NBER WORKING PAPER SERIES

THE ABCDS OF HEALTH: EXPLAINING THE REDUCTION IN AIDS IN UGANDA

Marcella M. Alsan
David M. Cutler

Working Paper 16171
http://www.nber.org/papers/w16171

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
July 2010

We are grateful to David Bangsberg, Seth Berkley, Esther Duflo, Max Essex, Erica Field, Ron Gray, Howard Hiatt, Guido Imbens, Joseph Konde-Lule, Michael Kremer, Yuka Manabe, Frank Nalugoda, Thomas Quinn, David Serwadda, Maria Wawer, and staff at the Ugandan Ministry of Health Libraries for helpful comments. Seth Berkley and Joseph Konde-Lule graciously supplied the data for the paper. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

The ABCDs of Health: Explaining the Reduction in AIDS in Uganda
Marcella M. Alsan and David M. Cutler
NBER Working Paper No. 16171
July 2010
JEL No. I1,O55

ABSTRACT

Uganda was widely viewed as a public health success for curtailing its AIDS epidemic in the early 1990s. To understand the reasons for the dramatic decline, we build a simulation model of HIV transmission using newly discovered data on HIV status and sexual behavior from the relevant time period. We then model the impact of abstinence, fidelity, condom use, and selective mortality on the prevalence of HIV among various subgroups. Among young women, who experienced the greatest decline in HIV prevalence, the most important component was delaying sexual debut, accounting for 57 percent of the drop in HIV prevalence. Condom use by high risk males and to a lesser extent death (of older males) also played a significant role, accounting for 30 and 16 percent respectively. However, for older women, the trend is reversed, with death being more important than abstinence or condom usage. All told, we explain 86 percent of the reduction in AIDS in Uganda.

Marcella M. Alsan
Department of Economics
Harvard University
1875 Cambridge Street
Cambridge, MA 02138
malsan@fas.harvard.edu

David M. Cutler
Department of Economics
Harvard University
1875 Cambridge Street
Cambridge, MA 02138
and NBER
dcutler@harvard.edu

The AIDS epidemic has exacted a terrible price worldwide. About 33 million people across the globe are HIV positive, and the epidemic has claimed more than 25 million lives (UNAIDS, 2008). Africa bears a disproportionate burden of disease. Two-thirds of all those infected with HIV live in sub-Saharan Africa, as do 12 million AIDS orphans. AIDS remains the number one cause of death in Africa and the sixth leading cause of death worldwide (Piot et al., 2009).

Despite this discouraging prologue, there are two prominent outliers: Thailand and Uganda.[1] In each case, AIDS was recognized as a major public health problem and control was made a national priority. In Thailand, HIV prevalence among brothel-based sex workers was 15 percent in 1991 and was also growing rapidly among young Thai military conscripts. In Uganda, 33 percent of pregnant women around Kampala tested positive for HIV in 1990. In both countries, AIDS rates have declined markedly. HIV prevalence in Thailand never climbed above 2 percent nationwide and fell among the most affected groups. Uganda's HIV prevalence fell from 15 percent of the general population in 1990 to 5 percent in 2007 (UNAIDS/WHO, 2008).

The cause of Thailand's success has been studied (see Ainsworth et al., 2003). HIV in Thailand emerged later in the global epidemic and was "caught" when the virus was still mainly concentrated in high-risk populations. Thailand has a thriving sex industry and is a destination for sex tourism. In 1992, a national survey found that 22 percent of men aged 15-49 had visited a sex worker in the past year (Sittitrai et al., 1992). Epidemiologic evidence in the early 1990s demonstrated that most new HIV infections in Thailand were occurring through commercial sex. The Thai government took decisive action. The Ministry of Public Health implemented the "100

[1] Senegal also is considered a "success" story in the global HIV epidemic. The country's small and relatively homogenous Muslim population (who are usually circumcised) make the Senegalese example less readily exportable to the larger, Christian, Southern African countries that have been hardest hit by the epidemic.

percent condom campaign." The program required prostitutes to use condoms with every sex act. The policy was strictly enforced and brothels not distributing condoms to workers were shut down. The government also instituted a massive media campaign including mandatory AIDS prevention spots broadcast hourly on radio and TV.

The centralized nature of the epidemic makes the Thai example less relevant for much of the rest of Africa. For example, AIDS in Africa is believed to result from decentralized sexual contacts throughout the continent, rather than a single centralized 'market.' For many historical, economic and cultural reasons, governments are not generally effective at enforcing societal changes. All of this makes the decline in AIDS in Uganda even more surprising. Uganda is widely viewed as an AIDS success story, and a model for much of sub-Saharan Africa (Schoepf, 2003).

