this paper available on the Quarterly Journal of Economics website. Although the structure of the model is relatively simple, the large number of groups make the presentation of analytic solutions difficult. Therefore, I now make a number of assumptions that retain the structure of the model but simplify it such that explicit expressions for evolution and steady state are possible.

A Simple Example

I assume that there is no variation across groups in marital status or age and that male and female sexual behavior is identical. Sexual behavior is simple – a share $(1-\lambda)$ of individuals have one partner, and the remaining share λ have two partners. Transmission rates male-to-female are identical to those female-to-male, and equal to β. A share $\mu = 0.1$ of infected individuals die in each period (the magnitude is consistent with a ten year survival time and is a slight simplification). There is growth in the population each period consisting of new, uninfected individuals, with rate $\alpha = 0.025$[4] (in the full model this is accomplished by the introduction of young people).

Denote the infection rate for women in period t as w_t and for men as m_t. Given the assumptions, the equations for incidence and prevalence outlined at the start of this section combine to yield:

$$m_t = \frac{(1-\mu)m_{t-1} + (1-m_{t-1})((1-\lambda)\beta w_{t-1} + \lambda(1-(1-\beta w_{t-1})^2))}{1-\mu m_{t-1}+\alpha} \tag{3}$$

$$w_t = \frac{(1-\mu)w_{t-1} + (1-w_{t-1})((1-\lambda)\beta m_{t-1} + \lambda(1-(1-\beta m_{t-1})^2))}{1-\mu w_{t-1}+\alpha} \tag{4}$$

Note that these equations imply that infection rate among men and women are identical in each period. I am interested in both the evolution of the model over time and the steady

[4]The size of α makes little difference as long as it is larger than zero; I have chosen 0.025 because it is consistent with growth if the population contains people ages 15 to 60 and one age group of uninfected individuals enters each period.

state. Evolution can be easily calculated from equations (3) and (4) above.

Turning to the steady state, there is a trivial steady state at zero – if the virus is never introduced then it is not able to spread. Once the virus is introduced in any positive amount, then there is a unique stable steady state of the model that is strictly positive as long as the transmission rate is sufficiently large ($\beta > \frac{\mu+\alpha}{1+\lambda}$). The positive steady state level $w^* = m^*$ can be expressed:

$$(5) \quad w^* = m^* = \frac{-\mu + \lambda\beta^2 + \beta + \lambda\beta - \sqrt{(\mu - \lambda\beta^2 - \beta - \lambda\beta)^2 - 4\lambda\beta^2(\beta + \lambda\beta - \alpha - \mu)}}{2\lambda\beta^2}$$

To facilitate intuition, the importance of β and λ can be illustrated graphically, as is done in Figure I. This figure shows the steady state of the model and the twenty-year infection rate graphed against the viral transmission rate β (when calculating the twenty-year infection rate I assume the virus was introduced with 1 percent prevalence in the first year). Figure I also illustrates the effect of variation in λ, showing the infection rates for $\lambda = 0.25$ (25 percent of the population has two partners) and $\lambda = 0.5$ (50 percent of the population has two partners).[5]

Figure I demonstrates the importance of transmission rates in the course of the epidemic. Estimated transmission rates (these estimates are discussed in more detail in Section IV.A) for the United States and Sub-Saharan Africa are marked on the figure: The difference in transmission rates across these two areas translates to very large differences in infection rates. The twenty-year infection rate at the United States level of transmission is virtually zero; in the case of Sub-Saharan Africa, it is between 10 and 20 percent. Changes in the level of λ also play a role in the epidemic, but the magnitude of the difference

[5]Unlike the transmission rate, there is more than one way to change sexual behavior. Figure I shows only one possible mechanism and should be viewed as an example.

(particularly when considering steady state levels) is relatively smaller.

A final note before moving on to the simulations: The model presented here relies on group-specific homogeneity in sexual behavior. Between periods transition is random with respect to behavior, so individuals with high rates of casual sex in this period do not have a greater probability of having casual sex in the next period. This is a potentially restrictive assumption, and likely to be at least somewhat unrealistic. However, robustness checks on the simulation (available from the author) suggest that even introducing a very extreme form of heterogeneity into the model (i.e. there is a high sex group and a low sex group and no movement between groups over time) makes relatively little difference in the results.

IV. Sub-Saharan Africa and the United States

This section calibrates the model above using data on sexual behavior and transmission rates from the United States and Sub-Saharan Africa. The results speak to whether the large differences in HIV infection rates between these regions should be attributed to differences in behavior, in transmission rates or in something else. It is important to note that if the model above is not able to fit the magnitude of the differences in HIV rates, that would suggest a role for other factors.[6] The first subsection below discusses transmission rates in the two regions. The second subsection outlines the data on sexual

[6]I am ignoring the possibility that an epidemic among homosexuals and bisexuals could impact the HIV rates among heterosexual groups. In Africa this seems realistic, as most experts think that nearly all HIV infection is due to heterosexual contact: in 2002 the World Health Organization estimated that 99 percent of HIV infection among adults was due to heterosexual contact [World Health Organization 2002]. Since this includes infections among men, it suggests there is little role for non-heterosexual contact. In contrast, in the United States it is more likely that some infection among women is due to contact with bisexual men. For example, one survey of HIV infected individuals [Montgomery et al. 2003] indicates that 13 percent of the White men and 34 percent of African-American men who have sex with men also have sex with women. This suggests that bisexual behavior may play a role in the United States and that the HIV rates seen might be an overestimate of what would be experienced in the absence of any of this activity.

behavior and the third presents results.

IV.A. HIV Transmission Rates

Before moving into a discussion and estimation of transmission rates, it is useful to define the "HIV transmission rate" – β from the equation (1) in Section III. The "per-partnership transmission rate" is the chance of an uninfected person becoming infected during an entire partnership (including what may be many sexual contacts) with an infected person, with no condom use.[7] Condom use does play a role in the simulation, but will be introduced separately from transmission rates.

It is widely accepted that HIV transmission rates vary across methods of infection – transmission rates are close to one for transmission with infected blood, around 30 percent for mother-to-child transmission and higher for homosexual than heterosexual partnerships [Royce et al, 1997]. This paper deals exclusively with heterosexual viral spread, but there are still good reasons to believe that transmission rates may vary widely across space. Perhaps the most widely cited reasons for this is variation in rates of other sexually transmitted infections (STIs), which are thought to increase the transmission rate of the virus (for a review of this theory, see Kapiga and Aitken [2003]).

Untreated STIs, particularly herpes and syphilis, cause open genital sores that

[7] I use per-partnership rather than per-contact transmission rates in this paper. There exists good evidence that the relationship between HIV transmission in a partnership and number of sexual contacts with the partner may be highly nonlinear [Kaplan 1990]. For example, studies of infected hemophiliacs and their uninfected partners suggested that the length of partnership had no affect on probability of infection [Ragni et al 1989; Downs and Di Vincenzi 1996]. In addition, there is evidence that reporting using condoms "sometimes" provides no more protection than reporting no use, suggesting that lowering the number of unprotected sexual acts has little effect, assuming that there are at least some unprotected acts [Di Vincenzi 1994]. Both of these pieces of evidence support a "lock-and-key" interpretation of HIV transmission – infection either happens within a partnership or not. This supports the use of per-partnership rather than per-act transmission rates

dramatically increase the probability of blood transmission during sex, and therefore increase the probability of HIV transmission. Most of the evidence on the STI-transmission relationship is non-experimental. Genital ulcers have been shown in a number of studies [Gray et al. 2001; Di Vincenzi 1994] to be associated with higher rates of HIV transmission. Non-ulcerative STI infection is associated with a greater HIV viral load in the semen of infected men [Cohen et al. 1997]. Increased viral load is associated with increased chance of infecting one's partner [Gray et al. 2001; Di Vincenzi 1994]), implying that untreated STIs may increase not only the chance of any individual contracting HIV, but also the chance of infecting someone else.

There are also examples of randomized controlled trials to address this question. Grosskurth et al [1995] use a randomized controlled trial and find large decreases in HIV incidence in Tanzania when STI treatment is offered. In contrast, Wawer et al [1999] finds no effect of STI treatment on HIV transmission in Uganda, though this result may be due to other interventions going on simultaneously in Uganda, relative infrequency of STI treatment and the stage of the epidemic [Kapiga and Aitken 2003]. It is also worth noting that, especially given the non-experimental evidence, the conflicting estimates are more likely to suggest differences in the success of different forms of intervention than different conclusions about the effect of STIs on HIV transmission.

Sub-Saharan Africa has much higher rates of STIs than the developed world, with the prevalence of untreated curable STIs in Africa estimated around 11.9 percent, versus 1.9 percent in the United States and 2 percent in Western Europe.[8] In addition, untreated sexually transmitted viral infections (in particular genital herpes) are much more common in

[8]http://www.avert.org/STIstatistiFSWorldwide.htm

Sub-Saharan Africa and other developing countries. This evidence suggests there are good reasons to believe that HIV transmission rates are higher in Sub-Saharan Africa than in the United States.[9] This evidence does not, however, provide estimates of these rates. For that, I turn to the medical evidence on viral spread and implied transmission rates.

There are no widely accepted estimates of the rate of HIV transmission in heterosexual partnerships, particularly in the developing world. In the developed world, most studies on this issue rely on spousal partnerships where one spouse is a hemophiliac. In Panel 1 of Table I, I list a set of studies that report transmission rates within heterosexual partnerships in the developed world. These studies generally include only hemophiliacs, although some cover transfusion recipients or mixed groups as well.[10] Although the data are relatively sparse, particularly for female-to-male transmission, the transmission rates are quite low. In the rest of the paper I will use a sample-size weighted average of these rates – a per-partnership transmission rate of 10 percent male-to-female and 5 percent female-to-male.[11]

A number of studies in Sub-Saharan Africa have, either directly or indirectly, provided transmission rate estimates. For the purposes of the estimation used in this paper, transmission rates are calculated for four prospective studies of HIV incidence in Africa (these are the only prospective studies for which all the relevant data are available). All studies follow a cohort over time and record the number of new HIV infections over the period, as

[9] Although other STIs are the most prominent explanation for differences in transmission rates, there is also evidence that other factors may play a role. For example, infection with malaria appears to increase viral load, which would, in turn, increase the chance of infecting a partner [Corbett et al. 2002; Quinn et al. 2000; Kapiga et al. 2002].

[10] Transmission rates in the "mixed" group are likely to be higher because they may include women who have IV drug user partners, and are thus more likely to be users themselves.

[11] These studies cover both the United States and Europe. The estimation, however, focuses on the United States. I use an average of all the studies due to sample size issues, but it is worth noting that there is no large difference between the estimates for the US and Europe (particularly when identical groups are compared). This is not surprising given the similar rates of untreated STIs and, generally, similar medical availability.

well as information on sexual behavior. For each study information is recorded on the number of new HIV infections, the average number of sexual partners for people in the study, the latent HIV rate among individuals of the opposite sex in the region and the rate of condom use. From these studies, the per-partnership transmission rate is calculated by inverting the process detailed in Equation (1) in Section III.[12]

The second panel of Table I reports the estimated transmission rates from the four studies. It is clear that these rates for Sub-Saharan Africa are somewhat higher than those in the developed world. Throughout the rest of the paper I use a sample-size weighted average of the transmission rates in Panel 2 of Table I: 27 percent male-to-female and 12 percent female-to-male. I use a slightly lower transmission rate for male contact with female sex workers, because sexual contacts are much less frequent in these partnerships [Cameron et al 1989].

