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WHY DID HIV DECLINE IN UGANDA?

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ABSTRACT

Uganda is widely viewed as a public health success for curtailing its HIV/AIDS epidemic in the early 1990s. We investigate the factors contributing to this decline. We first build a model of HIV transmission. Calibration of the model indicates that reduced pre-marital sexual activity among young women is the most important factor in the decline in HIV. We next explore what led young women to change their behavior. We estimate that approximately one-third the reduction in HIV in this cohort and almost 20 percent of the overall HIV decline was due to a gender-targeted education policy.

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While the AIDS epidemic has exacted a terrible price in sub-Saharan Africa, Uganda is widely viewed as a success story and a model for the rest of the continent (Schoepf, 2003). The reduction in AIDS in Uganda was rapid: HIV prevalence fell from an estimated 15% of the general population in 1990 to 5% in 2007 (UNAIDS, 2008). The steepest decline occurred in the early 1990s: between 1990 and 1995 the prevalence of HIV among urban pregnant women dropped from approximately 30 to 15%. This fall in HIV prevalence was most impressive for women under the age of 25.

A number of hypotheses have been put forward to explain the downward trend. One set of theories focuses on behavioral changes (USAID, 2002), potentially spurred on by fear of HIV acquisition or Uganda's well-known Abstinence, Be Faithful and use Condoms (ABC) Campaign. A less encouraging theory stresses epidemic maturity and high rates of AIDS-related mortality (Wawer et al., 2005).[1]

Epidemiologists have investigated the decline in HIV among young cohorts in Uganda, but the results have been inconsistent. Contrasting behaviors in Uganda with behaviors in other countries, Stoneburner and Low-Beer (2004) emphasize the role of reductions in non-regular partnerships, particularly among urban males, in explaining the reduction in HIV. Using the same data but a slightly different definition of non-regular partnerships, however, Asiimwe-Okiror and colleagues (1997) do not find a significant decline in non-regular partnerships.[2] Rather, they conclude that the reduction in HIV was due to a delay in sexual intercourse and increased condom use. However, both studies lack micro-level data on HIV prevalence in the general population. And neither study examines *why* behavior changed.

[1] In the time period we are considering, there were no effective AIDS treatment in Uganda, and so we do not consider a theory of better medical care.

[2] The definition of non-regular partnerships (NRP) used by Stoneburner and Low-Beer are sexual partnerships within the last 12 months, other than those with a spouse or a regular partner, divided by all persons in the survey. The Asiime-Okikor et al., (1997) definition is the same numerator, divided by only those individuals who are sexually active. This mechanically makes the NRP number for Stoneburner and Low-Beer lower. This difference may account for the disparate conclusions. Using the Stoneburner Low-Beer definition, NRP could be reduced due to population growth or an increase in the number of people who used to have non-regular partners, now not having sex (in which case the measure is conflating a measure of secondary abstinence with a reduction in partnerships). In this paper, we have and therefore use data on extra-marital partnerships as opposed to non-regular partners.

The aim of this paper is to understand both how and why HIV declined in Uganda. We first develop a model of HIV transmission. The model is used to identify the most important factors contributing to the decline. To calibrate the model, we rely heavily on HIV testing and sexual behavior questions from the 1987 Knowledge, Attitudes and Practices (KAP) Survey conducted by the Ugandan Ministry of Health (Konde-Lule et al., 1989). We supplement these data with questions on behavior change from the Demographic and Health Surveys (DHS) and HIV data from antenatal clinics.[3]

We simulate the impact of reduced pre-marital and extra-marital sex, greater condom use, and selective mortality on the time pattern of HIV. All told, we explain 83% of the reduction in AIDS in Uganda. Our results show the rapid decline in HIV was driven by a reduction in pre-marital sex among young women. This factor accounted for 59% of the decline in this cohort and 36% overall.

We next ask what led young women to abstain. The time period of rapid HIV decline among this cohort coincided with a dramatic rise in secondary school enrollment. To identify whether there is a causal relationship between these contemporaneous trends, we instrument for secondary school enrollment with distance to secondary school, conditional on county fixed effects. We find that girls' enrollment in secondary education significantly increases the likelihood of abstaining from sex.

One could argue that girls stayed in school because they were fearful of contracting HIV. Although this may be partly true, we find that some of the schooling increase among young women was in response to a 1990 affirmative action policy giving women an advantage over men on University applications. Using men as a placebo group and exploiting heterogeneity in birth year and distance of birthplace to public Universities, we show that the University preference policy was effective in recruiting women into higher education. Our findings suggest that one-third of the 14 percentage point decline in HIV among young women and

[3]Another survey we might have used in the analysis is the Uganda Knowledge Attitudes and Behavior (KAB) Surveys in 1989 and 1995. Unfortunately, the questionnaire design differed across time, making it difficult to compare indicators. Sample coverage also varied and was not always nationally representative (see Appendix B of Bessinger et al. (2003) for a discussion of the merits of the DHS over the KAB).

approximately one-fifth of the overall HIV decline can be attributed to this policy.

Our study contributes to the debate regarding the role of behavior change in curtailing the HIV epidemic in Africa. Oster (2012) has argued that behavior change is muted in Africa due to the (perceived) high risk of mortality, which deters investments in healthy behaviors. Other social scientists have questioned the malleability of sexual norms in Africa, citing entrenched cultural practices as obstacles to behavior change (see UNECA (2008) for a review). On the other hand, Lakdawalla et al. (2006) as well as Philipson (2000) suggest HIV-related mortality may actually reduce risky behavior. Our study documents behavior change in the face of very high HIV mortality.

Our study also contributes to the literature on the role of education in health. Many investigators have noted a positive relationship between maternal education and child survival (see Schultz (2002) for a review). Our findings concur with Baird et al. (2011) and Duflo et al. (2011), who have documented a negative correlation between girls' schooling and sexual activity in randomized trials. However, this is the first study to link increased opportunities for higher education among women with decreasing HIV prevalence on a population scale.

The rest of the paper is structured as follows. The first section discusses the AIDS epidemic in Uganda. Section II presents our simulation model and tests of validity. Section III then examines the impact of risk practices and mortality on AIDS prevalence over time. Section IV investigates the factors that led to behavior change and the last section concludes.

I HIV in Uganda

HIV is postulated to have been introduced to Eastern Africa from Central Africa, where it emerged in the 1960s. Findings by Smallman-Raynor and Cliff (1991) suggest that the Ugandan HIV epidemic became generalized in the mid-1980s, when civil war ended and infected troops returned to civilian life. Oster (forthcoming) has noted a positive relationship between HIV incidence and export value, particularly in the early stages of the HIV epidemic in sub-Saharan Africa.

The most complete data on HIV status over the time period of interest comes from antenatal clinics (ANC, described below); most pregnant women attend such clinics, and many Ugandan clinics have standard HIV screening.[4] The screened prevalence rates from antenatal clinics are typically sent to the Joint United Nations Programme on HIV/AIDS (UNAIDS), which compiles them.[5] We use ANC data from four cities that report serial, age-specific infection rates and sample size (see figure 1).[6]

Figure 2 shows HIV positive rates in Uganda between 1987 and 2005. In 1987, about one-quarter of pregnant women in urban areas in Uganda tested positive for HIV. Between 1987 and 1990, the HIV positive rate rose by 5 percentage points. It then fell markedly. The cumulative reduction in HIV in urban areas of Uganda was approximately 20 percentage points, or about two-thirds of the pre-reduction peak. The majority of this decline occurred between 1990-1995. This is the time period of focus in our analysis.

The trends in Uganda are in stark contrast to the trends in other sub-Saharan African countries (figure 3). Kenya, just east of Uganda, had an urban ANC-based HIV prevalence rate of about 16% from 1993 to 2001 and has only recently experienced a decline. Tanzania, a Southern neighbor of Uganda, likewise has not seen an appreciable decline. The ANC rates of Southern African countries hardest hit by the epidemic (for example, Botswana and South Africa) continued to climb over this time period.

To presage our later analysis, we start by analyzing the dimension of the AIDS epi-

[4] Montana and colleagues (2008) compared HIV prevalence estimates from recent antenatal clinic surveillance with population-based surveys in sub-Saharan Africa. Overall, the prevalence estimates for Uganda were roughly similar (6.4% in the population-based survey vs. 6.0% in the ANC surveillance data). For all countries included, the ANC estimates were most accurate in urban areas, which is the location of greatest HIV decline in Uganda (see figure 2). In the 1980s, the majority of Kampalan women attended an antenatal clinic at least once during their pregnancy.

[5] In compiling the data, UNAIDS uses the median percentage rate of all antenatal clinics reporting in a given year, since some of the clinics do not include the number of women tested. The trends are the same, however, when looking at clinics present over the entire time period or forming a chain index linking clinics in adjacent years.

[6] Sample size is needed so the HIV prevalence can be averaged across sites. Detailed ANC data are available from the United States Census Bureau. The specific data used in our modeling exercise was obtained by using filters for high-quality data, age group and urban/capital city. Including rural areas in the filter allows Gulu district to be incorporated into the analysis, but Gulu does not start reporting until 1993. Moreover, Gulu is located in northern Uganda where the Lord's Resistance Army was active during the period of study. Entebbe starts reporting age-specific data in 2002 and is used to verify broad trends.

demic in Uganda, especially between 1990 and 1995, and the groups for which it declined most rapidly. Population-level HIV sero-surveillance data are available for 1987 from the Knowledge, Attitudes and Practices Survey. The Survey was carried out in two peri-urban communities: the Kasangati region which lies 10-20 km north of Kampala and the Nsangi region which lies 10-20 km south. The data include responses from nearly 4000 Ugandans over age 15 as well as serological testing. Respondents were asked general questions about HIV knowledge, and detailed sexual histories were obtained. Blood samples were collected and tested for the presence of antibody to HIV.[7]

Seroprevalence data from the KAP Survey in 1987 are shown in figure 4. The hardest hit demographic groups were slightly older never married women and young women in all marital categories, with older, never-married men also having high rates. The rate for never married young women is always above the rate for never married men, and the rate for married young women is above the rate for married young men.