A number of theories have been put forward to explain the reduction in AIDS deaths in Uganda. One set of theories focuses on behavioral changes among Ugandans. The response of the Museveni government to the AIDS problem in the late 1980s was the ABC campaign: Abstinence, Be faithful, and use Condoms. Some argue that delayed sexual debut and reduced extramarital sex were the key to lower AIDS rates (Asiimwe-Okiror et al., 1997, Hallett et al., 2006, Stoneburner et al., 2004, Green et al., 2006). Others argue that increased condom use alongside poverty reduction and enhanced gender equality were more important factors (Fenton, 2004, Sindling, 2005). A less encouraging theory stresses high rates of AIDS mortality early in the crisis (Wawer et al., 2005).[2,3] If people with AIDS died without many sexual interactions,

[2] There is debate in the medical literature whether the virus reduces fecundity. However, Young (2005) has found that HIV reduces fertility through behavioral mechanisms: a reduction in the willingness of women to engage in unprotected sexual activity and a decreased demand for children. On the other hand, Juhn, Kalemli-Ozcan and Turgan (2008) find that community HIV prevalence has no effect on non-infected women's fertility. Fortson's (2009) study of HIV and fertility noted no difference in post 2000 and pre 1990 fertility rates.

[3] Others have a more nuanced view: it was pragmatic safe sex (such as less commercial and casual sex) that led to the decline in HIV prevalence and not absolute abstinence or monogamy (Okunozi and Epstein, 2005).

AIDS would naturally die out.[4] Understanding the importance of A, B, C, and D (death) in the AIDS pandemic is clearly vital in deciding which policies to promote in other countries in Africa and throughout the world.[5]

To understand the factors leading to reduced HIV rates, we build a simulation model of AIDS transmission. In our model, as is generally believed to be true in sub-Saharan Africa, AIDS spreads through heterosexual contact. The age sequencing of sexual acts is important. Young, unmarried women often have sex with older men. The sex is often gift or cash related. When women marry, their sexual activity outside of marriage ceases. Young men, generally with fewer resources, have relatively little sex. As they age, they marry – often to multiple wives – and have sexual relationships outside of marriage with younger women. The model suggests a natural sequence for sustaining high AIDS rates: older men acquire the disease and pass it along to young women, who then pass it on to the men they marry. These men, in turn, spread the disease to the next generation of young women. Cross-generational sex and concurrent partnerships thus fuel the epidemic (Morris et al., 1996).[6]

We calibrate the model using a number of sources. We rely most heavily on newly discovered data from the 1987 Knowledge, Attitudes and Practices (KAP) survey (Konde-Lule et al., 1987). 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 of Kampala (see figure 1). 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

[4] The molecular epidemiology of the HIV-1 virus in sub-Saharan Africa shows clade D to be most prevalent in Eastern Africa, particularly Uganda. Recent studies by Vasan et al. (2006) and Kiwanuka et al. (2008) demonstrate that clade D may lead to faster progression to AIDS and death than other subtypes.

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

[6] Concurrent partnerships help fuel the epidemic since the virus reaches very high levels in the bloodstream immediately after infection, so that one newly HIV positive individual can transmit to many of those in his or her sexual network efficiently.

detailed sexual histories were obtained. Blood samples were collected and tested for the presence of serum antibody to HIV. We also use survey responses from Uganda Demographic and Health Survey (DHS) 1988, Tanzania DHS 1991-2 and Uganda DHS 1995. The 1995 Uganda DHS does not include serological status. Thus we fit our baseline and behavioral change analyses with serological data from antenatal clinics in Uganda. The medical literature provides estimates of HIV transmission probabilities between discordant sexual partners (Boily et al., 2009, Pettifor et al., 2007, Quinn et al., 2000).

We use this combination of data to simulate who was having sex with whom, and how likely it was they would contract HIV. We show that our model predicts well the number of births to women of different ages, an important validation point.

We then examine the impact of delayed sexual debut, fewer out-of-marriage sexual acts, greater condom use, and selective mortality on the time pattern of HIV. Our results show the reason for the decline varied by age cohort. Among young women, who experienced the greatest decline in HIV prevalence, the most important component was delaying sexual debut; this accounted for 57 percent of the drop in HIV prevalence in this cohort. Condom use by high risk males and to a lesser extent death (of older males) also played a significant role, accounting for 30 and 16 percent respectively. However, for older women, the trend is reversed, with death being more important than abstinence or condom usage. All told, we explain 86 percent of the reduction in AIDS in Uganda. Delayed debut was the most important factor (41 percent) followed by condom use (26 percent) and death (20 percent). Interestingly, because increased faithfulness implies more high risk men were having sex with their spouses, this change actually led to a 1 percent increase in HIV prevalence among young women.