It is worth noting that the populations used to calculate transmission rates in Panels 1 and 2 differ. Ideally, there would be data on transmission in the general population for both areas; given the extremely low level of HIV in the general population in the United States and Europe, this is not possible. Therefore, the rates for the United States and Europe may be an overestimate of the transmission rate in the general population. Hemophiliacs and transplant recipients are more likely than the general population to have other STIs, due to transfusions with untested blood, and are generally less healthy. Estimates for drug users may overstate the effect of sexual transmission because some needle transmission is ignored. Overall, this

[12]The procedure for doing this is relatively simple: Each study records the share of uninfected people in the study who become infected – this is the incidence I from Equation (1). In addition, the studies record latent HIV rate among the opposite sex (h from equation (1)) and the average number of partners, which corresponds to n. Given the values of I, h and n it is simple to invert equation (1) to yield $\beta = \frac{1-\sqrt[n]{1-I}}{h}$. If there is condom use among individuals in the study then the β calculated in this way will need to be adjusted – if condoms are used a share c of the time, then the true β per unprotected partnership is estimated by dividing by $1-c$.

suggests the transmission rate difference between the United States/Europe and Sub-Saharan Africa is, if anything, understated by these data.

Before moving on to data on sexual behavior, it is worth mentioning the possibility of transmission rate heterogeneity across sexual partnerships – i.e. partnerships between two individuals with STIs result in infection more often than partnerships where only one or neither partner is infected. With this modification to the model, the predicted infection rate is almost identical to what is calculated in the original model as long as the weighted average transmission rate remains the same.

IV.B. Sexual Behavior

In the model presented in Section III four parameters are required to predict HIV rates: Data on the distribution of sexual partnerships within each age-gender-marital status cohort, data on the number of sexual partners for those having sex outside of marriage, data on condom use and data on the share of individuals in each age-gender cohort who are married. All data for Africa come from the Demographic and Health Surveys (DHS)[13] for 14 countries and, for the United States, from the General Social Survey (GSS) [Davis, Smith and Marsden 2002]. Data for Africa in this section is a population-weighted average of the data for the 14 countries.

The DHS surveys are household surveys run in many countries in Sub-Saharan Africa (and elsewhere), beginning in the late 1980s. In a subset of the surveys individuals are asked about their sexual behavior outside of marriage, including questions to unmarried individuals about premarital sex and questions to married individuals about extramarital sex.[14] For each

[13]Source: DHS datasets, www.measuredhs.com, MEASURE DHS, Macro International Inc

[14]There are obvious potential problems with underreporting. They are, in many ways, unavoidable. Nearly all surveys on sexual behavior are subject to downward biases. This is discussed in more detail in Section V, but

individual (single and married) in the DHS survey, an indicator is created for whether or not they report having had a non-marital partner in the last year. These data are then averaged by age-gender-marital status cohort to create a measure of the share of individuals in each group having sex outside of marriage. A similar exercise is done for the number of partners for each gender-marital status cohort (these data are not divided by age due to sample size limitations).

Women in the model have sex with a spouse, if married, and may also have casual partners. The information from the DHS surveys is therefore sufficient to fully calibrate the model for women. For men choices include a spouse (if married), casual partners and female sex workers (FSWs). The data directly from the DHS are therefore not sufficient, and it is necessary to make some assumptions about the distribution across these groups of men. Educated assumptions (based on information from the literature) are made about the share in each group and calculations are done assuming a closed system and using information on female casual partnerships (details are in Appendix B).

Data on the same sexual behavior parameters for the United States are drawn from the General Social Survey (GSS), an individual-level survey conducted almost yearly since 1972 on a representative sample of adult Americans. In addition to basic demographic information, for years between 1988 and 1994 individuals were asked about their non-marital sexual partners, including whether they have had any in the last year and how many.

The same datasets – the DHS and GSS – are used to calculate marriage rates by age-gender cohort in the two areas. Finally, the DHS and the GSS provide point estimates on

it is worth noting here that this is probably less of an issue for the United States-Africa comparison than for the comparison within Africa. In particular, people in the United States are probably more likely to underreport non-marital sexual behavior, so any bias will bias in favor of concluding that sexual behavior plays a larger role.

condom use late in the 1990s for these Sub-Saharan Africa and the United States, respectively. For Africa, I assume that condom use increased linearly from zero beginning in the first year of "condom social marketing" (as reported in the World Bank Millennium Indicators).[15] In the case of the United States, I assume low but non-zero levels of condom use at the beginning of the epidemic, with an increase when the epidemic arrived [Anderson, Santelli and Mugalla 2003; Smith 2003].

Condom use is differentiated across gender, martial status and partnership type. In the DHS surveys it is possible to calculate different rates of condom use across gender and martial status. In general, men use condoms slightly more than women and single individuals more than married ones (even in non-spousal partnerships). In addition, for a subset of the DHS surveys information is asked not only about condom use with non-spousal partners, but also about condom use with particular partner types. From these data I infer (for men) the share of time a condom was used with a FSW, relative to the average. Using this information I adjust the data to allow higher condom use with FSWs than with non-spousal partners from the general population. Data on condom use in the United States are drawn from survey results reported in Anderson, Santelli and Mugalla [2003] and Smith [2003].

Before moving on to the results, Table II presents a side-by-side comparison of the sexual behavior parameters used in the analysis here for the United States and Sub-Saharan Africa. For descriptive purposes the data have been aggregated by age, so it is only gender-marital status specific. In addition, rather than reporting share married by age, I report it for two specific age groups to give an overall sense of the marital status patterns.

[15]This appears to be roughly consistent with the pattern of condom sales in Kenya and Cameroon (Hearst and Chen, 2004)

Condom use reported is the average for non-sex worker partnerships.

Also included in Table II are data on HIV rate in the two regions. For Sub-Saharan Africa, the data come from the U.S. Census HIV/AIDS Surveillance Database. This database collects all studies of HIV/AIDS prevalence since the early 1980s and extracts information on number of subjects, prevalence, population and other factors. From this, I create an average prevalence rate for pregnant women in the late 1990s in the countries for which sexual behavior data are available. This will then be matched with the predicted rate for women of child-bearing age from the simulation model.[16] For the United States, data on HIV rates come from the CDC [Davis et al 1998], and are aggregated from hospital data on HIV rate among pregnant women by state.

It is worth noting here that in both this section and in Section V the data for Africa are limited to urban areas. Relatively little antenatal clinic testing is done in rural areas so limiting the HIV data to urban areas seems the most reliable. Similarly, the data on sexual behavior are limited to respondents from urban areas.

Table II demonstrates that, with the exception of the rates of condom use, the United States and Africa look very similar on parameters of sexual behavior. Single individuals in the United States are more likely to have premarital sex, whereas married individuals in Africa are more likely to have extramarital sex. Although condom use is higher in the United States, it is quite close to the top of the range for the African countries in the sample. I show in the next subsection that differences in condom use are not driving the differences in the epidemic.

One issue that is not addressed in Table II is the possibility that frequency of sexual

[16]By using the actual and predicted rates for pregnant women, I avoid many of the issues that have been recently noted in surveillance data in which the rate for the entire adult population is estimated from that for pregnant women. A separate issue is the possibility that HIV lowers fertility (as in Young [2005]). If this is true, then rates for pregnant women in the model may overstate the rates for pregnant women overall.

contact within a partnership is higher in Sub-Saharan Africa, and this difference in behavior drives differences in transmission rates. There are reasons to think this is unlikely to be an important factor. First, as noted in Kaplan [1990], Saracco[1993] and discussed in Section IV.A., transmission probability does not seem to vary much with length of partnership within a steady relationship. Second, survey data suggests similar sexual frequency. In the GSS, among married individuals, the median person reports sex with their spouse once weekly. Similarly, in the DHS surveys used here, 51 percent of the respondents report sex with their spouse within the last week, but not more frequently. Although the results are not directly comparable, as the question was posed somewhat differently, they suggest similar behavior in the two regions and do not provide support for a theory of differences in contact frequency.

Two final notes before moving on to the results concern the introduction of the epidemic into each area. I am concerned here with estimating the predicted HIV rates at the end of the 1990s; this necessitates the assumption of an epidemic starting date. I will assume that the epidemic started in the first years of the 1980s in both regions. Further, I assume that the epidemic is introduced into the population by FSWs. Based on data on the HIV rates among FSWs from the U.S. Census Surveillance Database, I assume that in Africa at the start of the epidemic FSWs have an HIV rate of 10 percent. For the United States, I assume an HIV rate of 5 percent [MMWR 1987; MMWR 1989].

IV.C. Results

Figure II illustrates the central result in the paper. The first columns show the HIV rates predicted by the model in the United States and Sub-Saharan Africa using the actual

transmission rates and sexual behavior parameters for each region. The model provides quite a good fit to the infection rates in the two regions. In the United States, the estimate is around 0.23 percent, quite close to the actual prevalence estimate of 0.15 percent. Similarly, in Africa, the estimate is 12.7 percent, versus an actual estimated prevalence rate of 11.9 percent in the countries considered. Considering only the first set of columns, the results suggest that the model is able to fit the magnitude of the difference between the United States and Africa quite well.

The second and third sets of columns in Figure II consider two counterfactuals. The second set of columns uses the model to simulate infection rates if both areas experienced the transmission rate seen in Sub-Saharan Africa, given their observed sexual behavior; the third simulates infection rates if both areas experienced the same sexual behavior as Sub-Saharan Africa (including condom use), but retained their observed transmission rates. Together, these columns demonstrate that the difference across regions is being driven by differences in transmission rates and not by sexual behavior. The simulated rate for the United States under African transmission rates is over 11 percent, whereas the simulated rate under African sexual behavior is only 0.3 percent.

An alternative way to explore what is driving the differences between the United States and Sub-Saharan Africa is to estimate what level of sexual behavior in Africa would produce the current HIV rate if transmission rates were equal to those in the United States. There is more than one way to alter the parameters of sexual behavior to achieve this, but it is worth considering the simplest – changing the share of people with casual partners and the number of casual partners. The results demonstrate that extremely large increases in sexual behavior would be necessary to produce the rate seen in Africa with the transmission rates

from the developed world. One possible mechanism includes everyone having non-marital sex (everyone has both premarital sex and extramarital sex in all periods), all women having an average of four non-marital partners and all men having an average of five non-marital partners per year. Such a scenario involves sexual behavior that would have to be many times higher than in the reported DHS data summarized in Table II.

V. HIV Across Countries Within Africa

Section IV argues that the difference in HIV rate between the United States and Sub-Saharan Africa is due largely to differences in transmission rates, with relatively little (if any) role for differences in sexual behavior. I move now to considering differences in infection rates across countries within Africa. HIV rates vary quite widely across Africa – from a low of less than 1 percent to a high of over 30 percent. The results below suggest these differences can be attributed to differences in sexual behavior and epidemic timing, and do not rely on differences in transmission rates.