These differences suggest a division of the population into six groups, which we employ below: three groups of men and three groups of women. We label the male groups α, β, and δ (table 1). α males are never married men aged 20 and older, and widowed or divorced males. As these men age, they accumulate wealth and are therefore better situated to buy sex from young girls or commercial sex workers (CSW). The 1987 KAP Survey shows this group has a HIV prevalence of 17%. β males are younger and have little financial means and are therefore less likely to be engaged in commercial sex transactions. Their observed HIV positive rate is 10%. δ males are the lowest risk group. These are older men with several wives. We find these men have the lowest observed HIV (6%) and reported sexually

[7]The regions were divided into administrative zones with approximately 10 clusters of equal size. Initial homesteads for interview were identified with the help of local chiefs and twenty neighboring homesteads were surveyed in order to achieve a representative sample. Interviews were conducted in the local language by 10 teams who had received health training for a week prior to survey onset. Surveys were undertaken using a standardized questionnaire and blood samples were drawn from each respondent and tested for the presence of antibody to HIV. HIV status was assessed using Enzyme Linked Immunosorbent Assays (ELISA). It is standard in developed countries to confirm a positive ELISA with a Western Blot. Financial considerations prevented the investigators from performing Western Blots on all samples; however, a concurrent study in the same laboratory reported that 93% of positive ELISAs were true positives as evidenced by confirmatory testing.

transmitted infections (STI) rate.

Corresponding to the α males are a group of μ women, who represent CSWs. Our sample does not contain self-described CSWs. This may be because women are hesitant about admitting they are engaged in such an activity. We take the HIV prevalence among older never married women as the rate among CSWs. The λ group includes never married and young (<25) married women. Because these women frequently have sex with older, never married and widowed/divorced men, they are at high risk of HIV. Their observed HIV prevalence is 19%. ω women are older married women and women who are widowed or divorced. Older married women rarely have high risk sex, and thus have relatively low HIV positive prevalence rates–9% in the KAP Survey.

The 1995 Ugandan DHS does not contain data on HIV status of individuals. However, we do have data on age-specific changes for women from the ANC.[8] Figure 5 shows the trend in HIV positive rates by age in Kampala among pregnant women. The HIV rate fell most dramatically at younger ages, and was relatively unchanged among older women. Indeed, the rate was flat among women aged 30 and older. In our analysis, we concentrate on explaining the trend for young women in the early nineties, though we remark on the older women as well.

II Modeling the HIV Epidemic in Uganda

Background

Several models of AIDS transmission within Africa have been developed in the literature. All have to contend with the biological fact that the rate of HIV transmission is very low and the HIV prevalence among certain age and sex groups is particularly high. Jeremy Magruder (2011) argues that age-independent models of HIV transmission that focus on heterogenous preferences for risk or networks of concurrent sexual partners cannot explain

[8]Since only some clinics report data by age, the mean rate across all groups is not the same as the unconditional mean across all clinics.

the age-distribution of HIV-related deaths in South Africa. Magruder develops a model of spousal search showing that the riskiness associated with finding a partner through serial monogamy (at a time of acute HIV infection) can generate a positive skew in the age distribution of HIV. His paper reaches the same general conclusion as ours, namely that pre-marital sexual relations–which Magruder refers to as "marital shopping"–are key to understanding the dynamics of HIV spread in Africa.[9]

However, there are several points of difference between our model and Magruder's. First, while Magruder is trying to understand what pattern of sexual activity could have generated a generalized HIV epidemic, our question is what caused HIV to decline. Second, the matching model might not fit the Ugandan experience as well as in South Africa. For example, the median person stays single for 8.5 years in that model, which does not fit the empirical data for Uganda well (table 2). Finally, the richness of our dataset allows us to calculate and predict, as opposed to stochastically generate, events in an individual's lifetime. Age of sexual debut and age of first marriage are not randomly drawn from the uniform distribution but predicted using conditional distributions. Mixing probabilities are not obtained via random draws but are computed from a system of linear equations.[10]

Data and Model Assumptions

The majority of HIV transmission in sub-Saharan Africa is from heterosexual intercourse. We thus model transmission through that route. An individual has a probability of contracting

[9] Our analysis does not explicitly model concurrency. Through our mixing probabilities (see discussion below) individuals distribute their sexual activity among many partners. This does not entirely capture the phenomenon of concurrency because it neglects the enhanced risk associated with having sex with a partner who has recently acquired HIV. However, a large cohort study designed to test for the role of concurrency in the HIV epidemic in sub-Saharan Africa demonstrated that it had no explanatory value; rather, it was the number of partners over time that predicted HIV infection (Tanser et al., 2011). Concurrency should be contrasted with intergenerational sex, which we also do not assume but is a prediction of our model (see table 3).

[10] Compartmental models (such as the Susceptibility-Infected (SI) model of May and Anderson (1991), are common in the HIV modeling literature as well (i.e., Stoneburner and Low-Beer (2004)). SI models assume all behavior in a compartment is identical (usually the average value of some parameter) which may lead to bias when parameters (i.e., number of sexual partners) are not normally distributed, or when the relationship between sexual behavior and HIV is nonlinear.

HIV with each unprotected coital act with a person who is HIV positive. That probability depends on four factors: the presence of another STI, HIV viral load, gender and age.

Taking these factors into account, the probability that an individual female i (individual male j) who is a member of group f (member of group m) contracts HIV during a given coital act with a member of group m (member of group f) can be expressed as:

$$\begin{aligned} & \Pr\left(i \text{ contracts HIV per sex act with member of group } m\right) \\ = \; & \Pr\left(HIV\ transmission\right) \cdot \Pr\left(member\ m\ HIV+\right) \cdot \Pr\left(no\ condom\ use\right), \end{aligned} \tag{1}$$

where m is an element of the set $\{\alpha, \beta, \delta\}$ for women and f is an element of the set $\{\lambda, \mu, \omega\}$ for men. The N_i total coital acts of female i (N_j for male j) are calculated as follows:

$$N_i = D_i \cdot C_i, \tag{2}$$

where D_i refers to the duration sexually active and C_i refers to the frequency of coital acts. The N_i are distributed across the m groups (f groups) according to the mixing probabilities, $\pi_{fm}\left(\pi_{mf}\right)$. The mixing probabilities must satisfy two conditions. The first is the conservation of sex acts: coital acts between groups A and B must equal those between B with A. Let $N_f = \sum_i N_i$ conditional on i being a member of group f, and similarly, $N_m = \sum_j N_j$. The conservation of sex acts then implies:

$$\pi_{fm} \cdot N_f = \pi_{mf} \cdot N_m. \tag{3}$$

The second condition is that all sex acts are accounted for–that is, there is a partner for every coital act. This amounts to the restrictions that:

$$\sum_{m=1}^{m=3} \pi_{fm} = 1 \text{ and } \sum_{f=1}^{f=3} \pi_{mf} = 1. \tag{4}$$

The equation governing whether female i has contracted HIV is:

$$\Pr\left[i \text{ contracts HIV}\right] = 1 - \prod_{m} \left\{1 - \Pr\left[i \text{ contracts HIV from group } m\right]\right\}^{N_i \cdot \pi_{fm}}, \qquad (5)$$

and similarly for male j :

$$\Pr\left[j \text{ contracts HIV}\right] = 1 - \prod_{f} \left\{1 - \Pr\left[j \text{ contracts HIV from group } f\right]\right\}^{N_j \cdot \pi_{mf}}. \qquad (6)$$

Estimating equations (5) and (6) requires three types of data: individual sexual histories (including HIV status), mixing probabilities, and HIV transmission probabilities. We discuss the source and construction of these data in several steps outlined below. Parameters and sources are summarized in Online Appendix C.

The sexual history of each individual includes when the individual debuted sexually, when they married (if applicable), how frequently they have sex, how many partners they have had within and outside of marriage, whether they have contracted a STI and the use of male condoms. Because HIV prevalence rates were low prior to the mid-1980s, we take the sexual history to be the past 10 years or the time from sexual debut, whichever interval is shorter.

Estimating D_i. The 1987 KAP Survey includes age, marital status, wife rank, HIV status, STI and fertility history, and the number of sexual contacts in the last six months for each individual. Data on age of first marriage, age of first sex and coital frequency are imputed. For women, we regress age of marriage/first sex on education and current age using the 1988 Uganda DHS (Online Appendix A table A1). These coefficients are applied to the KAP Survey and allow us to calculate duration of pre-marital sex and duration married. The 1988 Uganda DHS does not include male respondents. Thus, we use the 1995 Uganda DHS and perform the same OLS analysis restricting the sample to men alive during the late 1980s. This may introduce survivorship bias into our estimates as men who survived until 1995 could be different than those who died from HIV or other causes.

Estimating C_i. To predict coital frequency, we employ a negative binomial model using

a quadratic form in age (Online Appendix A table A2). For married men, coital activity is predicted from the females' by randomly matching each husband with their reported number of wives and ascribing the wives' sexual activity to their husband.[11] It would not be appropriate to assume single men were as sexually active as young single women; as noted, many single women will have sex with married men. Thus for this category, we estimate coital frequency using the Tanzanian DHS 1991-2. We compare the relative frequency of sexual activity among single males versus single females in the Tanzanian data from around the same time period and use those proportions as coefficients multiplying the mean number of acts per month of the Ugandan single women to obtain acts for single Ugandan men.

Table 2 reports the imputed age at first marriage, age of sexual debut and coital frequency among KAP respondents. For women, the mean age at first marriage is 17.3 years (in the 1988 Uganda DHS, the average age of marriage is 17.0). For men, the age at first marriage is older, 23.4 years (in the 1995 Uganda DHS, the mean age at first marriage for the surviving cohort is 22.5). The mean age of sexual debut is 14.9 for women and 17.3 for men. Married men have on average more sex with their wives than wives do with their husbands; this is due to polygamy.