We explore briefly, but not in great detail, why young women choose to debut later. One possibility is that the high prevalence of HIV disease in Uganda incentivized individuals to change their behavior in ways that would curtail the spread of the virus. Tomas Philipson (2000) has dubbed this *prevalence elastic behavior* (see also Young, 2005 and 2007). Other possibilities include education of women, who then delay their sexual debut or perhaps change their subjective discount rate. Knowledge that HIV can be spread from mother to child may have also been a factor.

The paper is structured as follows. The first section discusses the AIDS epidemic in Uganda and compares it to other sub-Saharan African countries. The second section shows the dimensions of the AIDS epidemic and its decline in Uganda and trends in HIV positive status by demographic group. Section III describes the simulation model we build to understand AIDS rates and also presents our approach to parameterizing the model. We discuss validation results for the indicated parameters in the fourth section. The fifth section then examines the impact of social changes and mortality on AIDS prevalence over time. The sixth section discusses the possible factors leading to behavioral changes. The last section concludes.

I. HIV in Uganda

HIV has been present in Africa since at least 1960, and probably longer. The most widely accepted view regarding the origin of HIV is that a simian virus jumped from primate to human in Western Africa in the early 20th century (Sharp and Hahn, 2008). The first documented case of HIV is from the Republic of Congo in 1960.[7] The virus is then thought to have spread from Central to Eastern Africa through trade along the trans-African highway. By

[7] Fragments of a lymph node biopsy from Kinshasa preserved since 1960 were recently sequenced and found to contain HIV-1 genetic code (Worobey et al., 2008).

1986, one third of lorry drivers and the majority of sex workers along the trade route tested positive for HIV (Slutkin et al., 2006). Kampala, the capital of Uganda, is on the trans-African highway. In addition, Uganda's well-known social upheavals brought great opportunities for transactional sex. All of this made Uganda a country affected relatively early by HIV.

The most complete data on population-level HIV status comes from antenatal clinics (ANC, described below); most pregnant women attend such clinics, and many clinics have standard HIV screening.[8,9] The screened prevalence rates from antenatal clinics are typically sent to the Joint United Nations Programme on HIV/AIDS (UNAIDS), which compiles them within countries. Given that almost all sexually-active Ugandan women are pregnant with some regularity, the rates are likely fairly accurate for that group.

In Uganda, antenatal clinic testing is available for around 35 clinics, representing the areas of the Central region as well as the Eastern and Western Region (see figure 1). The Central region, which includes Kampala, has 27 percent of the country's population, followed by the Western (26 percent) and Eastern (25 percent) regions (Kiregyera et al., 2002).

Figure 2 shows HIV positive rates in Uganda between 1987 and 2005 among pregnant women. HIV status is checked at a number of clinics. 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 there over the entire time period (figure 3), or forming a chain index linking clinics in adjacent years. 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

[8] In the late 1980s in urban areas, the vast majority of Kampalan women attended an antenatal clinic at least once during their pregnancy (UDHS, 1988).

[9] Population-based epidemiological studies show that ANC data tend to be most accurate around urban areas, which is the focus of our study (Montana et al., 2008). According to the World Bank, ANC data overstated HIV prevalence compared to population-based surveys in every country except Uganda (World Bank, 2006).

then fell markedly. The cumulative reduction in HIV prevalence in urban areas of Uganda was approximately 20 percentage points, or about two-thirds of the pre-reduction peak.

There was a decline in HIV prevalence among rural women as well. About 13 percent of pregnant women living in rural areas were HIV positive in the late 1980s, and that fell to about 5 percent in the early 2000s, before rising again in 2003.

The trends in Uganda are in stark contrast to the trends in other sub-Saharan African countries (figure 4). Kenya, just east of Uganda, had an ANC-based HIV prevalence rate of about 16 percent from 1993 to 2001 and has only recently experienced a mild decline. Tanzania, a Southern neighbor of Uganda, likewise has not seen an appreciable decline.[10] The ANC rates of Southern African countries hardest hit by the epidemic (for example, Botswana and South Africa) continue to climb.

The Ugandan Response to HIV

Yoweri Museveni, a general who helped topple the dictatorships of both Milton Obote and Idi Amin, became president of Uganda in 1986. Museveni has recounted that he learned of his country's AIDS problem from Cuba's president, Fidel Castro. 'At the September 1986 meeting of Non-Aligned Heads of State, Castro pulled Museveni aside. "He ... told me that of the 60 soldiers we had sent to Cuba for training, 18 of them had the virus of AIDS,"' (Frontline, 2006). Indeed, HIV had already reached alarming rates in areas outside Kampala, as noted above.