V.A. Cross Country Data

Unlike in Section IV, in this section I assume the same transmission rate for all countries within Africa. This is potentially problematic, given that all of the studies in Panel 2 of Table I took place in East Africa, raising the possibility that transmission rates in West or South Africa might be different. In particular, given the lower HIV rates in West Africa, one might suspect that lower transmission rates are the cause.

Unfortunately, there are no prospective studies from West Africa of the type used in Table I so it is not possible to directly calculate transmission rates. However, rates of other

STIs, which are important determinants of transmission rates, do not seem to differ significantly across the regions (see Buve et al [2001] for a comparison of rates of bacterial STIs). The average rate of female herpes simplex virus (HSV-2) from studies in three West African countries (Cameroon, Benin and Nigeria) is 47 percent, whereas the average rate from studies in four East African countries (Kenya, Tanzania, Ethiopia and Zambia) is 50 percent [Glynn et al 2004; Mihret et al 2002; Msuya et al 2003; Ashley-Morrow et al 2003]. Rates for South Africa are also similar, at 53 percent [Auvert et al 2001]. While these data do not cover the full range of countries, it is comforting that all the published data suggest no major intra-continental differences.

Another piece of evidence arguing against intra-continental differences in transmission rates is that the epidemic grew at similar rates after its start date in both East and West Africa. For example, in Cote d'Ivoire the HIV rate at the end of the decade – about 15 years after the HIV rate reached 1 percent – is around 10 percent. The comparable HIV rate in Kenya (a country with similar levels of sexual behavior) 15 years after reaching 1 percent is around 12 percent. This small discrepancy suggests that the epidemic is not growing faster in East Africa, and supports the assumption of similar transmission rates.

The data on sexual behavior used here are a disaggregated version of the DHS data used for Africa overall in Section IV; instead of combining the data I use it for each country separately.[17] As in the analysis in Section IV, the DHS data are used to create a measure of the share of individuals in each age-gender-marital status group who have sex outside of

[17]The countries included are Benin, Burkina Faso, Cameroon, Chad, Cote d'Ivoire, Ethiopia, Guinea, Kenya, Malawi, Mali, Namibia, Niger, Tanzania and Zambia. Ideally, I would use all countries in Sub-Saharan Africa, but I am limited by the availability of data on sexual behavior. However, the data I have cover the major regions of Sub-Saharan Africa that have had very different HIV experiences (the range of HIV rates at the end of the decade is 1.6 percent to over 25 percent).

marriage and the number of partners for these groups. Marriage rates by age and gender are calculated as in the aggregate African analysis, as is information on condom use across countries. As before, condom use is presumed to increase linearly from the first year of condom social marketing, which will now vary across countries. In addition, as before, condom use varies across gender, marital status and partnership type.

When considering data across countries within Africa, issues of underreporting of sexual behavior become more relevant. In particular, if individuals in some countries are systematically more likely to lie about sexual behavior than this could bias the results. Two things are worth noting, however. First, the levels of risky sexual behavior reported here are similar to other studies of sexual behavior in these regions (in particular, the Four Cities Study [Ferry et al 2001] and the CAPS data from Kenya and Tanzania). Given that these other studies focused more closely on sexual behavior and attempted to do a careful job of eliciting the truth about partnerships, this provides some confidence in the DHS reporting. Second, there is a high correlation between the rate of risky sexual behavior reported by the group that is probably the most likely to lie (married women) and the group that is probably the least likely to lie (single men). To the extent that a low correlation would suggest that reporting was not consistent across countries, the high correlation is comforting.

In Section IV, the issue of epidemic start date was relatively unimportant. Within Africa, however, there is clear heterogeneity in the arrival date of the epidemic. By the late 1970s, even before the virus was identified, countries in East Africa were experiencing deaths that are now known to have been AIDS-related. In West Africa, however, it was not until the mid-1980s that countries began to see cases of HIV in the general population. To calibrate the model, it is important to identify the beginning of the epidemic.

Information on the first year of the epidemic is calculated in two ways. First, information from a number of sources, primarily UNAIDS, the CDC and the World Bank was collected and used to suggest starting dates for each country. Second, data on HIV rates across countries were used. Data for each country were searched and the first year in which any study reported a rate of higher than 1 percent for pregnant women was noted. As expected, it becomes clear from these data that the "start date" in most of East Africa was in the late 1970s and early 1980s, whereas it was in the mid-1980s for West Africa.

There are clear endogenaity issues with this method, particularly with using the data on HIV rates to pick a starting date. Behavior, transmission rates and other factors influence when the country reaches 1 percent, as well as the growth rate afterwards, so the date is partially endogenous. There are two responses to this. The first is to note that to the extent that the paper is concerned with exploring epidemic growth, this concern may be less vital.

In addition, there is substantial evidence suggesting that the timing of the epidemic is related to distance from the presumed epidemic origin, in the Democratic Republic of the Congo, which is arguably exogenous.[18] The correlation between start date calculated as described above and distance to the presumed viral origin in an overall sample of African countries is 0.78, suggesting that distance plays a major role in the epidemic arrival date and providing confidence that a significant share of the variation in start date is exogenous.

Before turning to the main results, it is useful to look at some simple graphical comparisons across countries. There are two primary sources of identification in the model: differences in the share of individuals having premarital or extramarital sex, and differences in

[18]The origin of the HIV virus remains somewhat of a mystery. However, there is good evidence that the earliest cases (scattered cases in the 1960s and 1970s) were in the former Zaire [Vangroenweghe 2001; Sharp et al 2001]. These cases were found in cities on both sides of the country, suggesting the origin was somewhere in the middle.

epidemic timing. Figure III illustrates a relationship between sexual behavior and HIV prevalence across countries. This figure shows HIV prevalence graphed against the estimated share of women in the population having sex with a non-spouse (a weighted average of single women having premarital sex and married women having extramarital sex). The corresponding graph for men is similar. Within West Africa and East Africa, there is a positive relationship between HIV rate and sexual behavior – more people engaging in risky sexual behavior implies a higher HIV rate, but the difference between East and West Africa is probably not being driven by differences in sexual behavior.[19] Figure IV illustrates a similar relationship between the "start year" of the epidemic and the HIV rate. Here, there are significant differences between West and East Africa but relatively little difference within region.

Although Figures III and IV illustrate much of the data used in the simulations, they do not provide a full accounting. Table III therefore presents the data on sexual behavior, marriage rates at age 20, epidemic start date, condom use and condom social marketing start date. As in the previous section, all of the data reported are for urban areas only. Sexual behavior data are aggregated across all age groups, so it gives a sense of the information used in the simulation, but is not the full dataset. The information in Table III largely echoes that in Figures III and IV – differences across region appear to be due largely to differences in epidemic start date, while differences within region rely largely on differences in sexual behavior.

[19]It is interesting to note that from this graph sexual behavior appears to be related to the share of the population that is Muslim in each country. Heavily Muslim countries such as Niger, Chad and Mali have much lower rates of risky sexual behavior than, for example, Cameroon. This is consistent with existing evidence on the HIV rate-percent Muslim relationship [Gray 2004].

V.B. Cross Country Results

The primary within-Africa simulation results are presented in Figure V, which graphs the HIV rate predicted by the model against the actual HIV rate in the 14 countries included in the analysis. I consider the fit of the model in three time periods – "early" (1990-1992), "mid" (1994-1996) and "late" (1998-2001).[20] Both the predicted and actual HIV rates in this figure reflect the rates for women of childbearing age, because nearly all of the data on HIV rates in Africa come from antenatal clinic testing. I restrict to country-periods in which at least 1000 individuals are tested and exclude time periods in which all the testing is done in the capital city.

Figure V demonstrates that the model is generally a good fit to the actual data. In particular, considering the last period (in which the data on HIV rates are likely to be the most accurate), the model has an average prediction error of 3.6 percentage points.

Although using the data on pregnant women has significant advantages, particularly since it is clear what the providence of the data is, there are obvious drawbacks. The population is somewhat limited, and I am relying on a single source for HIV rates (although that source is a compilation of many). It is worth considering a robustness check in which data for a more general population are matched to overall estimates of HIV rates. UNAIDS Factsheets [UNAIDS et al. 2002; UNAIDS et al. 2004] provide estimates of the overall adult HIV rate in Sub-Saharan African countries. These data have significant issues: There is no consistent information on how they are put together, so it is difficult to discern to what

[20]The HIV rates used are a weighted average of the HIV rates from the capital city and the HIV rates from other cities, with the weighting determined by the share of individual in the DHS that are from those respective areas. In addition, the predicted values for the time periods are weighted by the share of studies from each year within each time period. So, for example, if 75 percent of the studies for Namibia in the late period are done in 1998 then the predicted value for 1998 gets a 75 percent weight in the matching.

degree the estimates rely on studies of prevalence versus impressions from health ministries in the countries. In addition, it is clear that in many years no data are available, so the UNAIDS estimates are based on conjectures about increases in HIV rates. I match the rates estimated by UNAIDS to the model's prediction for all adults, both urban and rural.

Figure VI presents these results. The model is still a good fit to the data; although there are larger errors, particularly for Kenya and Benin, the general pattern still holds. Even including the outliers, the average error is only 5 percentage points.

As a final robustness check, it is worth noting that the model qualitatively fits the relationship between HIV rate and age by gender. In Kenya and Zambia, for example, the highest HIV rates are estimated for women in their early 30s, with the rates for men slightly lower and the peak for them in the late 30s (results available from the author). This is consistent with the evidence on HIV by age and gender in Kenya and Zambia [Central Bureau of Statistics et al. 2004; World Health Organization 2003]

V.C. Robustness

The model calibrated above assumes constant sexual behavior over the course of the epidemic (with the exception of increases in condom use). This may be problematic if sexual behavior is changing in response to HIV rates. Indeed, in the United States there has been significant behavior change (both positive and negative) among high-risk groups (particularly gay men) in response to the epidemic [McKusick, Hortsman and Coates 1985; Ekstrand et al, 1999]. It is worth considering whether this is a possibility in the case of Africa.[21]

[21]Knowledge of one's HIV status appears to be quite low through most of Africa. In six countries, DHS surveys asked individuals whether they had ever been tested for HIV; only 9 percent of those sampled said yes. This suggests that behavior change in response to one's own HIV status is unlikely to be significant.

Simultaneity bias makes it difficult to identify sexual behavior response to the epidemic – since higher levels of promiscuity lead to more HIV, even if more HIV leads to lower levels of promiscuity, the causal relationship may be elusive. One partial solution to this problem is to look over time in a single location and see if any changes in behavior can be observed. Several studies that have attempted to do this and the results generally suggest little, if any, behavior change [Mwaluko et al. 2003; Williams et al. 2003; Bloom et al. 2000].

Rather than relying solely other analyses, I consider data from ten countries in Africa for which the DHS surveys have been run more than once. Table IV shows data on the share of single women who are having premarital sex over the 1990s in these ten countries.[22] The data in this table generally support the hypothesis of little or no behavior change over time. Although in some countries behavior seems to have decreased over the period in question, in other cases it clearly increased. In fact, in the 15 changes listed in Table IV, seven represent increases, seven represent decreases and one represents no change.