Estimating π_{fm}. Apart from knowing how long and with how many partners an individual has been sexually active, we need an idea of who they were having sex with. Our data do not indicate who the sexual partners are. Thus, we need to estimate the probability that one group mixes with another. We use an algorithm based on equations (3) and (4) to calculate the group mixing probabilities. Without further restrictions, the model cannot be solved, since there are not enough linearly independent equations. Specifically, omitting unobserved CSWs, there are 15 unknown mixing probabilities and 11 equations (five are accounting equations and the other six are adding up constraints). In order to reduce the dimensionality of the matrix, we impose restrictions, explained in more detail in Online Appendix B. The primary restriction is that the rate of STIs among men in a given group is

[11] To derive coital frequency for men and women reporting extra-marital partnerships, the coital frequency within marriage is deflated by a constant of proportionality.

inversely proportional to the probability of mixing with older married females and directly proportional to the probability of mixing with CSWs.[12]

The sexual relationship probabilities for the baseline model are given in table 3. Each cell in the table reports two values. The first is the probability of a woman in the indicated row having sex with a man in the indicated column, conditional on having sex. The second is the probability of a man in the indicated column having sex with a woman in that row. Note that since we do not observe CSWs in our data, we do not estimate the distribution of sexual acts for CSWs.

The λ young women are mixing a disproportionate amount of time (84%) with the highest prevalence α males. λ women are spending the least amount of time with the lower prevalence, older married men. In addition, high risk α males not only mix with the λ women, but also mix around a third of the time with CSWs, making them an excellent bridge population, transmitting the disease from sex workers to young women. Three-quarters of the sexual activity of ω women is with older, lower risk, married males.

HIV Transmission Rates. Also needed to estimate equations (5) and (6) are HIV transmission rates. The transmission probabilities used in the study are presented in Online Appendix A, table A3, and reflect the aggregation of many biomedical studies. These studies find higher transmission rates during acute HIV, in the presence of genital ulcers, and among younger individuals, particularly young women (Boily et al., 2009; Gray et al., 2001; Pettifor et al., 2007).[13]

Testing the Predictions

We use the data on reported number of children to see if the estimates of coital frequency are reasonably accurate. A particular concern is that young unmarried women may underreport

[12] Older women have the lowest overall rate of STIs (10% for women 25 years of age or above, versus 15% for women younger than 25; whereas in men, the prevalence ranges from 24 to 50%).

[13] Young women are particularly at risk for acquiring HIV through receptive intercourse (Karim et al., 2010). Cervical ectopy (Moss and colleagues, 1991), reproductive tract immaturity and the forceful penetration associated with rape are all considered supportive factors in male to female HIV transmission.

their amount of sexual activity (Gersovitz et al. (1998)), which would affect not only the estimated HIV prevalence in this cohort, but among all cohorts since the mixing probabilities would be biased (see equations 3 and 4).[14] However, using the 1988 Uganda DHS, we do not find systematic evidence of duplicity—the predicted (based on reported sexual activity) and actual number of births is not statistically different (p-value=0.72). For never married females living in Uganda's Central region, 70 women out of 474 report having a child in the last year (15%); we predict 66 births (probability of conception from Wilcox et al. 1995). The paradoxical observation–young unmarried women have the highest HIV prevalence despite low coital frequency–is then explained by the fact that their partners were particularly high risk, which is precisely what our model suggests. We also find evidence that men were not lying about their higher number of partners and frequency of sex, as represented by their higher rate of reported sexually transmitted infections.[15]

We can use the model to estimate the predicted HIV positive rates directly, and compare them to the actual prevalence. We start by assuming that there was no HIV among any of the groups in 1977 except for CSWs (μ) who we seed with an (arbitrary) HIV prevalence of 5%. We further make the conservative assumption that men were mixing twice as often with CSWs during wartime (in the late 1970s when Uganda was at war with Tanzania, and when Yoweri Museveni overthrew President Obote in the mid-1980s).[16] We then iterate the model forward year by year, updating the prevalence among all groups each year until 1987.[17]

Predicted versus actual prevalence are reported in table 4. The model is calibrated fairly

[14] Although Gersovitz et al. (1998) did find large differences between men and women in the reporting of sexual activity, ultimately they were not able to distinguish between the external hypothesis (that the difference is due to an omitted group, namely, CSWs) versus the internal hypothesis (that the difference was due to under-reporting by females).

[15] For example, β males have a reported STI prevalence of 35% and α males have a STI prevalence of 50%.

[16] From a 1989 Ugandan survey (when Uganda was not at war), 24% of men engaged in casual sex. This designation was thought to capture sex with prostitutes. According to the report: "In the absence of subgroups designated as prostitutes and their clients... sexual behavior designated as 'casual' was considered' (MoH, 1989). From a survey of African troops stationed in southern Sudan on the border of Tanzania, Kenya and Uganda in the 1980s, 50% had had sex with a prostitute (McCarthy and colleagues, 1989).

[17] Although it is possible to perform year by year updating of prevalence among the various groups, this comes at the cost of additional assumptions on sexual behavior. We thus use this methodology as a loose verification of our model rather than to calibrate our main results since the plausibility of the assumptions are difficult to verify.

well across most groups. Predicted rates generally average about the actual rate. Among women, for example, the predicted rate is 13.2%, compared to an actual rate of 13.1%. Among men, the predicted and actual rates are 10.9 and 10.6%. The biggest difference between predicted and actual rates is for older married men, for whom the model overestimates HIV prevalence by 30% (6.0 versus 4.2%). Overall, however, the results are fairly close.

Finally, we verify that our model matches the baseline ANC data on HIV positive rates by age. We average the earliest three years of available ANC data (1990-92) to obtain mean HIV prevalence rates among pregnant women by age group. We then compare the observed values for HIV with a model-generated weighted average of HIV infection per age group, where the weights are the probability a particular woman was pregnant.

Table 5 panel A shows the actual and predicted HIV prevalence rates by age group. The predicted rate is very close to the actual rate for young women, within 0.8 percentage points. The gap gets bigger for older women, where the predicted rate is 30% below the actual rate. This may be because for women who are widowed or divorced, we assume that their time was divided equally between divorce and marriage for the period under observation. This assumption may underestimate their sexual activity, since divorced women report a much lower coital frequency than married women.

III Explaining the Reduction in HIV in Uganda

We now use our simulation model to explain the reduction in HIV prevalence in Uganda. Since the change in HIV prevalence was greatest for young women, we focus on this group. We consider three explanations: increased use of condoms, less non-marital sex (both premarital sex among girls and reduced out-of-marriage sex among men), and death among people with AIDS.

There are two modeling issues to address. Because we are focusing on a very short period, the major issue is the direct impact of the various interventions, which are immediate, and not the indirect (feedback) effects as a result of changing prevalence and thus subsequent

infections, which occurs over time. We model each behavior change separately, and sum up the change in prevalence of HIV attributable to each.[18]

A second modeling issue is what to assume about the amount of sex when the supply of an available type of partner changes. Consider the simulation where young girls delay the age at which they debut sexually. This clearly affects the number of contacts they have with men. What impact does this have on sexual activity among men? At one extreme, one might assume that there are simply fewer opportunities for men to have sex, and thus overall sexual contacts among men decline. Alternatively, however, men might see CSWs more, or married men might have more sex within marriage.

Based on the limited empirical evidence available, we assume that the supply of sexual partners is relatively elastic.[19] Thus, if younger women delay their sexual debut, men, in the short run, will turn to alternative sources of supply, such as CSWs.

Abstinence

We start by considering the impact of less pre-marital sex. As demonstrated in table 6, young women in Uganda under the age of 25 increasingly waited and abstained from sex in the pre-marital period over our time period. The share of 15-19 year old never married women who were virgins increased from 50.3% to 68.7% between 1988 and 1995. Similar to Ueyama and Yamauchi (2009) we do find a slight increase in the percent married in the presence of HIV, but it is not statistically significant.

Less pre-marital sexual activity reduces the number of sex acts that women in this age group have and changes who they have it with. Online Appendix table B1 shows the change

[18] The only exception to this general rule is when we are modeling death, since the direct effect of that "intervention" is the removal of certain individuals from the population, thereby changing prevalence directly.

[19] Dupas (2011) shows that an intervention which informed young girls about the relative risk of HIV acquisition (as well as other HIV prevention messages) led to a substitution of sexual acts towards younger men and away from older men, but not a large reduction in overall sexual activity. Similarly, Luke (2001) demonstrates that "sugar daddies" prefer sex without a condom. However, if age and economic disparities are not great enough to impose this preference on their partner, these same men will use a condom and have sex with older, less impoverished women or commercial sex workers. Both of these findings suggest the short-run demand for sex is relatively inelastic.

in sexual mixing rates implied by delayed sexual initiation. Relative to the base case in table 3, young women have slightly more sex with safer groups. The much larger impact, however, is that they engage in far fewer sexual acts. Since many of these contacts are with high risk men, the predicted HIV rate falls as well. Men, in contrast, have more sex with CSWs, though we do not model HIV positive rates among men.

Table 5 panel B reports the results. Reduction in pre-marital sex accounts for an 8.3 percentage point reduction in HIV positive rates among teenage girls, roughly 59% of the observed decline. The decline is 1.3 percentage points among women aged 20-24, or 19% of the observed change. Reflecting the longer length of time prior to marriage, the impact of abstinence is negligible in women aged 25 and older.

Reduced Out-of-Marriage Sexual Contact

Increased faithfulness was a second goal of the Ugandan program. This would show up in the first instance as fewer out-of-marriage sexual contacts for men (recall that our data omit CSWs). In the 1995 Uganda DHS, men were asked whether they had changed their sexual practices to prevent AIDS and, if so, how. Only 16% of married men admitted to changing their sexual behavior so as to remain active with only one partner. The percentages for single men are much smaller.

When married men are more faithful, they have more sex with their wives (given the same frequency of sexual contact). Online Appendix table B1 shows the changes in the overall pattern of sexual activity implied by increased faithfulness. Given the small change in faithfulness reported, the impact on sexual mixing probabilities is small, as is the change in HIV positive rates. Interestingly, since many married men are HIV positive, increased faithfulness actually increases slightly the share of women who are HIV positive in the short run. Table 5 panel B shows that HIV positive rates increase by about 0.9 percentage points among the youngest cohort of women as a result of this change.