Facing a national security threat, President Museveni vigorously promoted the now famous ABC program. He embarked on a nationwide tour educating the public about the virus

[10] The global prevalence of HIV infection appears to have plateaued in sub-Saharan Africa and elsewhere (UNAIDS, 2007).

and proclaimed avoiding AIDS was a patriotic duty (Parkhurst and Lush, 2004). The slogans "Love carefully" and "Zero grazing" featured prominently on radio and billboards. Condom promotion, however, lagged behind the abstinence and fidelity messages. Social marketing of condoms did not begin in earnest until the mid-1990s.[11]

Data on knowledge about HIV are presented in table 1, along with comparisons from Kenya. We use data from the 1995 Demographic and Health Survey in Uganda and the 1993, 1998, and 2003 surveys in Kenya. In the early 1990s, people in Uganda were more informed than people in Kenya, although not universally so. In 1995, 99 percent of Ugandans had heard of HIV/AIDS. Similarly, almost all Kenyans had heard of HIV/AIDS in 1993.

However, only 85 percent of men and 78 percent of women in Kenya thought there was a way to prevent infection, compared to 93 percent of Ugandan men and 89 percent of Ugandan women. Condom knowledge among men was similar in the two countries: about 30 percent of Ugandan and Kenyan men knew that condoms could be used to prevent HIV. However, there was a noticeable knowledge gap between Ugandan and Kenyan women. Many more people in Uganda cited abstinence as a method to prevent HIV than in Kenya. Thirty-five percent of Ugandan men and women cited abstinence as a way to prevent AIDS, compared to only 10 and 14 percent of adult Kenyans who endorsed this method.

Another striking difference between the two countries was in the personal experience of HIV/AIDS in the respondents' daily lives. About 90 percent of individuals interviewed in Uganda knew someone who was infected with or died from AIDS; in Kenya, the comparable

[11] According to Okware et al. (2005) the Ugandan government adopted in 1991 a policy of "quiet promotion and responsible use of condoms with appropriate education." Social marketing refers to the adaption of commercial marketing and sales techniques for the attainment of social goals (UNAIDS, 2001). Social marketing is thought to be useful for condom marketing since it allows people to receive messages about sexually transmitted illnesses without visiting health clinics.

figure was 40 percent. Ugandan women also had a greater perception of the risk of acquisition of HIV as compared to Kenyan women; 80 percent of Ugandan women responded they could catch AIDS compared to 46 percent of Kenyan women. Eventually, Kenyans did catch up in terms of HIV knowledge – from 1993 through 2003, increasing shares of Kenyans give responses similar to those in Uganda.

Although identical questions were not asked in Tanzania, some comparable data are in the 1992 DHS. In that year, over 90 percent of people interviewed had heard of AIDS. However, misconceptions were common. For example, most men thought condoms could be used more than once, and 24 percent of Tanzanian men thought that condoms caused AIDS. In terms of basic knowledge, then, the Ugandan response to AIDS certainly seemed to raise awareness of the issues.

II. Accounting for the HIV Decline

To presage our later analysis, we start by analyzing the dimension of the AIDS epidemic in Uganda, 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 1987 KAP survey was conducted in two counties outside of Kampala. The study was designed as a pre-intervention survey, prior to the introduction of an intensive education campaign by the newly formed National Committee for the Prevention of AIDS and National AIDS Control Program.[12] Only individuals 15 or older were included, representing roughly 55 percent of the population. The regions were divided into administrative zones with approximately 10 clusters of equal size. Initial homesteads for interview were identified with the

[12] A follow-up (post-intervention study) was never completed. A similar survey reportedly occurred at the national level; however, the data are currently missing.

help of local chiefs and twenty neighboring homesteads were surveyed in order to achieve a representative sample. No attempts were made to randomly select homesteads in a cluster. 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 using Enzyme Linked Immunosorbent Assays (ELISA; Wellcome Diagnostics, Dartford UK).[13]

Seroprevalence data from that survey are shown in figure 5. 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.

This difference suggests a division of the population into six groups, which we employ below: 3 groups of men and 3 groups of women. We label the male groups α, β, and δ (table 2). 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 prostitutes. The 1987 KAP survey shows this group has a HIV prevalence of 17 percent. 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 percent. δ males are the lowest risk group. These are older men with several wives. We find these men have the lowest observed HIV (6 percent) and reported sexually transmitted infections (STI) rate. It may be that

[13] It is standard in developed countries to confirm a positive ELISA with a Western Blot, a protein gel electrophoresis. The authors of the 1987 KAP survey note that financial considerations prevented them 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.

they have committed their finances to support their large families and therefore do not have the resources or desire to go outside their multiple marriages for sex.