Combining the data in Table IV with information on HIV rates suggests that, on average, there has been a very small decrease in sexual behavior with increases in HIV rate. More specifically, the data indicate that a 1 percent increase in HIV rate is associated with a 0.2 percent decrease in the share of single women having premarital sex. Inserting a mechanical decrease in sexual behavior of this magnitude into the model does not change the simulations very much (results available from the author), suggesting that endogenous behavior change does not play a major role in the current epidemic in Africa.

A second issue that is worth mentioning is the robustness of the estimates of HIV

[22]Single women are the only group for which these data are available over this period length, but given the high correlation across behavior of different groups within a country this is unlikely to lead to significant bias.

rates. Recently, several countries have done larger-scale population-based testing and revised their estimates of country-wide HIV rates (generally downward). The most extreme example is Kenya, where the HIV rate in the UNAIDS 2002 Factsheet is 15 percent, whereas a population-based survey estimated it the next year at 6.7 percent (this largely explains the fact that Kenya is an outlier in Figure VI). If more countries update their estimates and revise them downward in this fashion, it would call the realism of the model into question.

Although it is difficult to come to a conclusion on this issue without further HIV testing in more countries, a few things are worth noting. The first is that the central result of this paper – that the difference between the Untied States and Sub-Saharan Africa is largely about differences in transmission rates – is not likely to be affected by these updated estimates. Further, the new population estimates have their own issues and it is possible that they under-estimate the true HIV rate. For example, in Kenya 62 percent of respondents in urban areas were tested, versus 76 percent in rural areas [Central Bureau of Statistics et al. 2004], which could lead to downward bias (for a summary of why antenatal clinic and population prevalence estimates might differ, see World Health Organization and UNAIDS [2003]). Further testing may shed more light on the true HIV rates across Africa and, by extension, on the power of this particular model.

VI. Policy Implications

The evidence in the preceding three sections points toward two types of interventions to prevent further epidemic spread – interventions to decrease epidemic transmission rates, and interventions to decrease the frequency of risky sexual behavior. In this section, I

simulate the future of the epidemic in Africa under these intervention types, discuss specific intervention models and evaluate cost-effectiveness.

The simplest way to see the impact of these interventions is to assume that there is a discrete change in either transmission rates or sexual behavior at one point in time, and to compare the path of the epidemic with an intervention to the path without intervention. This analysis can be seen in Figure VII, which presents the simulated path of the epidemic in Africa over the next ten years under no intervention, a 20 percent decrease in transmission rates, and a 20 percent decrease in all aspects of sexual behavior (share having non-marital sex and the number of partners). As will be discussed in more detail below, these represent roughly equivalent monetary expenditures. Both interventions slow the course of the epidemic. The intervention on transmission rates, however, is substantially more effective. This intuition carries over to the cost-effectiveness results below.

Moving to more specific interventions, I consider two intervention models. The first, designed to decrease transmission rates, involves treating other untreated (bacterial) sexually transmitted infections. This intervention is modelled on the Mwanza, Tanzania intervention presented in Grosskurth et al. [1995]. That study finds that the intervention decreased transmission rates by 25 percent male-to-female and 36 percent female-to-male, at a yearly cost of $59,060 (in 1993 $US) for 150,000 individuals [Grosskurth et al 1995; Gilson et al 1997].[23] Aggregating up to the population of the 14 countries considered in this analysis, this suggests an overall ten-year cost of approximately $1.07 billion in year 2000 dollars.

The second type of intervention is designed to decrease risky sexual behavior. In this

[23]It is worth noting that this obviously includes some non-compliance to the treatment program. I am implicitly assuming that non-compliance to treatment would be the same in Africa overall as it was in this one study.

case, I consider a scaled-up version of the Ugandan experience. Through advertising and educational campaigns, Uganda appears to have decreased most aspects of risky sexual behavior and estimates suggest that the HIV prevalence there has gone down substantially (details of the intervention can be found in Hogle [2002]). Ugandan WHO/GPA and DHS surveys in 1989 and 1995 allow me to calculate the decrease in percent of women having premarital sex (35 percent to 22 percent), women having extramarital sex (6 percent to 3 percent) and men having extramarital sex (23 percent to 16 percent). Unfortunately, data are not available for single men, so their behavior change is assumed to be the same as for single women. Using data from 1995 and 2000, it is also possible to calculate decreases in the number of partners for those having casual partners (either premarital or extramarital). I find an estimated decrease of 10 percent for single men, 13.5 percent for single women, 10 percent for married women, and no apparent decrease for married men. This reduction in sexual behavior was achieved at a cost of $180 million over 10 years [Hogle 2002]. Aggregating up to the population of these countries, this suggests an overall ten year cost of approximately $2.8 billion, in year 2000 dollars.

I have simulated these interventions in the model for Africa overall, assuming that both interventions achieve their entire effect in the first year (this seems reasonable for the transmission rate intervention; perhaps less so for the educational case). Allowing for slower effectiveness will not change the results substantially. The model is simulated under the case of no intervention and for each intervention separately. Using information on current population at each age, measures of effectiveness are calculated from the model output. These include life years saved, disability-adjusted life years (DALYS)[24] saved and overall infections

[24]A DALY is a life year adjusted for changes in quality of life. In general, if individual quality of life is half

averted.

Table V reports results on effectiveness and cost-effectiveness. The intervention affecting transmission rates would save 291 million life years with 13 million infections averted (around 25 percent of the total infections over the next decade). This could be achieved at a cost of $3.67 per life year, and around $78 per infection. The sexual behavior intervention is slightly less effective, preventing 6 million infections at a cost of $16.82 per life year, and $436 per infection.

It is also worth noting that, in addition to greater cost effectiveness, an intervention designed to treat other STIs is likely to be more generalizable than one to change sexual behavior. It seems reasonable to infer the effect of treating STIs in one country based on the experience of another. However, changing sexual behavior may be more culturally specific, and therefore the experience of one country may not be transferable onto others. This predictability is another argument for the STI treatment intervention.

These results suggest that substantial strides could be made towards curbing the HIV epidemic by simply treating other STIs, using inexpensive, off-patent drugs. It is possible that an even more effective intervention (although somewhat higher cost) would be to treat bacterial STIs and to provide herpes suppressive drugs that would limit the number of herpes outbreaks. Since herpes is one of the major causes of genital ulcers, this type of treatment would have the potential to be even more effective. The Ugandan-style educational intervention appears to be somewhat more expensive, but the model still suggests it is quite inexpensive relative to, for example, treatment with antiretrovirals.

as good, this represents a loss of 0.5 DALYs. Following Gilson et al. [1997], here I assume that individuals lose 0.1 "life years" per year during the first nine years of infection, and 0.9 during the final year before death.

VII. Conclusion

The HIV/AIDS pandemic is a global problem. This paper is motivated by the desire to understand why Africa has been disproportionately affected and why infection rates vary so much within that continent. This exercise allows us not only to further understand the future of the epidemic, but may also help in the design of policy interventions.

The paper makes a case for the importance of transmission rates; indeed, I argue that the difference between Sub-Saharan Africa and the United States lies primarily in the difference in viral transmission rates. The model suggests that with the United States level of transmission, HIV prevalence in Africa would be around 0.3 percent. This indicates that interventions designed to affect these rates may be the best investment. In particular, I argue that treatment of other STIs – particularly those that cause genital ulcers – is an inexpensive and potentially effective intervention.

One remaining issue is feasibility. There is a limited amount of money available to address the epidemic in Africa, and thus far the paper has not presented any evidence that this funding would be sufficient for the type of intervention suggested. The model in this paper indicates that expanding the STI treatment program to cover the entire continent would cost on the order of $300 million per year. If this had been done in 2002, the results suggest that 25 percent of new infections, or about 835,000 infections, could have been prevented. Total expenditure on HIV/AIDS interventions in low and middle income countries in 2002 was around $3.7 billion, half of which went to Sub-Saharan Africa[UNAIDS 2003]. This implies that for around 15 percent of the 2002 expenditures, fully a quarter of new infections in Africa might have been prevented.

To this point, the argument for interventions to treat STIs has focused on their cost-effectiveness. It is worth noting, however, that this is one of the few interventions that individuals may want to participate in. For example, it may be difficult to convince people to have fewer sexual partners or use condoms even with dire warnings about the consequences. However, it may be less difficult to convince people to have other (uncomfortable) illnesses treated. By providing people with something they want it may be easier to affect this change. It is also worth noting that bacterial STI treatment is relatively short-term, which makes it relatively easy to administer, even in a place with limited medical care available.

The HIV/AIDS epidemic threatens to cripple Africa. Although there is conflicting evidence on whether the epidemic will promote economic growth or not, there can be little doubt that it is a humanitarian disaster [Bloom and Mahal 1995; Young 2005; Wendell and Werker 2004]. This paper suggests that the epidemic can be understood in a relatively straightforward way as largely the result of sexual behavior and transmission rate. Inexpensive interventions could decrease the incidence of the disease over the coming decade, particularly if the funds are focused on treatment of other sexually transmitted infections.

Appendix A

This appendix provides details of the equations used in the simulations. I discuss the incidence and prevalence calculations for single women, married women, single men and married men. I focus on a single age group, with the understanding that the equations are parallel for all other age groups. The only difference in the estimation across age groups is that the share of individuals who are married increases with age; actual data on marriage rates by single year of age are used so as the population ages the HIV rate among married individuals is more heavily weighted than the HIV rate among single individuals when calculating overall rate for individuals of a particular age.

Denote the base transmission rate (without condom protection) from men to women as β_w and from women to men as β_m. Further, denote the transmission rate from female sex workers to men as $\tilde{\beta}_m$ and from men to female sex workers as $\tilde{\beta}_w$.

Condom use varies with gender, marital status and partnership type, as well as over time, and there is no condom use in spousal partnerships. Denote condom use among single women in casual partnerships in period t as $c_{t,sw}$ and, among married women, $c_{t,mw}$. Further, denote condom use among single and married men in casual partnerships in period t as $c_{t,sm}$ and $c_{t,mm}$. Condom use for single and married men with sex workers is denoted $\tilde{c}_{t,sm}$ and $\tilde{c}_{t,mm}$.

I turn now to sexual behavior, beginning with single women. Denote the number of partners for those single women having casual partners as n_{k1}. Denote the overall infection rate for men in the general population in period t is m_t. Incidence for the two groups of single women are:

No partners	$I_1 = 0$
Casual Partners	$I_2 = (1 - (1 - \beta_w(1 - c_{t,sw})m_{t-1})^{n_{k1}})$

In all equations below, define $share_i$ as the share of the population that is in group i, where the incidence for group i is I_i as defined above.