Increased Condom Use

Condoms were introduced into Uganda in 1991, but remained relatively scarce until 1997 (USAID, 2002). The 1995 Uganda DHS asked men about condom use. The specific questions included whether a condom was used at last sex; whether it was used when the man last had sex with his wife; and whether the condom was last used with a partner other than his wife. From this, we infer data on condom use by male category and in some cases female partner. Online Appendix A table A4 shows the responses for condom use at last sex. The rate is lower among married men, especially with their spouse and higher among older, never married men.

We assume condom use in extra-marital sex acts exhibited linear growth from zero in 1991 to 40% in 1995.[20] We also assume that condoms were used correctly. Under these assumptions and correcting for the impact of condom use on fertility, we derive the contribution of condom usage to the decline in HIV. The results are reported in table 5. Among women aged 15-19, increased condom use explains a 3.3 percentage point reduction in predicted HIV positive rates. This is 24% of the actual reduction in rates between 1990-92 and 1994-96. Condom use becomes less important for explaining the pattern of HIV as women age.

Death of HIV Positive Men

Those that died over this time period were primarily the high risk males, either through combat, aging or AIDS. Since older men were infected early by CSWs, they would be the first ones to succumb in the generalized epidemic. Timaeus and Jasseh (2004), using DHS sibling reports of mortality from several African countries, suggest that 20% more men than women die early in the AIDS epidemic. In addition, studies on the natural history of HIV have shown that age is an important determinant of survival: the older one is, the shorter the incubation period and faster the time to death (Coutinho, 2000).

[20] This is approximately the average condom use across all groups.

To model AIDS-related mortality, we extrapolate from Kaplan-Meier estimates of the cumulative probability of death within a year after an individual has been diagnosed with AIDS (defined as a CD4 cell count <200 cells/cubic millimeter). Based on the natural history of HIV infection, we modeled death as a 15, 22.5 or 30% mortality rate among α males over a horizon of 5 to 10 years. Each rate directly impacts the prevalence of HIV in the α group and necessitates a recalculation of sexual mixing probabilities. The best fit appeared to be a 22.5% mortality rate.

As men with AIDS die, this implicitly means that young women have more sex with younger men, who are less likely to have AIDS. Online Appendix B shows the new mixing probabilities, accounting for increased mortality among α males. Young women tend to have relatively more sexual contact with lower risk men, although the magnitude is not enormous. The direct effect of fewer older men being HIV positive is the greater impact.

Table 5 shows that death was an important component to the HIV decline and explains 18.6% of the predicted decline among women ages 15-19. Unlike condom use, death explains a larger share of the modest decline in HIV prevalence as women age. Overall our analysis shows that the most important factor in the decline in HIV was primary abstinence among young women. Abstinence in this group explains 59% of the reduction in HIV for teenage girls and 36% of the reduction overall.

IV Why Did Young Women Abstain? The Role of Education

In this section we examine what could have motivated such changes in sexual practice. One obvious correlate of the reduction in HIV was an increase in secondary school enrollment of girls in the late eighties and early nineties. This trend is apparent in both the DHS and the Census (figure 6). According to the Ugandan DHS, secondary education rose by 12 percentage points (23.8 to 35.9%) among 15-29 year-old women over the period 1988 to 1995. A similar trend was not noted in Kenya (34.7 to 35.4% between the 1989 and 1993 Kenya DHS) or Tanzania (17.9 to 15.1% between the 1991 and 1996 Tanzania DHS) over

approximately the same time.

The mechanisms by which education may lead to better health are still being elucidated, but there is evidence to suggest that the better educated avoid risky health behaviors (Cutler and Lleras-Muney, 2006). In Africa, de Walque (2007) and Duflo et al. (2011) have found evidence to support the view that girls' education leads to a delay in the initiation of sexual activity. The evidence is mixed, however, as Fortson (2009) concludes that education is a risk factor for HIV acquisition in sub-Saharan Africa.[21]

To examine the link between education and sexual activity for young Ugandan women in the early 1990s, we estimate an equation of the form:

$$Virgin_{ic} = \beta_0 + \beta_1 School\ Enrollment_i + X_i'\beta_2 + \gamma_c + \varepsilon_{ic} \tag{7}$$

β_1 tests whether school enrollment is correlated with virginity. γ_c is a county fixed effect, and X is a vector of individual controls, including age, religion and ethnicity. We include all women aged 15-24 in the 1995 Uganda DHS.

Of course, education and sexual activity are correlated for many reasons. For example, girls in sub-Saharan Africa are often forced to drop out of school if they became pregnant. To mitigate the effects of reverse causality and omitted variables, we take three steps. First, we limit the sample to women who report that they did not leave a school due to pregnancy. Second, we include county fixed effects and demographic controls that may affect girls' access and ability to stay in school. Third, we instrument for education using distance to secondary school. In poor countries such as Uganda, distance to school has been shown to be correlated with schooling (Bommier and Lambert, 2000; Glewwe and Jacoby, 2004).

Figure 7 shows the identification strategy visually. Virginity (the reduced form) and enrollment (the first-stage) are plotted against distance to secondary school for young Ugandan women (ages 15 to 24). There is considerable co-movement between the two outcomes and

[21] Fortson (2008) discusses whether this relationship is causal or driven by omitted variable bias and concludes she is not able to distinguish between the two.

both are inversely related to distance.

The results of the enrollment equation are presented in table 7. The first column shows the OLS estimate with only controls for birth year. Enrollment in school increases the likelihood of being a virgin by 45 percentage points conditional on year of birth. Adding controls for ethnic and religious background, county fixed effects and a proxy for local HIV prevalence does not materially change the point estimate.[22]

The last column of table 7 reports the two-stage least squares results. The effect of enrollment on virginity is slightly larger though less precise, consistent with measurement error in the self-reported schooling variable (Ashenfelter and Krueger, 1994).[23] The F-test for the impact of distance on school enrollment is 24.5, well above standard critical values. The coefficient is extremely large; being enrolled in school increases the probability of being a virgin by 73 percentage points.

Thus, it seems clear that staying in school has a large effect on sexual debut. Coincident with the reduction in HIV, figure 6 shows a steady rise in secondary education for young girls in 1990. We now turn to explaining this trend.

Affirmative Action and Higher Education

The rule of Idi Amin, Uganda's military dictator from 1971-1979, was particularly detrimental for women. His militia frequently used rape to suppress dissent and banned all independent women's organizations (Tripp, 2002). Following Museveni's rise to power, women gained the right to organize and sought elected office. One of their priorities was gender equality in education. These efforts led to the institution of an affirmative action policy in 1990 that gave women 1.5 extra points on their application to public Universities (Muhewzi, 2003). David (2007, p.20) describes the policy as follows:

[22]The Baganda are the wealthiest ethnicity in Uganda. We experimented with other controls for socioeconomic status (such as sex of household head and possession of durables). The results are qualitatively the same.

[23]If we also include a control for distance to urban center, the instrumented enrollment coefficient is still significant and increases to 1.47, but is much less precise (s.e. 0.75).

For a long time, there was only one University in Uganda, Makerere University.[24] This limited opportunities for both boys and girls. Even then, boys were more favoured by the system until the famous 1.5 was introduced to enable eligible females to access University education. The 1.5 was added to the total score of all individual girls as an affirmative action policy. This has increased girls enrolment from about 20% to nearly half the total number of students currently at Makerere University.

The response to the policy occurred rather quickly. Female enrollment in Makerere increased from 24% in 1989/1990 to 35% by 1993/1994 (MOES, 2001). Although only a small percentage of girls received tertiary education, the policy was well-known and may have affected the perceived returns to girls' higher education, especially for those living near a public University.[25]

Using data from the 2002 Uganda Census, we examine whether the rise in attendance at secondary school was associated with the introduction of the affirmative action policy. In addition to timing and gender, we utilize the differential impact of the policy on individuals born closer to a public University versus those further away (see figure 1). Our setup is similar to that of Duflo (2001):

$$\begin{aligned} S_{idk} &= \beta_0 + \sum_k \alpha_k age_i + \sum_d \delta_d district_i + \beta_1 female_i + \beta_2 \left[young_i * \ln(dist)_i^{-1}\right] \\ &\quad + \beta_3 \left[\ln(dist)_i^{-1} * female_i\right] + \beta_4 [young_i * female_i] + \beta_5 \left[female_i * \ln(dist)_i^{-1} * young_i\right] \\ &\quad + \beta_6 Z_{dk} + \varepsilon_{idk}, \end{aligned} \tag{8}$$

where S_{idk} is an indicator variable for whether individual i in district d of age k completed secondary school or obtained more than seven years of schooling. Age is a set of cohort dummies. *dist* refers to distance from the centroid of the district the respondent was born in

[24] Makerere opened in 1922. Mbarara University of Science and Technology opened in 1989. No other public Universities opened until the late nineties (see figure 1). Three small private institutions also opened over this time period (two of which had an enrollment of approximately 80 students each).

[25] Perception of the returns to schooling has been found to be particularly important in determining years of schooling completed in developing countries (Jensen, 2010).

to the nearest public University.[26] We use the district of birth to avoid endogenous mobility. Young refers to children aged 8-12 in 1990, on the cusp of entering secondary school the time when the policy was initiated. The control group includes individuals ages 19 to 23 in 1990, who, on average, would have been too old for secondary school.[27] Z_{dk} is the (normalized) district-cohort population.

Table 8 panel A shows the results of the coefficient on the triple interaction between female, young, and the inverse log of distance. The triple interaction is positive and statistically significant–that is, secondary school completion rates rose more for young girls near the Universities than for older girls in those areas, or for boys living in those same areas. The same is true for the alternative measure of higher education, obtaining more than seven years of schooling (panel A, column 2).