Corresponding to the α males are the μ group of women, who represent prostitutes. Our sample does not contain self-described prostitutes, since most prostitution is in Kampala, which is not in our sample, and women are naturally hesitant about admitting they are prostitutes. Sex acts with prostitutes are calculated as the residual acts that remain after subtracting all coital acts attributed to women in the sample from those reported by men, as described below. We take the HIV prevalence among older never married women (37 percent) as the rate among prostitutes. 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 percent. ω women are older married women and women who are widowed or divorced. Such women rarely have high risk sex, and thus have relatively low HIV positive prevalence rates – 9 percent in our survey.

We do not have changes in HIV prevalence for these six groups over time, but we do have some data on age-specific changes for women from the ANC.[14] Figure 6 shows the trend in HIV positive rates by age in Uganda. The HIV rate fell most dramatically at younger ages, and was relatively unchanged among older women. For example, the HIV rate among 15 to 19 year-old women fell from 30 to 9 percent between 1990 and 1996, and the rate among 20 to 24 year-old women fell by 12 percentage points during the same time period.[15] In contrast, the rate was flat among women aged 30 and older. In our analysis, we thus concentrate heavily on explaining the trend for young women.

[14] Only some clinics report data by age. Thus, the mean rate across all age groups is not the same as the unconditional mean across all clinics, shown in Figure 2.

[15] The prevalence rate in the youngest sexually active cohort (e.g., 15-19 years of age) most accurately reflects incidence rates, since these infections are newly acquired.

III. A Model of HIV Transmission

To understand the decline in HIV positive prevalence in Uganda, we build a simulation model. We then use the model to understand the factors leading to the change in HIV positive status among pregnant women.

Several models of AIDS transmission have been developed in the literature, although none are ideal for our setting. One approach is based on 'compartmental models' common to epidemiology, such as the Susceptible-Infected (SI) model (May and Anderson, 1991). This is a set of differential equations which describes the evolution of the compartments over time. The key parameters are the probability of transmission, the contact rate between infectious and susceptible individuals, and demographic features of the population (i.e. birth rate, death rate, etc). Most of the literature operates on a macro level, with all individuals in a given subgroup acting identically.

Compartmental models have been used to project and to model how various interventions impact HIV prevalence (Hallett et al., 2006, Stoneburner and Low-Beer, 2004). In the case of sexually transmitted diseases, the specification of partnerships is crucial. Random mixing models assume that the chance a partner is selected from a different group is based solely on availability. Nonrandom mixing models are specified by the mixing probability π_{ij} – the probability a partner selected by a member of group i comes from group j (Kaplan, 1995). For example, Kremer and Morcom (1998) use a one-sex SI model with preferred mixing to prove the counterintuitive result that increasing sexual activity among low sexual activity people may lower the HIV prevalence.[16]

[16] The intuition for this result is as follows. Imagine the population is divided into high sexual activity-HIV positive, and no-activity HIV-negative individuals. By allowing those with no activity to mix with one HIV-positive partner (instead of only high-activity persons mixing with each other), the virus inhabits an inefficient (dead-end) vector.

SI models are limited in their ability to describe the HIV epidemic in Africa. For example, Leclerc and Garenne (2007) tried to use a compartmental model to fit the dynamics of the HIV epidemic in Zambia over time, and the age pattern of HIV prevalence from the 2001 DHS survey. Their model was standard and the parameters employed reasonable, yet the model predicted a decline of the epidemic, not an expansion. An explanation for the failure was that compartmental models tend to employ average parameters across the population instead of using individual-level data. This approach masks the distribution of sexual activity—which may be skewed with a long tail towards the right (Rothenberg et al., 2004).

In response to these critiques, microsimulation models such as STDSIM have been developed (Orroth et al., 2007). STDSIM is a dynamic stochastic simulation model which describes the mechanisms responsible for the transmission of five STIs, including HIV/AIDS. In this microsimulation model, the life histories of hypothetical individuals are simulated over time. Each individual is represented by a number of characteristics, of which some remain constant over time (i.e. gender) while others change (i.e. number of sexual partners). Changes in personal characteristics result from events which are stochastic: if and when an event occurs is determined by Monte-Carlo sampling from probability distributions. The outcomes for simulated individuals are aggregated to obtain outcomes for a simulated population. There are several modules in the simulation including transmission, natural history, health care, demography and sexual behavior. In the latter module, events such as age of sexual debut are drawn randomly from a uniform distribution (between 12-20 years of age).