Denote the prevalence for single women in period t as sw_t. Those infected ten years ago die this period; death in period t is therefore the incidence ten years ago (I_{t-10}), multiplied by the number of uninfected people ten years ago and adjusted for changes in population size. Denote the population in period t as p_t and the number infected in period t as n_t. The overall prevalence for single women in period t is therefore:

$$sw_t = \frac{sw_{t-1} + (1 - sw_{t-1}) \sum_{i=1}^{2} (share_i)(I_i) - I_{t-10} \left(\frac{p_{t-11} - n_{t-11}}{p_{t-1}} \right)}{1 - I_{t-10} \left(\frac{p_{t-11} - n_{t-11}}{p_{t-1}} \right)}$$

The equations for married women are nearly identical, with the addition of a spouse. The number of casual partners for married women who have them is denoted n_{k2}. Denote the infection rate for a spouse in period t is denoted S_t. The incidences for the groups of married women are:

Spouse only	$I_1 = (\beta_w S_{t-1})$
Spouse and Casual	$I_2 = (1 - (1 - \beta_w(1 - c_{t,mw})m_{t-1})^{n_{k2}}(1 - \beta_w S_{t-1}))$

Prevalence for married women is calculated the same way as for single women.

Turning to the equations for men: Denote the infection rate for women overall in period t as w_t (casual partners are drawn from this overall population). Sex workers are drawn from the separate sex worker population (discussed in more detail below) and have an infection rate of fsw_t in period t. The number of casual partners for those having casual partners is $n_{k3,1}$. The number of sex worker partners for those having sex worker partners is $n_{k3,2}$. Incidence for each group can be expressed as below:

None	$I_1 = 0$
Casual	$I_2 = (1 - (1 - \beta_m(1 - c_{t,sm})w_{t-1})^{n_{k3,1}})$
FSW	$I_3 = (1 - (1 - \tilde{\beta}_m(1 - \tilde{c}_{t,sm})(fsw_{t-1}))^{n_{k3,2}})$
Both	$I_4 = (1 - ((1 - \tilde{\beta}_m(1 - \tilde{c}_{t,sm})(fsw_{t-1}))^{n_{k3,2}})((1 - \beta_m(1 - c_{t,sm})w_{t-1})^{n_{k3,1}}))$

Prevalence is calculated as for single women.

Finally, turning to married men, denote the number of casual partners for those having them as $n_{k4,1}$ and the number of sex worker partners as $n_{k4,2}$. Incidence is expressed below:

Spouse	$I_1 = (\beta_m S_t)$
Sp., Cas.	$I_2 = (1 - ((1 - \beta_m(1 - c_{t,mm})w_{t-1})^{n_{k4,1}})(1 - \beta_m S_t))$
Sp., FSW	$I_3 = (1 - ((1 - \tilde{\beta}_m(1 - \tilde{c}_{t,mm})(fsw_{t-1}))^{n_{k4,2}})(1 - \beta_m S_t))$
All	$I_4 = (1 - ((1 - \tilde{\beta}_m(1 - \tilde{c}_{t,mm})(fsw_{t-1}))^{n_{k4,2}})((1 - \beta_m(1 - c_{t,mm})w_{t-1})^{n_{k4,1}})(1 - \beta_m S_t))$

Again, the equation for prevalence is parallel to that for single women.

The final element of the model is female sex workers (FSWs). As mentioned, I assume some heterogeneity across FSWs – some work in bars and have relatively few partners; others work in brothels and have many. Denote the rate among the non-brothel FSWs in period t as lf_t and the rate for FSWs in brothels as hf_t. The number of partners for each group is $n_{l,f}$ and $n_{h,f}$, respectively. The HIV rate of partners of FSWs are a weighted average of the HIV rates among men of different groups, with the weighting determined by the share of each group who visit FSWs. Denote this weighted HIV rate as $\hat{m}_{t-1}$; condom use will also be a weighted average of the condom use among visiting men, denoted Denote condom use as $c_{t,fsw}$. The incidence in each group is determined:

Non-Brothel	$I_1 = (1 - (1 - \tilde{\beta}_w(1 - c_{t,fsw})\hat{m}_{t-1})^{n_{l,f}})$
Brothel	$I_2 = (1 - (1 - \tilde{\beta}_w(1 - c_{t,fsw})\hat{m}_{t-1})^{n_{h,f}})$

The prevalence is calculated in the same way as for non-FSWs.

Appendix B

In general in the model, women can choose to partner with casual partners from the general population and with their spouse, if they are married. Men may choose to partner with a spouse if married, casual partners and female sex workers. The inputs to the model are the

share of individuals choosing each set of partnerships and the average number of sexual partners for those in each non-marital sex grouping. The survey data, however, are limited and additional assumptions must be made to identify the parameters in the model. This appendix details how those parameters are identified. Here, I do the analysis for single individuals only. The analysis for married individuals is identical, but they must also partner with a spouse with certainty.

For women, the model requires us to identify the parameters in the following table. Obviously the values will vary by country.

Group	**Share in Group**	**Average Non-Marital Partners for Group**
None	$1-x$	0
Casual Partners	x	y

This is not an issue. Both x and y are observable – x is simply the share of women reporting sexual partners, and y is the average partners they report.

The analysis is somewhat more complex for men. The parameters that need to be identified appear in the table below.

Group	**Share in Group**	**Average Non-Marital Partners for Group**
None	$1-a-b-c$	0
Casual Partners	a	d
FSWs	b	e
Casual and FSWs	c	$d+e$

I make the assumption that people who partner with both casual partners and female sex workers partner with the same number of each as those who partner with only one group. What is observed in the data is $t = a + b + c$, the share of men reporting any casual sex. In addition, I observe the average number of casual partners for these men: $p = \frac{ad+be+c(d+e)}{a+b+c}$.

Following Carael et al [1995], Voeten et al [2002] and DHS survey data I estimate the share of men in each group. Consistent with these studies and data, I assume that half the individuals who report non-marital sex are having sex only with casual partners, another 40 percent are having sex with casual partners and FSWs and the remaining 10 percent have sex with only FSWs.

In order to close the model, it must be the case that the number of male casual partners taken on by the women is the same as the number of female casual partners taken on by the men. Assuming equal sample sizes, this implies that $xy = ad + cd$. Solving for d and e yields:

$$d = \frac{xy}{a+c} = \frac{xy}{.5t}$$
$$e = \frac{pt - xy}{.5t}$$

The calculations above apply for all countries in Africa. For the United States I modify the parameters somewhat to reflect the lower use of FSWs. In particular, I assume that 70 percent of those with any non-marital partners are having sex with women from the

overall population, 20 percent are having sex with women from the overall population and FSWs and 10 percent are having sex only with FSWs.

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TABLE I
HIV Transmission Rates

Panel 1: Developed World

Citation	# of Subjects	M-to-F	F-to-M	Subpopulation	Location
Di Vincenzi et al. [1994]	121	10.8%	8.5%	Transfusion Recipients	Europe
Ragni et al.[1989]	45	13.3%	N/A	Hemophiliacs	United States
van der Ende et al.[1988]	13	0.0%	N/A	Hemophiliacs	Europe
Rockstroh et al.[1995]	198	10.1%	NA	Hemophiliacs	United States
Saracco et al. [1993]	113	11.5%	N/A	Hemophiliacs	Europe
Peterman et al. [1988]	80	18.2%	8.0%	Mixed	United States
Padian et al. [1991]	186	13.2%	1.4%	Transfusion Recipients	United States
Laurian et al. [1989]	31	9.7%	N/A	Hemophiliacs	Europe
Allain [1986]	148	6.8%	N/A	Hemophiliacs	Europe

Panel 2: Sub-Saharan Africa

Citation	# of Subjects	M-to-F	F-to-M	Subpopulation	Location
Gray et al [2001]	174	17.50%	27.30%	General Population	Uganda
CAPS Data[1]	750	27.50%	7.10%	General Population	Kenya and Tanzania
Grosskurth et al [1995]	523	29.30%	14.50%	General Population	Tanzania
Quinn et al [2000]	415	26.70%	17.50%	General Population	Uganda

Notes: For the United States, transmission rates in each study are as reported for heterosexual, monogamous couples. "Mixed" may include intravenous drug users. For Sub-Saharan Africa, transmission rates are calculated from reported data on HIV incidence, sexual behavior of the population, condom usage and HIV prevalence in the study area. Details of this calculation are in Section IV.A. All transmission rates are per unprotected sexual partnership with an infected individual of the opposite sex.

[1] The CAPS study is an individual-level survey run in Nairobi, Kenya and Dar-es-Salaam, Tanzania. Individuals were tested for HIV at the beginning of the study and then remained in the study population for a year. Follow-up surveys did additional HIV testing and asked individuals about their sexual behavior over the study period.

TABLE II
Sexual Behavior: United States and Sub-Saharan Africa

	United States	Sub-Saharan Africa
Share with Casual Partners		
Single Women	72.7%	27.6%
Married Women	2.4%	8.9%
Single Men	81.8%	53.6%
Married Men	6.2%	21.1%
Number of Casual Partners		
Single Women	1.49	1.18
Married Women	1.52	1.24
Single Men	2.33	1.80
Married Men	2.13	1.57
Marital Status		
Percent Women Married at 20	19.60%	48.10%
Percent Men Married at 20	4.80%	5.20%
Percent Women Married at 40	55.50%	97.50%
Percent Men Married at 40	64.70%	95.20%
Condom Use (late 1990s)	63%	44.5%
HIV Rate (1998-2000)	0.15%	11.9%

Notes: Data on sexual behavior, marital status and condom use for the United States are from the annual General Social Survey for 1988-1994 for all individuals ages 18-59. Data for Sub-Saharan Africa are drawn from the Demographic and Health surveys in Benin(1996), Burkina Faso(1998), Cameroon(1998), Chad(1996), Cote d'Ivoire(1998), Ethiopia(2000), Guinea(1999), Kenya(1998), Malawi(2000), Mali(2001), Namibia(2000), Niger(1998), Tanzania(1996) and Zambia(1996), and cover individuals ages 15-59. The data for Africa overall are a population-weighted average of the data for each country. Information on HIV rates are from the CDC for the United States, and from the U.S. Census HIV/AIDS Surveillance Database for Sub-Saharan Africa. For Africa, the HIV rate is a population-weighted average of the HIV rates in the countries used. All data for Africa are limited to urban areas.