Table 8 panel B presents the results of the placebo experiment (comparing women aged 19-23 in 1990 to those aged 24-28). Using either measure of higher education, the triple difference fails to achieve significance at conventional levels and is significantly different from the estimates in panel A.

We can extend the analysis by allowing the treatment intensity to vary for each birth cohort. Specifically, we replace the young dummy variable with a set of age dummies in each of the parts of equation (8):

$$\begin{aligned} S_{idk} &= \beta_0 + \sum_k \alpha_k age_i + \sum_d \delta_d district_i + \beta_1 female_i + \sum_k \zeta_k \left[age_i * \ln(dist)_i^{-1}\right] \\ &\quad + \beta_2 \left[\ln(dist)_i^{-1} * female_i\right] + \sum_k \mu_k \left[age_i * female_i\right] + \sum_k \lambda_k \left[age_i * \ln(dist)_i^{-1} * female_i\right] \\ &\quad + \beta_3 Z_{dk} + \varepsilon_{idk} \end{aligned} \tag{9}$$

Individuals age 30 in 1990 are considered the control and this dummy is omitted from the regression.

[26] Distance is measured in meters (so there are no distances ≤ 1). We use the inverted log of this distance so that we are capturing the effect of being relatively close to a public University.

[27] Primary school usually starts around age 6 though some children in Africa start later. Secondary school starts around age 13 until age 18.

The coefficients of interest are the $\lambda_k's$, which show the secondary schooling completion rates for girls versus boys, near versus far from the public Universities, before and after the policy was put in effect. The $\lambda_k's$, as well as their three year moving average, are graphed in figure 8. The vertical lines represent the approximate age of entry into primary school, secondary school, and University in 1990. There is a steep upward slope in female secondary school attendance between ages 12 and 18 (lines 2 and 3), the age groups where University admissions would be increasingly important. The effect flattens out for those in primary school at the time of the policy change.

The one anomalous feature of the chart is a decline in relative secondary school completion for girls less than 6 years old in 1990. This is likely because many young children have not yet reached the age of secondary school completion at the time of the 2002 Census. In addition, Uganda implemented a Universal Primary Education initiative in 1997, which led to compositional changes in the students enrolled in primary school, and this may have had spillover effects for those in secondary school.

The evidence presented in this section suggests that education policies targeting women can account for some of the increase in secondary education during the early nineties. To evaluate the impact of the education policy on HIV we need three estimates: the impact of the policy on female enrollment, the impact of enrollment on virginity and how abstinence affects HIV. For the first of these, we modify equation (8), changing the educational outcome to be a dummy for whether an individual was still in school at a given age, to find the average enrollment induced by the educational policy for girls aged 13-19. This increase is 14.4 percentage points. We multiply this number by the two-stage least squares estimate of the impact of enrollment on virginity (73 percentage points, from table 7) to calculate that the policy increased virginity by about 10 percentage points. Our simulation model implies a 10 percentage point increase in virginity would lead to a 4.6 percentage point reduction in HIV infection, about one-third of the reduction in HIV for this cohort of women and one-fifth of the overall decline. While this calculation clearly has uncertainty, our tentative conclusion

is that about one-third of the reduction in HIV for younger women seems to result from the education policy supporting women in higher education admissions.

V Conclusion

The decline in HIV in Uganda in the early 1990s was concentrated among young women. Among 15-19 year old pregnant women in Kampala, for example, HIV positive rates fell from 30% in the early 1990s to 15% in the mid-1990s. By building a simulation model of HIV, we established abstinence among young women as the most important factor contributing to this decline.

We next explored why young women chose to abstain. We found that the percentage of females enrolled in secondary school increased greatly in Uganda as compared to other East African countries over this time period. Instrumenting for school attendance with distance to secondary school, enrollment was strongly predictive of (reported) virginity. Exploiting heterogeneity by gender, birth year and location, we found girls stayed in school longer partly in response to the increased opportunities for higher education brought about by an affirmative action policy.

There are two natural extensions to our work. First, it would be useful to extend our analysis to later years to explain the second phase (albeit much less steep) of Uganda's HIV decline. Researchers tackling more recent periods have to contend with the introduction of HIV treatment and the complexities (moral hazard, decreasing transmission due to reduced viral load) that the expectation and distribution of antiretroviral therapy introduces into the analysis. Further, HIV prevalence may mechanically rise as more people with HIV are kept alive on treatment.

Second, our results support the view that encouraging girls to stay in school delays their sexual debut and reduces their lifetime risk of acquiring HIV. Do these results imply that gender-targeted education policies are especially effective ways to curtail HIV? A complete answer to this question would involve additional research on the costs and benefits of such

policies for both men and women in the long run. For example, a greater supply of educated women may lead to more female policymakers promoting an agenda of gender equity (Duflo and Chattopadhyay, 2004). On the other hand, if men prefer less-educated spouses, this could tighten the marriage market and lead to more marital shopping. This, too, we leave to future research.

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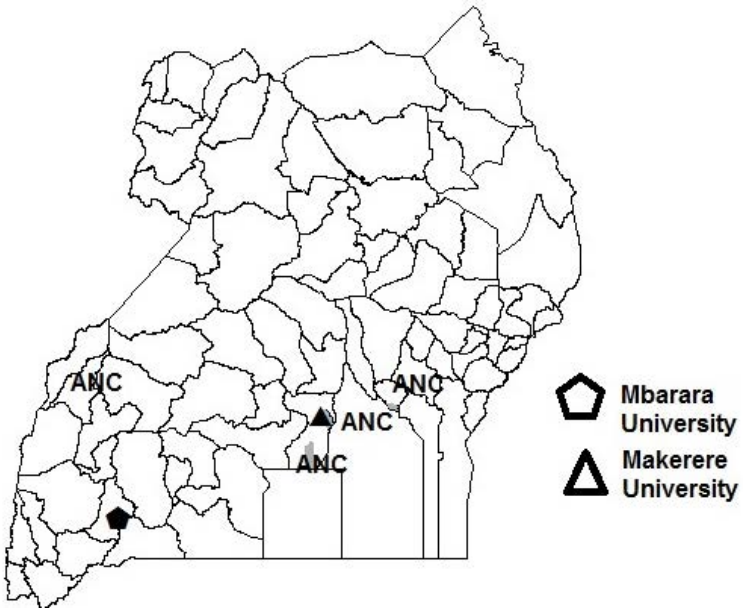
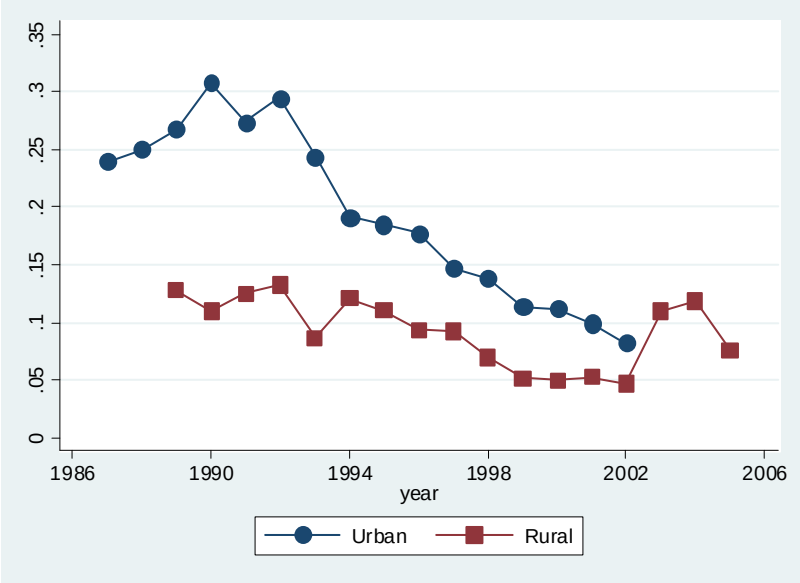
VI Figures

Figure 1: Map of Uganda



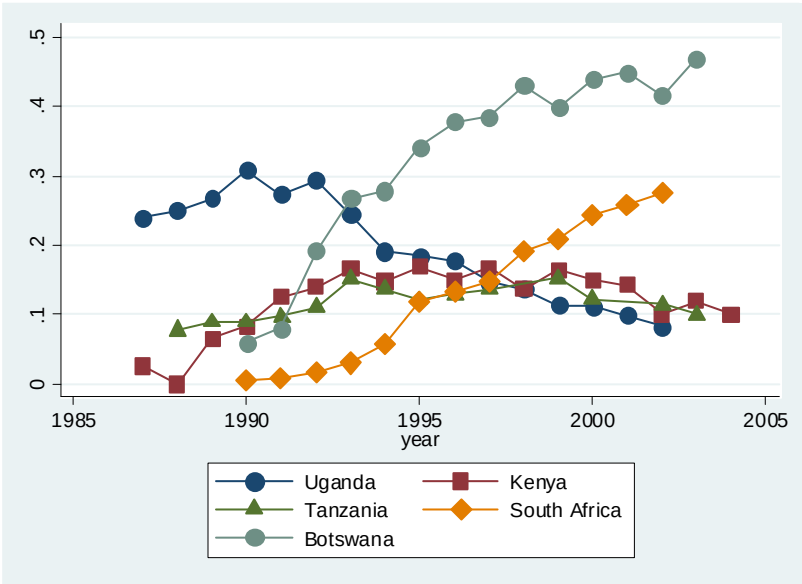
Notes: Map of Uganda showing ANC cities, districts and public Universities.