Our microsimulation is similar to STDSIM in that we reconstruct the sexual histories at the individual level and aggregate to form population estimates. However, the richness of our

---

It's possible that if enough "no-activity" persons become low activity, the epidemic could die out in low prevalence settings.

dataset allows us to calculate and predict, as oppose to stochastically generate, events in an individual's lifetime. For example, age of debut and age of first marriage are not randomly drawn from the uniform distribution but predicted using conditional distributions. Furthermore, mixing probabilities are not obtained via sampling but are computed from a system of linear equations. Finally, we verify our model's predictions via robustness checks not commonly found in the mathematical epidemiology literature. STDSIM does have the advantage when it comes to modeling the impact of Herpes Simplex Virus 2, syphilis, etc., on HIV acquisition – but that is not the focus of our analysis.

Model Overview

The majority of HIV transmission in Uganda is from heterosexual intercourse. We thus model transmission through that route. An individual has a probability of contracting HIV with each unprotected coital act with a person who is HIV positive. That probability depends on three factors: the viral load of the HIV positive person, gender[17] and age.

Taking these factors into account, the probability that individual *j* contracts HIV during a given coital act with *i* can be expressed as:

$$\Pr\begin{bmatrix} j\ Contracts\ HIV \\ in\ one\ act\ with\ i \end{bmatrix} = \Pr\begin{pmatrix} Viral\ transmission \mid \\ Viral\ load, gender, age \end{pmatrix} \cdot \Pr\begin{pmatrix} Partner\ i \\ infected \end{pmatrix} \cdot \Pr\begin{pmatrix} No\ Effective \\ Condom\ Use \end{pmatrix} \quad (1)$$

where *i* is an element of the set {α, β, δ} for women and {λ, μ, ω} for men. After K acts distributed across *i* partners according to the mixing probabilities, the equation governing whether individual *j* has contracted HIV is:

[17] Receptive vaginal intercourse implies a larger surface area of viral exposure for women than men; thus, women acquire HIV from men more readily than the reverse (CDC, 2007).

$$\Pr[j\ Contracts\ HIV] = \ 1 - \prod_{k=1}^{K}\{1 - \Pr\left[\begin{matrix}Contract\ HIV \\ in\ act\ k\end{matrix}\right]\} \quad (2)$$

Because HIV positive prevalence rates were likely very low prior to the mid-1980s, we take the lifetime history to be the past 10 years. We thus construct individual sexual histories for up to 10 years prior to the 1987 survey.

Estimating equation (2) requires three types of data. The first is data on transmission probabilities. The second is information on who has sex with whom, and the HIV positive probability of those partners. The third is data on use of condoms. In 1987, condoms were essentially unused in Uganda (see discussion below). We thus assume there was no effective condom use in the baseline. We explain the other parts of our model.

A. HIV Transmission Rates

Viral transmission probabilities in the biomedical literature vary from study to study. One of the earliest reports on HIV transmission in the developing world was the Rakai study group, using data from Uganda (Gray et al., 2001). That study followed discordant monogamous couples over time until seroconversion occurred. The main result of this analysis was that viral load (number of copies of HIV virus per milliliter of blood, which is highest immediately following seroconversion and peaks again during end-stage AIDS) is the best predictor of transmission. Other significant factors were genital ulceration and age. Transmission probabilities were highest among those ages 25-29.

A recent meta-analysis pooled all the studies which reported empirical per-act heterosexual HIV-1 transmission probability estimates, or sufficient information to derive these estimates (Boily et al., 2009). The low-income pooled transmission estimates were above those in high-income countries and had larger confidence intervals. One possibility for the greater

heterogeneity among the low-income country study estimates was poor data quality and lack of concomitant information on co-factors of transmission such as circumcision, genital ulcerative disease and condom use. A survey of South African women ages 15-24 by Pettifor and colleagues (2007) calculated a per coital act male-to-female transmission probability between 0.02 and 0.06. These estimates were much higher than the Rakai group. Pettifor suggests this could be due to forcible sex, underreporting of contacts by women and underestimation of HIV prevalence among men. The transmission probabilities we use reflect the findings above (table 3). Specifically, we use asymmetric probabilities: higher for male-to-female transmission then female-to-male transmission and for younger individuals. Young women are particularly at risk for acquiring HIV through sexual acts (Karim et al., 2010). Cervical ectopy[18] (Moss et al., 1991, Myer et al., 2006), reproductive tract immaturity and the violent penetration associated with coerced sex are all considered supportive factors in successful male to female transmission (Foss et al., 2008, Ghanotakis et al., 2008).