TABLE III
African Country Data Used in Simulations

Country (DHS Year)	**Share with Casual Partners**[a]				**Number of Partners**[a]				**Condom Use**[a]		**Condom Start Date**	**% Married at 20**[a]		**Start Date**[b]
	SW	MW	SM	MM	SW	MW	SM	MM	Men	Women		Men	Women	
Benin (1996)	41.1%	7.7%	69.1%	40.1%	1.13	1.34	2.09	1.78	34.8%	37.6%	1989	19%	68%	1985
Burkina Faso (1998)	33.8%	7.9%	47.8%	33.3%	1.14	1.08	1.76	1.85	78.0%	55.0%	1991	8%	80%	1985
Cameroon (1998)	57.5%	23.1%	64.2%	57.8%	1.45	1.47	2.25	1.94	46.4%	28.1%	1989	12%	61%	1985
Chad (1996)	21.3%	6.5%	44.6%	30.3%	1.14	1.23	2.15	1.85	55.5%	31.5%	1992	32%	89%	1986
Cote d'Ivoire (1998)	55.1%	20.5%	68.1%	48.8%	1.22	1.24	2.00	1.72	70.0%	36.3%	1990	9%	47%	1985
Ethiopia (2000)	10.3%	4.1%	29.4%	7.7%	1.13	1.32	1.63	1.44	62.7%	61.8%	1990	10%	63%	1980
Guinea (1999)	29.9%	11.9%	64.9%	42.8%	1.17	1.23	1.83	1.55	47.3%	24.8%	1991	13%	83%	1987
Kenya (1998)	35.5%	19.3%	57.1%	29.3%	1.25	1.21	1.77	1.67	48.9%	48.0%	1989	9%	50%	1979
Malawi (2000)	32.8%	4.2%	42.8%	16.8%	1.03	1.25	1.55	1.56	49.0%	48.0%	1994	19%	78%	1980
Mali (2001)	36.3%	4.6%	48.7%	17.3%	1.09	1.10	1.68	1.54	43.6%	25.8%	1992	27%	84%	1985
Namibia (2000)	47.4%	21.2%	60.5%	26.6%	1.05	1.05	2.36	2.21	75.2%	58.5%	1992	17%	28%	1982
Niger (1998)	7.2%	5.5%	28.8%	18.5%	1.07	1.41	2.02	2.02	70.0%	17.5%	1994	25%	87%	1987
Tanzania (1996)	29.5%	2.8%	32.6%	19.2%	1.29	1.24	1.78	1.61	54.7%	33.3%	1993	12%	64%	1981
Zambia (1996)	29.9%	16.3%	42.2%	28.0%	1.25	1.30	2.00	1.54	48.0%	46.2%	1992	9%	67%	1979

Notes: SW – single women; MW – married women; SM – single men; MM – married men. All Data are for urban individuals only in the DHS surveys. Years reported in parenthesis after country names are the DHS survey years. There are an average of 145 people in each age-gender cell.

[a] Sources: Data from Demographic and Health Surveys

[b] Sources: Start date imputed from U.S. Census Surveillance Database, UNAIDS and World Bank publications.

TABLE IV
Sexual Behavior Change Over Time

Country	Year	% Women Having Premarital Sex
Benin	1996	38.0%
Benin	2001	44.8%
Burkina Faso	1992	20.3%
Burkina Faso	1998	27.5%
Cameroon	1991	51.0%
Cameroon	1998	55.4%
Ivory Coast	1994	57.6%
Ivory Coast	1998	56.1%
Kenya	1988	32.4%
Kenya	1993	37.5%
Kenya	1998	31.6%
Kenya	2003	24.9%
Malawi	1996	30.5%
Malawi	2001	29.0%
Namibia	1992	46.2%
Namibia	2000	52.6%
Nigeria	1990	38.2%
Nigeria	1999	31.2%
Nigeria	2003	31.0%
Tanzania	1992	32.5%
Tanzania	1994	33.6%
Tanzania	1996	25.7%
Tanzania	1999	35.0%
Zambia	1992	41.0%
Zambia	1996	38.7%
Zambia	2001	34.9%

Notes: Data on sexual behavior come from DHS surveys run between 1988 and 2003. Share of women having premarital sex is equal to the share of never-married women reporting having had sex.

TABLE V
Cost-Effectiveness of Interventions

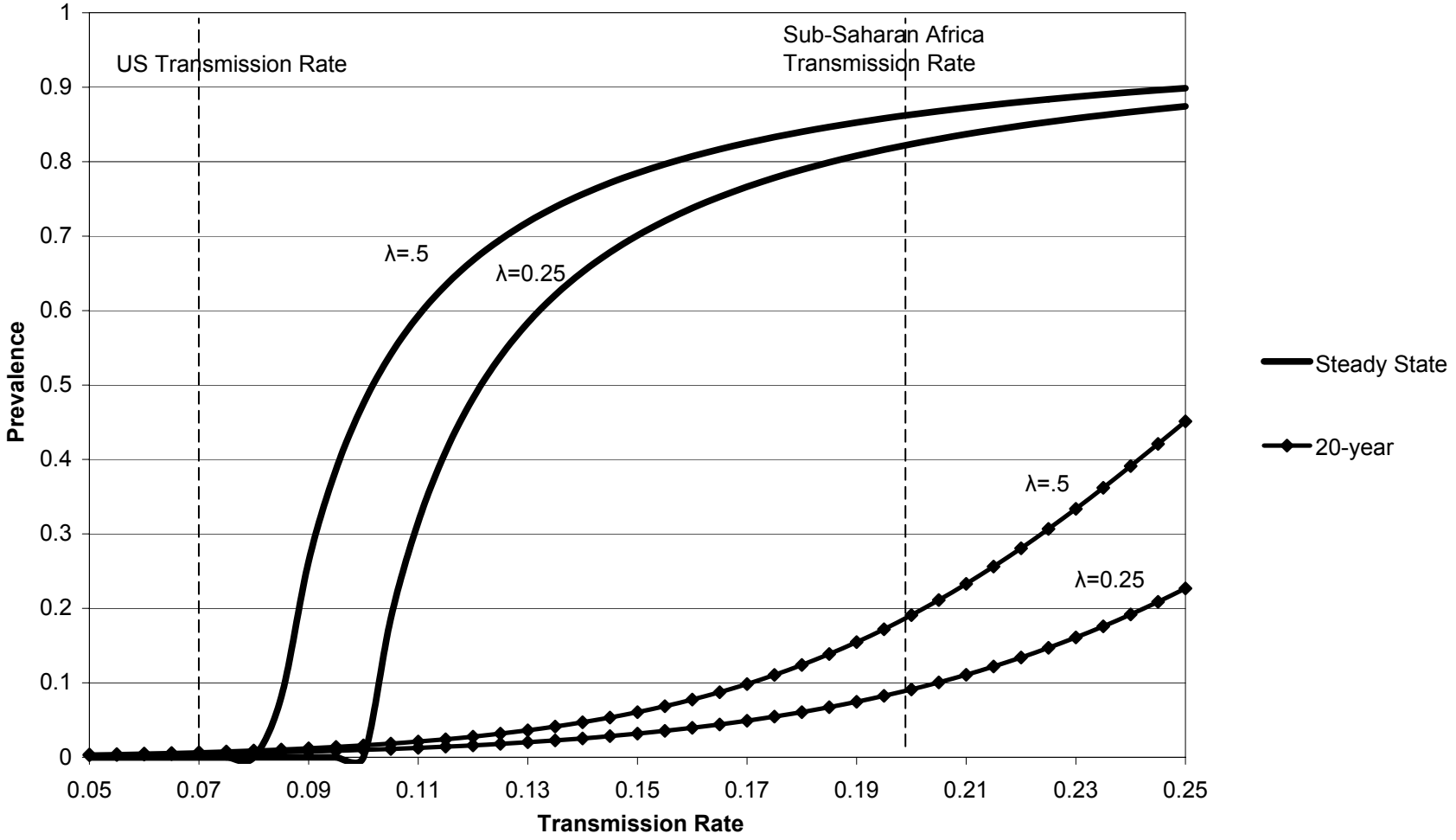
	Change Transmission Rates Through STI Treatment[a]	Change Behavior Through Education[b]
Cost	$1,068,326,724	$2,800,263,630
Life Years	291,338,142	166,476,290
DALYs	311,746,543	176,081,992
Infections Averted	13,655,027	6,409,195
Cost per LY	$3.67	$16.82
Cost per DALY	$3.43	$15.90
Cost per Infection	$78.24	$436.91

Notes: All costs include both delivery costs and drug costs

[a] treatment of bacterial STIs, including syphilis, gonorrhea, chancroid and others.

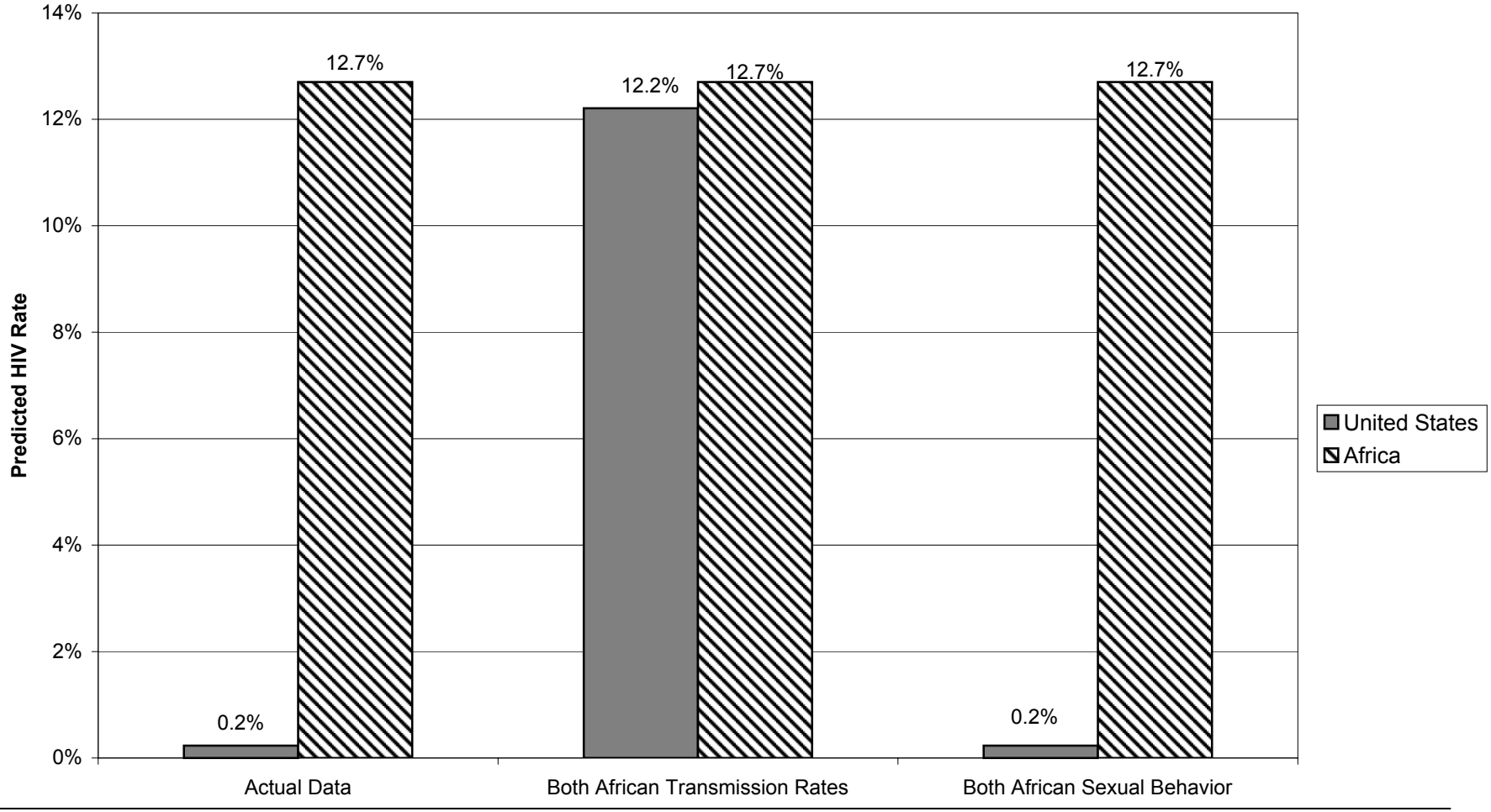
[b] intervention to decrease number of sexual partners; assuming intervention works on the population gradually