Figure 2: Median HIV Prevalence Among Pregnant Women, Uganda



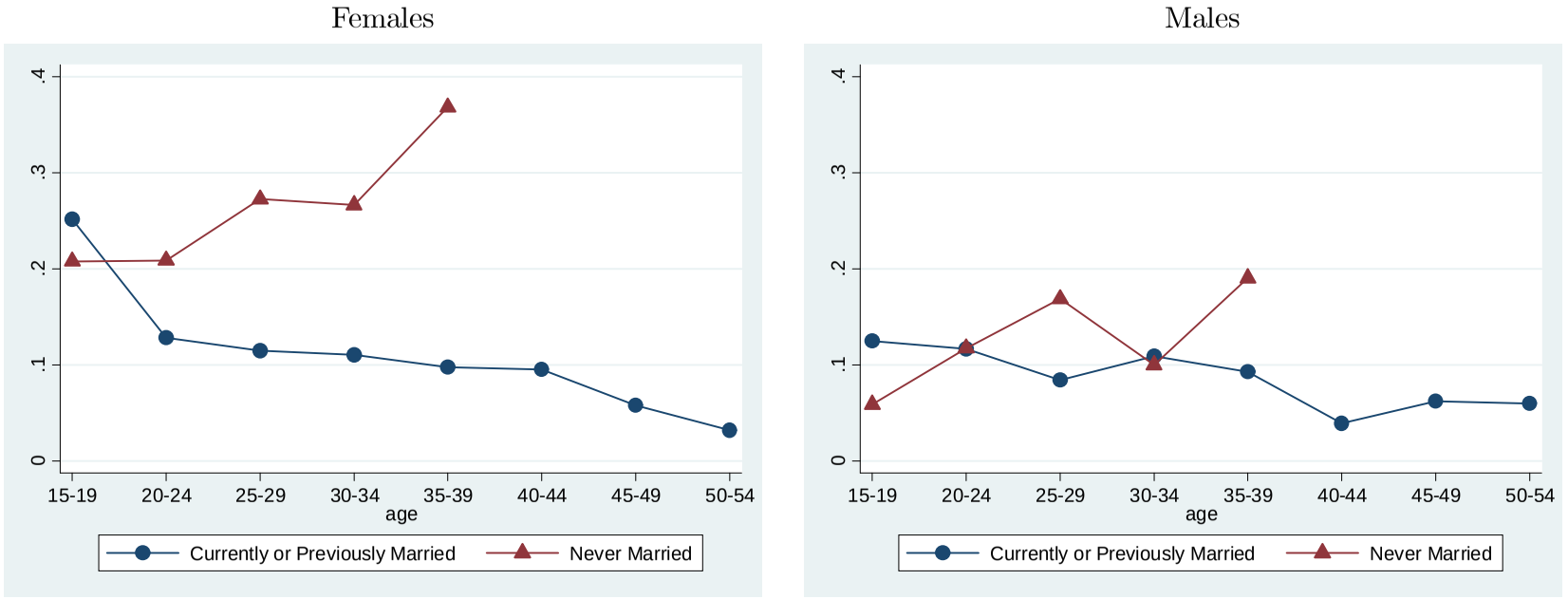
Notes: Data are from the UNAIDS Uganda Epidemiological Fact Sheets on HIV/AIDS from 2004, 2006 and 2008. Median annual prevalence is from antenatal clinics that performed surveillance HIV testing.

Figure 3: Median HIV Prevalence Among Pregnant Women in Urban Areas, Select Countries in Africa



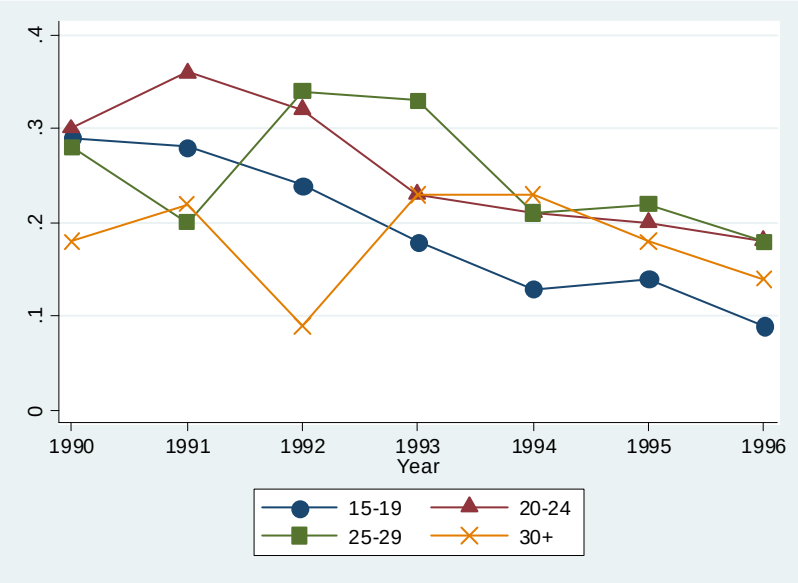
Notes: Data are from the UNAIDS Epidemiological Facts Sheets on HIV/AIDS for the relevant countries from 2004, 2006 and 2008. Median annual prevalence is from urban antenatal clinics that performed surveillance HIV testing.

Figure 4: HIV Prevalence by Marital Status and Age



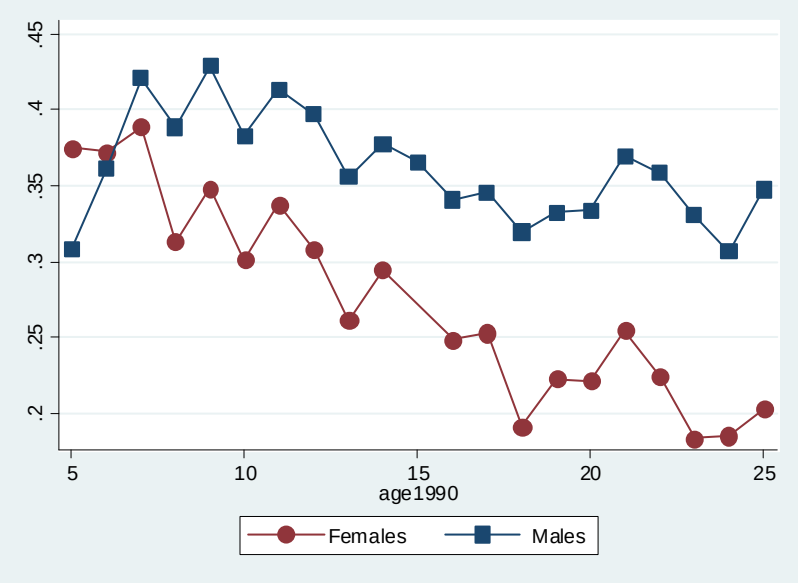
Notes: Data are from the 1987 KAP Survey. The prevalence is the percent of individuals in each subgroup with a positive HIV-1 antibody test. For never married women and men, after a certain age (45 for men, 40 for women), there are very few people. Specifically, there are 24 females and 17 males above these cutoffs. We omit those values.

Figure 5: HIV Prevalence Among Pregnant Women by Age Group, Kampala



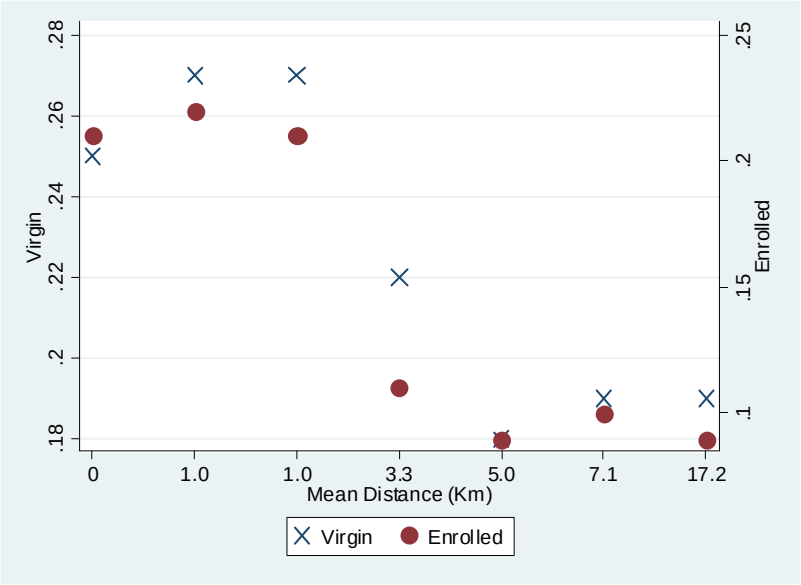
Notes: Data are from the United States Census Bureau, HIV/AIDS Surveillance website.

Figure 6: Secondary Schooling by Gender, Uganda



Notes: This figure shows the trend in receipt of greater than seven years of education for Ugandan females and males living near to a public University. Data are from the 2002 Uganda Census.

Figure 7: Distance to School, Virginity and Secondary School Enrollment Among Young Ugandan Women



Notes: This figure plots the correlation between virginity, enrollment and distance to the nearest secondary school. To construct these figures, we bin distance into 7 equal sized bins and plot the mean of the outcome within each bin. The mean of the distance within each bin is indicated on the x-axis. Data are from the 1995 Uganda DHS. Sample includes Ugandan women aged 15-24.

Figure 8: Effect of Affirmative Action on Girls' Secondary School Completion

Notes: This figure plots the triple interaction coefficients and their three year moving average from equation (9). The sample includes all men and women in the 2002 Uganda Census between the ages of 0 and 30 in 1990, the year in which an affirmative action policy granting women preferential treatment in their application to public Universities was enacted. The vertical lines represent the approximate cutoffs between primary secondary and University education. Standard errors are clustered at the district level. Filled in circles are coefficients significant at $\leq$10% level.

VII Tables

Table 1: HIV Prevalence in Uganda, 1987

Group	Description	% HIV+
α males	Never married males ≥ 20 and widowed divorced males	17%
β males	Never married males 15-19 and young ≤ 30 married males	10%
δ males	Older married males >30	6%
μ females	Commercial sex workers	37%
λ females	All never married females and young < 25 married females	19%
ω females	All widowed/divorced females and older ≥ 25 married females	9%

Notes: Data are from the 1987 KAP Survey. Prevalence is the percent of those in a given subgroup with a positive HIV-1 antibody test, except for Commercial Sex Workers, who are assumed to have the HIV prevalence of older, never married females.