B. Sexual Histories

The KAP survey has some data on the sexual history of people, but it is insufficient to estimate all of equation 2. Thus, we need to impute it. We perform the imputation in several stages. First, we estimate the age of first marriage and age of sexual debut. Women are assumed to have intercourse at marriage if not beforehand. We estimate these dates for women using the Uganda DHS in 1988.[19] For men, we use the DHS from 1995 including only those men who

[18] The cervix is composed of an ectocervical region open to the vaginal canal and an endocevical region which opens to the uterus. The ectocervix is covered in stratified squamous epithelium, which functions as a protective barrier against trauma and micro-organisms. The endocervix is a uni-layer of columnar cells designed for lubrication. Cervical ectopy is when endocervical columnar cells are present on the surface of the ectocervix resulting in the loss of an important barrier against microbial invasion.

[19] For age of sexual debut, we restrict the sample to central Uganda since age of debut was significantly earlier than in other regions of Uganda.

debuted and/or married in the relevant time period. This may introduce attrition bias into the analysis if the men who survived until 1995 debuted or married at much different ages than those who died. The DHS has information on education (categories include: primary, secondary and tertiary), and birth year, which we use to predict age of first marriage and age of sexual debut. Most of the married women respondents in 1987 KAP gave their wife rank and married men reported how many wives they had. For approximately 20 percent of respondents for whom this information was missing, we imputed wife rank/number using the coefficients from a zero truncated poisson regression of wife rank/number on age. The distribution for the total population (including imputed values) matched the actual values quite closely.

The first rows of Table 4 report the age at first marriage and the age of sexual debut. For women, the mean age at first marriage is 17 years, with a standard deviation of 0.58 years. For men, the age at first marriage is older, 23 years. The mean age of sexual debut is 15 for women and 17 for men. In both Kenya and Tanzania around the same period, the average age of sexual debut was 16 for women and the average age of first marriage was 18 years of age (Kenya DHS 1989, Tanzania DHS 1991).

Table 5 shows the regression results predicting age of first marriage and age of sexual debut. We used age and highest educational level as independent variables. There is abundant evidence suggesting educated women marry later (Jejeebhoy, 1995), which is true in our data as well. We use these coefficients to impute age at first marriage and age of sexual debut in the KAP, and in turn duration married, duration sexually active, and duration of premarital sex.

C. Mixing Probabilities

After knowing when people started having sex, we need to know who they have 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 the total number of sexual acts to perform this estimation. 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*. These parameters enter equation (1) in the partner probability terms.

The π_{ij}'s and π_{ji}'s 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. Denote the total number of coital acts for each group as N_i and N_j. The conservation of sex acts then implies:

$$\pi_{ij} N_i \;=\; \pi_{ji}\, N_j \qquad (3)$$

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 restriction that:

$$\sum_{j=1}^{3} \pi_{ij} = 1 \quad \text{and} \quad \sum_{i=1}^{3} \pi_{ji} = 1 \qquad (4)$$

Without further restrictions, the model cannot be solved, since there are not enough linearly independent equations. Specifically, there are 15 unknown mixing probabilities[20] and 11 equations.[21] In order to reduce the dimensionality of the matrix, we impose two restrictions, explained in more detail in the Appendix A. The first is that the rate of sexually transmitted infections among men in a given group is inversely proportional to the probability of mixing with older married females and directly proportional to the probability of mixing with prostitutes. The counterpart to the former assumption is that older women mix relatively less frequently with

[20] For each of the three groups of men, there are 3 groups of women they can have sex with, for a total of 9 probabilities. Similarly, there are 9 probabilities for women having sex with men. Since we do not observe prostitutes, this eliminates 3 of the probabilities, leaving 15 total probabilities.

[21] Five of the equations are the accounting equations (4) (omitting the equations for prostitutes), and the other 6 equations are the adding up constraints in equation (3) (again, omitting the prostitutes).

those groups of men reporting a higher rate of STIs. Indeed, older women have the lowest overall rate of STIs (10 percent for women 25 years of age or above, versus 15 percent for women younger than 25; whereas in men, the prevalence ranges from 24 to 50 percent).

D. Number of Coital Acts

Finally, we also need to determine the number of coital acts in which each person engages, the N_i's and N_j's. The KAP data do not have information on coital frequency, but the DHS has such data for women. Specifically, the DHS asks women how many times they have had sex in the last four weeks. Table 4 shows mean coital frequency for married individuals. Married men have on average more sex with their wives than wives do with their husbands because approximately 20 percent of men in the KAP data have more than one wife.

We use a negative binomial model to predict coital frequency for women. Table 6 shows the results. We use a quadratic form in age since older wives tended to have less sex than their younger counterparts. We then impute these data into the KAP. The average number of sex acts for never married women was only once per month.

The DHS 1988 does not have data on male coital frequency.[22] 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.