Figure I
HIV Prevalence in Simplified Model



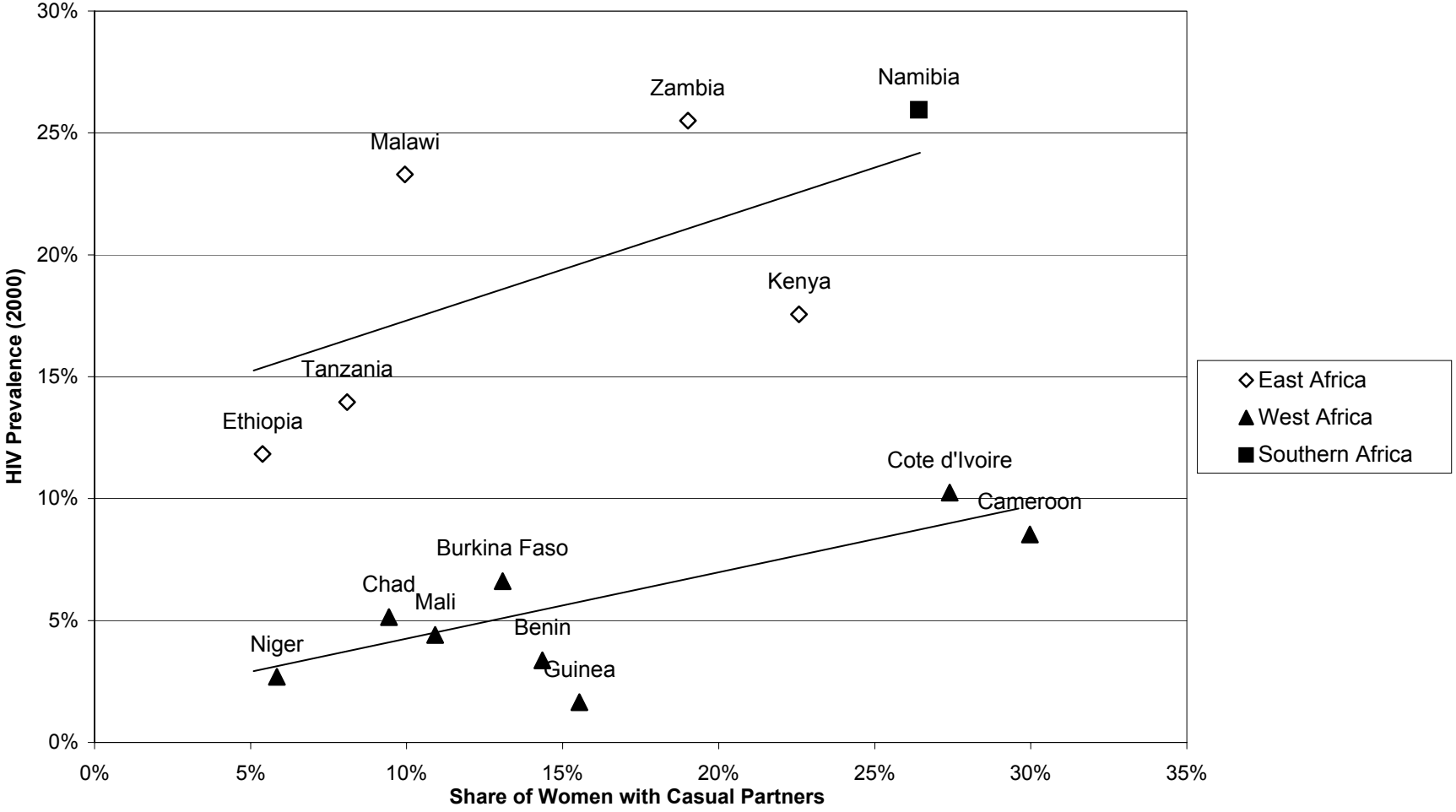
Notes: This graph shows the relationship between transmission rate, sexual behavior and steady state HIV rate in the simplified model in Section 3. λ represents the share of individuals having two partners; the rest have only one partner. Transmission rate is per unprotected sexual partnership with length equal to one period. Lower lines are the 20-year infection rate; the upper lines are the steady state of the model.

Figure II
Predicted HIV Rates for the United States and Sub-Saharan Africa



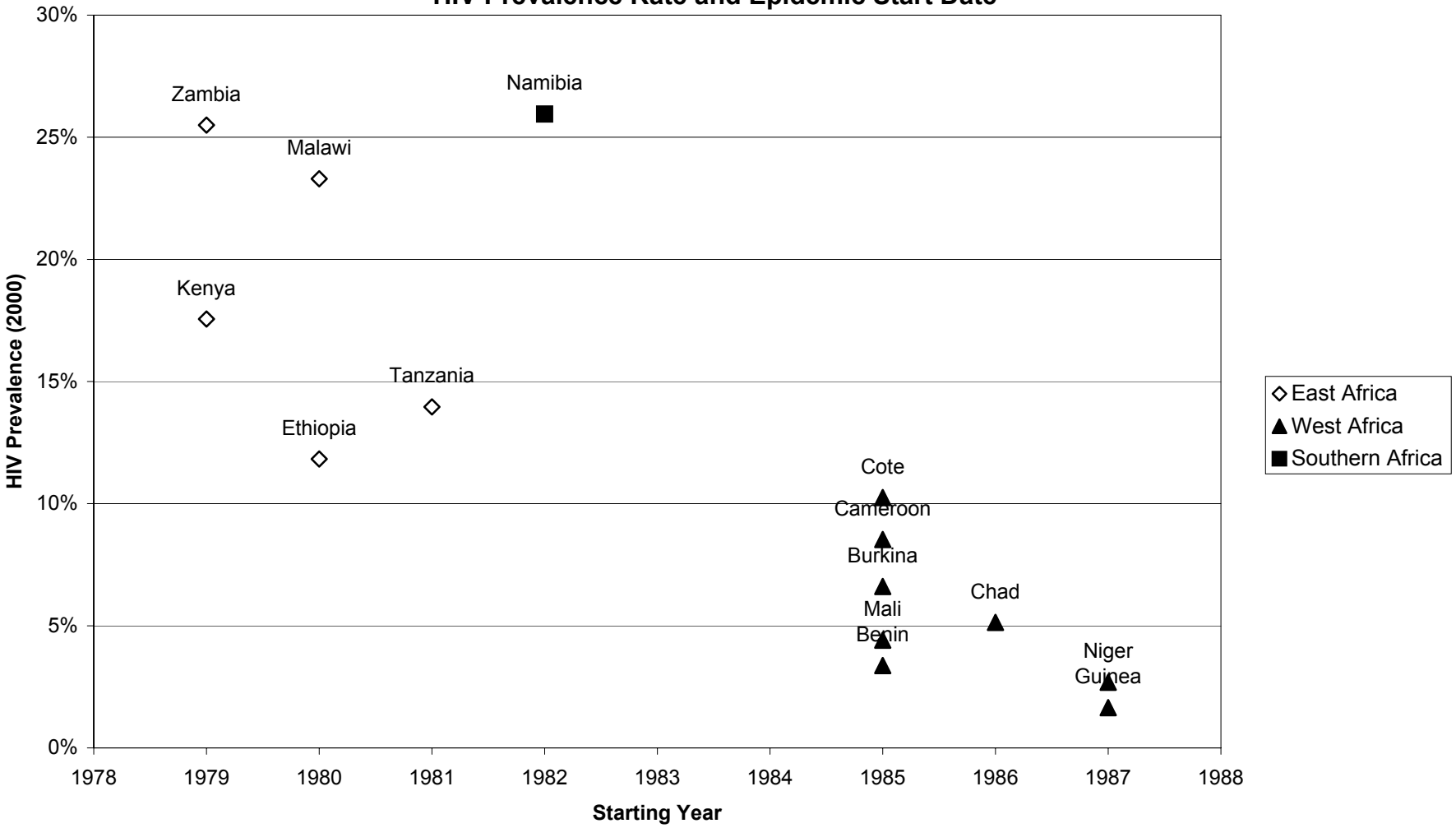
Notes: Figure reports HIV rates predicted by the simulation model for the United States and Sub-Saharan Africa. HIV rate is an average for 1998-2000. First columns: predicted rates using own sexual behavior and transmission rates; second columns: predicted rates using own sexual behavior, and Sub-Ssaharan Africa transmission rates; third columns: predicted rates using own transmission rates, Sub-Saharan Africa sexual behavior.

Figure III
HIV Prevalence and Female Sexual Behavior



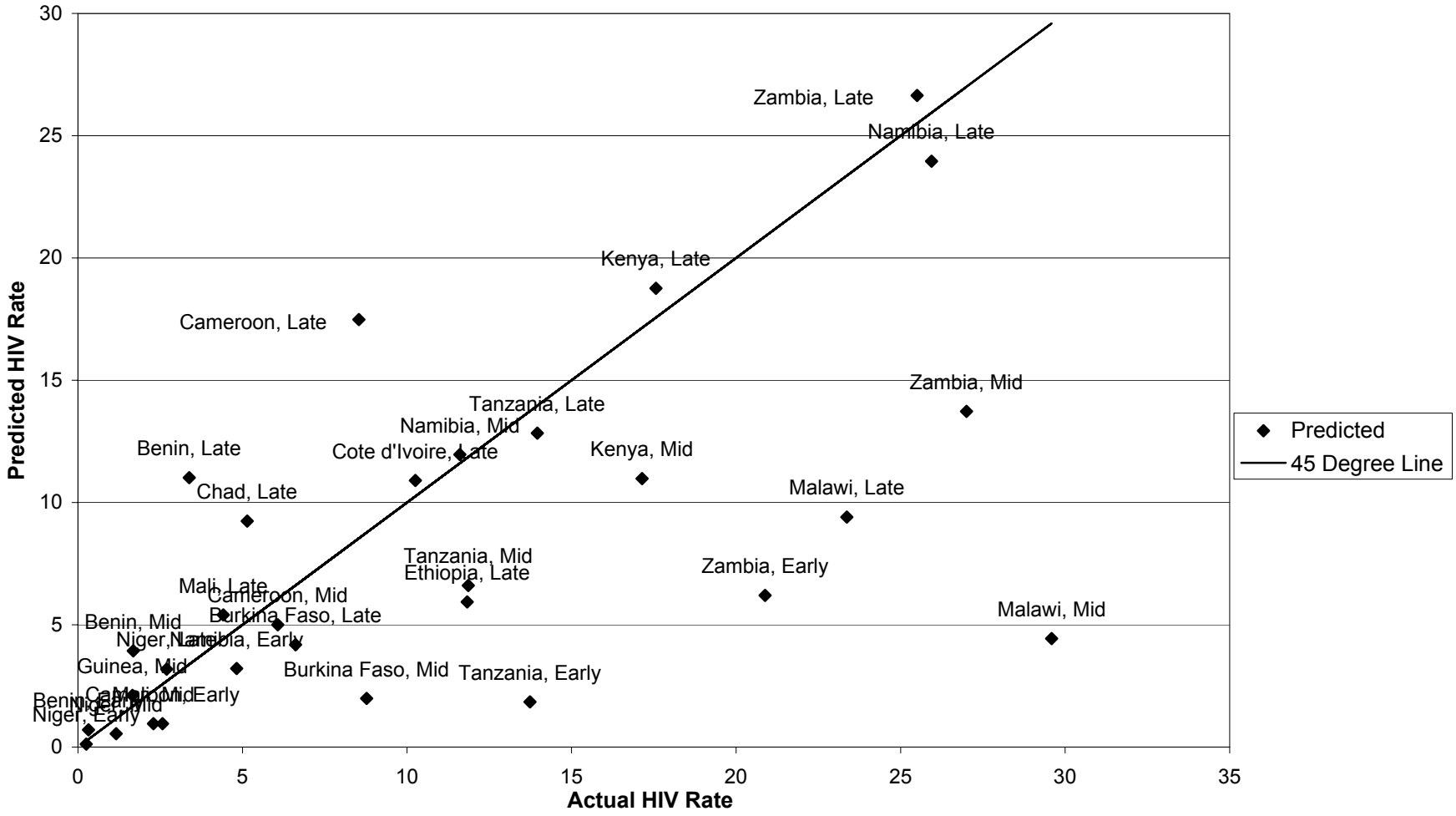
Notes: HIV prevalence calculated from the U.S. Census Surveillance Database as an average for urban-dwelling pregnant women in the country between 1998 and 2000. Share of women with a casual partner is a weighted average of the share of single women with a casual partner (weight: 20%) and married women with a casual partner (weight: 80%). Best fit lines for East Africa and West Africa are included. Data from Demographic and Heath Surveys, and is limited to urban individuals.