Table 2: Characteristics of Sexual Activity, 1987

Characteristic	Females	Males
Age of sexual debut		
Mean	14.9	17.3
2.5%ile, 97.5%ile	14.0, 15.6	15.5, 18.8
Age of first marriage		
Mean	17.3	23.4
2.5%ile, 97.5%ile	16.2, 18.2	19.5, 29.3
Coital frequency for married individuals[#]		
Mean	6.3	7
2.5%ile, 97.5%ile	0, 25	0, 26
Number of extramarital partners[*]		
μ females / α males	NA	2.4
λ females / β males	1.1	2.1
ω females / δ males	0.6	1.1

Notes: Average age of sexual debut and first marriage are predicted using the coefficients in Online Appendix table A1. Coital frequency for women is predicted using the coefficients in Online Appendix table A2. Coital frequency for married men is derived by randomly matching each husband with their reported number of wives and ascribing the wives' sexual activity to their husband. See text for further details. Number of extra-marital partners is based upon responses in the 1987 KAP Survey.

[#] In the last four weeks.

[*] If unmarried, this is the average total number of partners.

Table 3: Estimated Mixing Probabilities in the Baseline Model

	Males			
Females	α	β	δ	Total
μ	NA / 30%	NA / 21%	NA / 14%	100% / …
λ	84% / 41%	14% / 37%	2% / 24%	100% / …
ω	17% / 30%	7% / 43%	76% / 62%	100% / …
Total	… / 100%	… / 100%	… / 100%	

Notes: The first item in each cell is the share of people in that row having sex with people in that column. The second item is the share of people in that column having sex with people in that row. The probabilities are derived from the model in Online Appendix B using the data sources described in Online Appendix C.

Table 4: Predicted and Actual Baseline Prevalence of HIV

Demographic group	Actual prevalence	Predicted prevalence	Percentage point difference	% difference
Women				
Total	13.1%	13.2%	-0.1%	0.4%
Never married	22.5%	20.9%	-1.6%	-7.3%
Young married	17.5%	19.1%	-1.6%	-9.0%
Old married	8.3%	9.0%	-0.7%	-7.9%
Divorced/widowed*	9.5%	7.5%	2.0%	21.4%
Men				
Total	10.6%	10.9%	-0.3%	-2.4%
Young never married	5.9%	5.2%	0.8%	12.7%
Old never married	13.7%	13.3%	0.0%	3.2%
Young married	10.9%	9.4%	-1.5%	-13.7%
Old married	6.0%	4.2%	-1.8%	-29.6%
Divorced widowed*	20.7%	23.1%	-2.4%	-11.5%

*Assumed married for 1/2 time sexually active

Notes: Starting with a 5% HIV prevalence among CSWs in 1977.

Table 5: Simulated Impact of Abstinence, Faithfulness, Condom Use, and Death on HIV Positive Rates Among Pregnant Women

	Pregnant Women of Age			
	15-19	20-24	25-29	30+
Panel A: Baseline Simulation				
Actual rate, 1990-1992	26.5%	26.8%	23.1%	15.7%
Predicted rate	25.7%	24.4%	24.6%	11.0%
% difference	3.0%	9.0%	6.5%	30.0%
Percentage point difference	0.8%	2.4%	-1.5%	4.7%
Change in rate: 1990-92 to 1994-96	-14.0%	-6.8%	-2.3%	0.2%
Panel B: Simulating Population Changes				
Reduction in Pre-Marital Sex (Women)				
Predicted rate	17.4%	23.1%	24.7%	11.0%
Percentage point change in predicted rate	-8.3%	-1.3%	0.1%	0.0%
Reduction in Extra-Marital Sex (Men)				
Predicted rate	26.6%	24.7%	24.5%	11.0%
Percentage point change in predicted rate	0.9%	0.3%	-0.1%	0.0%
Impact of Condom Use (Men)				
Predicted rate	22.4%	23.1%	24.5%	10.8%
Percentage point change in predicted rate	-3.3%	-1.3%	-0.1%	-0.2%
Impact of AIDS-Related Deaths (Men)				
Predicted rate	23.1%	22.2%	24.2%	10.7%
Percentage point change in predicted rate	-2.6%	-2.2%	-0.7%	-0.3%
Cumulative Impact	-13.3%	-4.5%	-0.8%	-0.5%
Share of total decline	95.0%	66.0%	35.0%	---

Notes: All estimates come from the model described in the text.

Table 6: Abstinence, Sexual Debut and Marital Trends, Young Ugandan Women, Urban Areas

	Ages 15 to 19		Ages 20 to 24	
	1988	1995	1988	1995
Married	18.7%	21.2%	45.1%	49.4%
Virgin | never married	50.3%	68.7%**	20.3%	21.2%
Age of first sex | having sex	14.9	14.8	15.9	16.1
- Married	15	15	15.6	16
- Never married	14.9	14.6	16.7	17
Age of first marriage | married	15.9	15.9	17.7	17.7

Notes: Data are from the 1988 and 1995 Uganda DHS. Percentages do not add to 100% because of omitted marital categories.

Table 7: Enrollment and Virginity

	(1)	(2)	(IV)
Enrolled	0.45***	0.46***	0.73***
	(0.022)	(0.022)	(0.214)
Birth year	0.06	-0.06***	0.05***
	(0.002)	(0.003)	(0.010)
Catholic		-0.007	0.002
		(0.012)	(0.015)
Baganda		-0.02***	-0.04*
		(0.016)	(0.024)
Know someone who died of AIDS		-0.03***	-0.04*
		(0.019)	(0.024)
County FE	N	Y	Y
First-stage *F*- stat			24.5
No. obs	2987	2940	2940
R^2	0.40	0.40	0.36

Notes : Data are from the 1995 Uganda DHS. Sample includes females ages 15-24 who did not self-report leaving school due to pregnancy. Robust standard errors in parentheses.

* Statistically significant at the 1% level

** Statistically significant at the 5% level.

*** Statistically significant at the 1% level.

Table 8: Effect of the Policy on Higher Education: Coefficients on the Interaction of Gender, Proximity to Public University and Cohort Dummies

	Secondary School Completion	Completed >7 Years of School
Panel A: Experiment Comparing Educational Outcomes for Girls aged 8-12 versus those aged 19-23 in 1990		
Female*Young*(1/Ln(Distance))	2.79***	3.42***
	(0.361)	(0.469)
District FE	Y	Y
Cohort FE	Y	Y
Cohort-Size	Y	Y
No. obs	338545	353782
No. clusters	55	55
R^2	0.11	0.12
Panel B: Placebo Comparing Educational Outcomes for Girls aged 19-23 versus those aged 24-28 in 1990		
Female*Young*(1/Ln(Distance))	0.521	0.568
	(0.646)	(0.513)
District FE	Y	Y
Cohort FE	Y	Y
Cohort-Size	Y	Y
No. obs	230502	245130
No. clusters	55	55
R^2	0.11	0.11

Notes: Data are from the 2002 Uganda Census. Robust standard errors clustered at the district level in parentheses.

* Statistically significant at the 1% level

** Statistically significant at the 5% level.

*** Statistically significant at the 1% level.

VIII For Online Publication: Appendices

Appendix A

Table A1: Predicted Age of First Marriage and Age of Sexual Debut

	Females		Males	
Independent Variable	Age of first marriage	Age of sexual debut	Age of first marriage	Age of sexual debut
Education	1.05***	0.74***	0.34	-0.64
	(0.21)	(0.17)	(0.34)	(0.59)
Age	0.036**	0.11**	0.19***	0.34**
	(0.01)	(0.05)	(0.04)	(0.16)
Age^2	---	-0.002**	---	-0.004
		(0.001)		(0.002)
Constant	14.90***	12.83***	15.34***	11.21***
	(0.40)	(0.74)	(1.07)	(3.24)
No. obs	1154	1287	423	568
R^2	0.05	0.05	0.1	0.03

Notes : For females, the data are from the 1988 Uganda DHS. For males, the data are from the 1995 Uganda DHS. Only men who married or debuted at least 10 years prior to the survey were included in the analysis. Robust standard errors in parentheses.

** Statistically significant at the 5% level.

*** Statistically significant at the 1% level.

Table A2: Predicted Coital Frequency

	Number of coital acts per month		
Independent Variable	Zero inflated negative binomial Married females	Negative binomial Never married females	Zero inflated negative binomial Divorced/widowed females
Age	0.001	0.574**	0.114
	(0.016)	(0.226)	(0.089)
Age^2	-0.0002	-0.013***	-0.002
	(0.0003)	(0.005)	(0.001)
Constant	2.060***	-6.089**	-0.29
	(0.225)	(2.494)	(1.318)
(inflate) Constant	-1.995***	---	0.711***
	(0.108)		(0.148)
Natural log alpha	-0.357***	1.677	-0.328
	(0.091)	(0.139)	(0.447)
Nonzero observations	2455	471	177
Vuong test	5.59	---	2.75

Notes : Data are from the 1988 Uganda DHS. Robust standard errors in parentheses. Depending on Vuong test results, a zero inflated negative binomial or a negative binomial model was used.

** Statistically significant at the 5% level.

*** Statistically significant at the 1% level.

Table A3: Transmission Probabilities

Male to Female				
Females	Husband	α	β	δ
Age < 20	0.0025	0.015	0.01	0.005
Age 20-29	0.0025	0.0075	0.005	0.0025
Age ≥30	0.00125	0.006	0.003	0.002
Female to Male				
Males	Wife	λ	μ	ω
Age ≤ 30	0.0016	0.0025	0.005	0.0016
Age >30	0.00125	0.0025	0.005	0.0016

Notes: Probabilities are based on the medical literature. See text for references.

Table A4: Condom Use Among Urban Men, 1995

	Males				
Females	Young, never married	Old, never married	Young, married	Old, married	Divorced/ widowed
Never married	50%	50%	34%	20%	61%
Young, married	50%	50%	7%	20%	61%
Old, married	50%	50%	34%	4%	61%
Divorced/widowed	50%	50%	34%	20%	61%

Notes: Data are from the 1995 Ugandan DHS.

Appendix B

This Appendix explains our mixing equations and the assumptions we use to identify those equations.

Denote π_{ij} as the probability that a women in group i has sex with a man in group j and π_{ji} as the probability that a man in group j has sex with a woman in group i. There are 9 probabilities for each of three groups of men having sex with three groups of women ($\pi_{\alpha\lambda}$, $\pi_{\alpha\omega}$, $\pi_{\alpha\mu}$; $\pi_{\beta\lambda}$, $\pi_{\beta\omega}$, $\pi_{\beta\mu}$; and $\pi_{\delta\lambda,}$ $\pi_{\delta\omega}$, $\pi_{\delta\mu}$) and 9 probabilities for each of three groups of women having sex with each of three groups of men ($\pi_{\lambda\alpha}$, $\pi_{\lambda\beta}$, $\pi_{\lambda\delta}$; $\pi_{\omega\alpha}$, $\pi_{\omega\beta}$, $\pi_{\omega\delta}$; and $\pi_{\mu\alpha}$, $\pi_{\mu\beta}$, $\pi_{\mu\delta}$). Since we do not observe CSWs in our data, we omit $\pi_{\mu\alpha}$, $\pi_{\mu\beta}$, $\pi_{\mu\delta}$ from consideration. This leaves 15 unknown parameters.

These parameters are identified through two types of equations. The first equations are the observation equations: every sexual act is with one of these groups. This corresponds to the assumptions that:

1. $\pi_{\lambda\alpha} + \pi_{\lambda\beta} + \pi_{\lambda\delta} = 1$
2. $\pi_{\omega\alpha} + \pi_{\omega\beta} + \pi_{\omega\delta} = 1$
3. $\pi_{\alpha\lambda} + \pi_{\alpha\omega} + \pi_{\alpha\mu} = 1$
4. $\pi_{\beta\lambda} + \pi_{\beta\omega} + \pi_{\beta\mu} = 1$
5. $\pi_{\delta\lambda} + \pi_{\delta\omega} + \pi_{\delta\mu} = 1$

The second equations are the conservation of sex acts: the total number of acts that group A has with group B is the same as the number of acts that group B has with group A. Again excluding CSWs, this yields the equations:

6. $\pi_{\lambda\alpha} N_\lambda = \pi_{\alpha\lambda} N_\alpha$
7. $\pi_{\lambda\beta}\ N_\lambda = \pi_{\beta\lambda} N_\beta$
8. $\pi_{\lambda\delta} N_\lambda = \pi_{\delta\lambda} N_\delta$
9. $\pi_{\omega\alpha} N_\omega = \pi_{\alpha\omega} N_\alpha$
10. $\pi_{\omega\beta} N_\omega = \pi_{\beta\omega} N_\beta$
11. $\pi_{\omega\delta} N_\omega = \pi_{\delta\omega} N_\delta$

Thus, there are 11 equations and 15 unknowns.

To solve the equations, we need an additional assumption. We do this with knowledge of sexually transmitted infections (STIs). Let a, b and d be the prevalence of sexually transmitted infections in the α, β, and δ groups of males, respectively; where $a > b > d$. In practice, we calculate $a = .50, b = .35$,and $d = .24$. We use these fractions to weight the coefficients and reduce the dimensionality of the matrix from 11 to 9 equations. Specifically, we assume that the probability that ω (older, safer) women have sex with δ (older) men is declining in the probability of STIs among older men, the probability that men have sex with ω women

is inversely proportional to STI rates, and that the probability that men have sex with μ women (CSWs) is directly proportional to STI rates. Thus:

$\pi_{\omega\delta} = (1-d)$
$\pi_{\omega\alpha} = (1-\pi_{\omega\delta})\left(\frac{b}{a}\right)$
$\pi_{\omega\beta} = (1-\pi_{\omega\delta})\left(1-\frac{b}{a}\right)$
$\pi_{\alpha\omega} = \left(\frac{d}{a}\right)(\pi_{\delta\omega})$
$\pi_{\beta\omega} = \left(\frac{d}{b}\right)(\pi_{\delta\omega})$
$\pi_{\beta\mu} = \left(\frac{b}{a}\right)(\pi_{\alpha\mu})$
$\pi_{\delta\mu} = \left(\frac{d}{a}\right)(\pi_{\alpha\mu})$

This translates into the following 9 equations, which can be used to solve for 8 unknowns: $\pi_{\lambda\alpha}$, $\pi_{\lambda\beta}$, $\pi_{\lambda\delta}$, $\pi_{\alpha\lambda}$, $\pi_{\alpha\mu}$, $\pi_{\beta\lambda}$, $\pi_{\delta\lambda}$, and $\pi_{\delta\omega}$.

1. $\pi_{\lambda\alpha} + \pi_{\lambda\beta} + \pi_{\lambda\delta} = 1$
2. $\pi_{\omega\alpha} + \pi_{\omega\beta} + \pi_{\omega\delta} = 1$
3. $\pi_{\alpha\delta} + \frac{d}{a}\pi_{\delta\omega} + \pi_{\alpha\mu} = 1$
4. $\pi_{\beta\lambda} + \frac{d}{b}\pi_{\delta\omega} + \frac{b}{a}\pi_{\alpha\mu} = 1$
5. $\pi_{\delta\lambda} + \pi_{\delta\omega} + \frac{d}{a}\pi_{\alpha\mu} = 1$
6. $\pi_{\lambda\alpha}N_\lambda = \pi_{\alpha\lambda}N_\alpha$
7. $\pi_{\lambda\beta}N_\lambda = \pi_{\beta\lambda}N_\beta$
8. $\pi_{\lambda\delta}N_\lambda = \pi_{\delta\lambda}N_\delta$
9. $N_\omega = \pi_{\delta\omega}\left[N_\delta + \frac{d}{a}N_\alpha + \frac{d}{b}N_\beta\right]$

Table B1: Estimated Mixing Probabilities Under Alternative Simulations

	Males		
Females	α	β	δ
Reduced Pre-marital Sex			
μ	NA / 39%	NA / 27%	NA / 19%
λ	83% / 31%	15% / 30%	2% / 19%
ω	17% / 30%	7% / 43%	76% / 62%
Reduced Extra-marital Sex			
μ	NA / 29%	NA / 20%	NA / 14%
λ	85% / 41%	14% / 37%	1% / 23%
ω	17% / 30%	7% / 43%	76% / 63%
Death of α males			
μ	NA / 12%	NA / 9%	NA / 6%
λ	83% / 51%	15% / 40%	2% / 20%
ω	17% / 36%	7% / 51%	76% / 75%

Notes: The first item in each cell is the share of people in that row having sex with people in that column. The second item is the share of people in that column having sex with people in that row. CSWs are not in our sample; thus, we do not estimate the probability that they have sexual contact with any particular set of men.

Appendix C

Condom Use: Sample includes men in urban areas, stratified by the groups described in table 1. The variable is constructed from a question regarding condom use at last sex (mv761) and a question on whether condom was used with wife (mv851) or with others (mv850a) to determine which acts were protected for married men (see Online Appendix A table A4) Source: 1995 Uganda DHS.

Distance to Public Universities: This was calculated using coordinates for Makerere University (0.35 , 32.68) and for Mbarara University of Science and Technology (-0.62 , 30.66). A map of Ugandan districts was projected into Africa_Equidistant_Conic. The centroid of each individual's birth district (BPLUG) was then used. Distance was calculated in meters using the Near tool in ArcMap 10, as the distance between the centroid of the respondent's birth district and the nearest public University. Source: 2002 Uganda Census.

Distance to Secondary School: This variable is defined as distance to secondary school and is measured in kilometers, (c111b). Source: 1995 Uganda DHS.

Enrolled: This variable is an indicator variable equal to 1 if the respondent answers yes to the question of whether they are still in school (v148). Source: 1995 Uganda DHS.

Fidelity: Sample includes men in urban areas who have ever been married and is defined specifically as an indicator equal to one for those who meet the joint condition of responding yes to the question on whether they have reduced their partners (mv760e) and now only have one partner (mv760d). Source: 1995 Uganda DHS.

Secondary School Attendance: This variable is an indicator equal to one if the individual completed secondary school. Specifically, EDATTAND must be equal to 221 or 311 in the data set, corresponding to completion of lower or upper secondary general school. Secondary technical track was excluded since this would not have been affected by the 1990 affirmative action policy (technical training is a substitute for University education). Source: 2002 Uganda Census.

Virginity: This variable equals zero if the respondent gives an age to the question on age of first intercourse and one if they have not yet had intercourse, (v525). Source: 1988 Uganda DHS and 1995 Uganda DHS.

Table C1: Model Overview and Data Sources

Parameter	Data Source/Methodology	Values
Pr {HIV transmission}	Biomedical literature (see text for references)	Table A3
Prevalence of STIs	1987 KAP Survey	α=.50, β=.35 and δ=.24
Pr {HIV_f } and Pr {HIV_m }	1987 KAP Survey	Table 1
N_i = duration* coital frequency		
Duration sexually active	1988 and 1995 Uganda DHS	Table A1
Coital frequency	1988 Uganda DHS	Table A2
Mixing probabilities (π_{fm})	Calculated from equations (3) and (4)	Table 3 and Appendix B
Behavior Change (A)	1988 and 1995 Uganda DHS	Tables 6 and B1
Behavior Change (B)	1995 Uganda DHS	Reduction in the number of extramarital coital acts lowers N_j changes $\pi_{mf.}$ Table B1.
Behavior Change (C)	1995 Uganda DHS	Table A4
Mortality (D)	Biomedical literature on time to developing AIDS. See text for references	Sensitivity analysis for death of α males which lowers Pr(HIV_α) and changes $\pi_{mf.}$ Table B1

Notes: Parameters given are those for estimating equation (5) for a representative female. The male equation (6) is symmetric except for the following: Prevalence of STIs in Females, μ=.50 λ=.15 and ω=.10 and coital frequency is found by pairing married males with married women, and, for single men, multiplying the ratio of acts for Tanzanian single men/single women by Ugandan coital acts for single women.