Figure IV
HIV Prevalence Rate and Epidemic Start Date



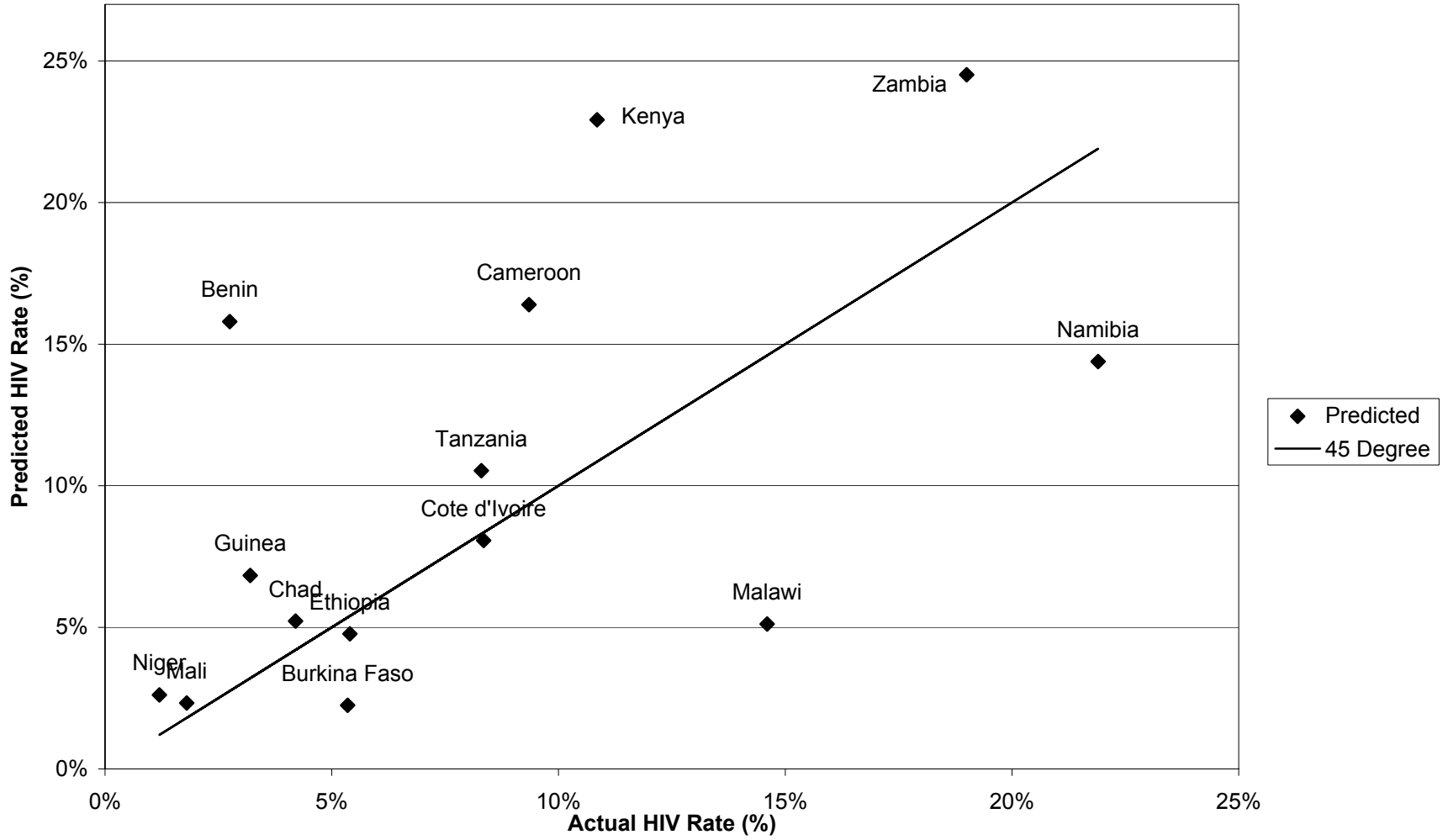
Notes: HIV prevalence calculated from the U.S. Census Surveillance Database as an average for urban-dwelling pregnant women in the country between 1998 and 2000. Starting year is the first year that any study in the U.S. Census Surveillance Database reported a rate of 1% or more for pregnant women or, if the epidemic began earlier than testing, the starting date reported by UNAIDS.

Figure V
Actual and Predicted HIV Rates for Urban Pregnant Women



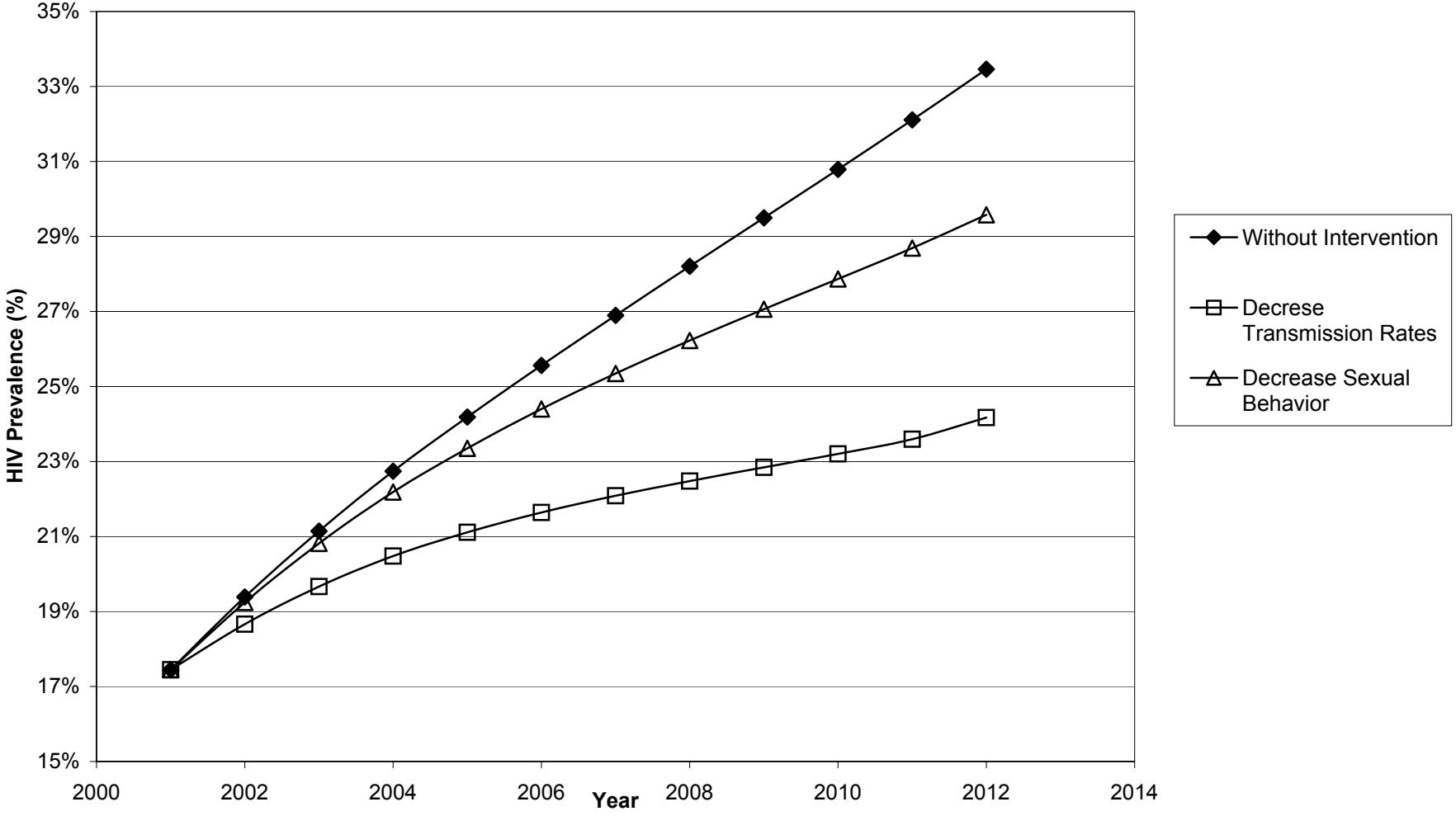
Notes: Actual HIV prevalence calculated from the U.S. Census Surveillance Database as an average for urban-dwelling pregnant women in the country between 1998 and 2000 (late), 1994-1996 (mid) and 1990-1992 (early). Predicted values are produced by the simulation model, and are an weighted average of the predicted rates for urban pregnant women from 1998-2000 (late), 1994-1996 (mid) and 1990-1992 (early), where the weighting is determined by the number of studies from each year that determine the actual HIV rate.

Figure VI
Actual and Predicted HIV Rates for All Individuals



Notes: Actual HIV prevalence is overall adult prevalence as reported by the UNAIDS 2001 and 2004 Factsheets . Predicted values are produced by the simulation model, and are an average for the end of the decade for all adults.

Figure VII
Epidemic Path With and Without Interventions



Notes: This figure shows the time path of the HIV epidemic in Africa under the case of no intervention, a 20% decrease in transmission rates and a 20% decrease in all aspects of sexual behavior.