Extramarital activity is calculated by subtracting the number of reported partners in the last 6-12 months from number of reported wives or husband. For single men, we estimate coital frequency using the Tanzanian DHS 1991 since Uganda DHS 1988 does not include males. It would not be appropriate to assume that single men and single women pair together exclusively;

[22] Because coital frequency, unlike age of marriage or sexual debut, is not an event confined to the past—we cannot use the men's survey from DHS 1995 to estimate it.

as noted, many single women will have sex with married men. Therefore, 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 (stratified by age cohort) to obtain acts for single Ugandan men.

We assume that married men do not have sex as regularly with their extramarital partners as they do with their wives. Specifically, we assume that younger men are 5 percent as active with their extramarital partners as they are with their wives; whereas older men are only 2.5 percent as active with their extramarital partners as they are with their wives. We choose to make a distinction between older and younger married men since older married men likely have accumulated more wives and therefore have a lower demand for extramarital sex. We choose these percentages because they reflect the relative difference of reported coital frequency between married Tanzanian men and women around the same time period, after adjusting for polygamy.

E. Estimated Sexual Relations

The sexual relationship probabilities for the baseline model are in Table 7. 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 have prostitutes in our data, we do not estimate the distribution of sexual acts for prostitutes.

The λ, young women are indeed mixing a disproportionate amount of time (84 percent) 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 prostitutes, making them an excellent bridge population; transmitting the disease from sex workers to young women and turning the epidemic from a centralized to a generalized one. ω women spend three-quarters of their time having sex with older, lower risk, married males.

IV. Verifying the Model's Predictions

We verify the model's predictions in three ways. First, we use the data on reported number of children to see if the estimates of coital frequency are reasonably accurate. In particular, we were concerned young unmarried women may underreport their amount of sexual activity, which would affect not only estimated HIV prevalence in this cohort, but among all cohorts since mixing probabilities would be biased. However, we do not find systematic evidence of duplicity—the predicted and actual number of births is not statistically different. For never married females, 70 women out of 474 report having a child in the last year (15%), and we predict 66 births (14%; probability of conception from Wilcox et al., 1995). The paradoxical observation−young unmarried women have the highest HIV prevalent rates despite low coital frequency−is then explained by the fact that their partners were particularly high risk, which is exactly 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. For example, beta males have a reported STI rate of 35 percent and alpha males have a STI rate of 50 percent.

Second, we can use the model to estimate the predicted HIV positive rates directly, and compare them to the actual prevalence (based on serological blood tests from 1987). Predicted versus actual prevalence are reported in Table 8. The model is calibrated fairly well across most groups. Predicted rates generally average about the actual rate. Among women, for example, the predicted rate is 13.3 percent, compared to an actual rate of 13.1 percent. Among men, the predicted and actual rates are 10.6 and 10.7 percent.

The biggest difference between predicted and actual rates is for divorced or widowed females, for whom the model overestimates HIV prevalence by 19.7 percent. Overall, however, the results are very 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 9 shows the results. The first block of the table shows the actual and predicted HIV positive prevalence rates by age group. The predicted rate is very close to the actual rate for young women, within 0.07 percentage points, or 0.02 percent. The gap gets bigger for older women, where the predicted rate is 30 percent below the actual rate.

Unfortunately, the model was not refined enough to predict exactly who would contract HIV – though the aggregate prevalence statistics for the groups are very near to the observed prevalence rates, individual predictions are less exact. This is not surprising given that we do not know the HIV status of the partners at an individual level.

V. Explaining the Reduction in HIV

We now use our simulation model to explain the reduction in HIV prevalence over time. 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 delay of premarital sexual debut among girls and being reduced out-of-marriage sex among men), and death among people with AIDS.

Any change in behavior or population structure will have general equilibrium effects as well as partial equilibrium effects. Consider an increase in condom use, for example. If men start using condoms more, fewer young women will acquire HIV in cross-age sexual intercourse, and this would lower the prevalence of HIV in young women. In turn, fewer young women will pass on HIV to their husbands, which will lower the prevalence in married men. This will then further affect the prevalence rate among the next generation of young women. The long-run impact could thus be much greater than the short-run impact.

The change in HIV prevalence in Uganda was very rapid, generally from the late 1980s through the mid 1990s. Indeed, our age-based ANC data go from roughly 1990-92 to 1994-96. Such a setting is decidedly partial equilibrium. We therefore consider the direct effects of the various interventions, which are immediate, and not the indirect effects as a result of changing prevalence, which occurs over time. 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 the prevalence.

A second modeling issue is what to assume about overall changes in sexual behavior as some components of sexual activity change. 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 prostitutes more, or married men might have more sex within marriage. The basic question is whether sexual activity for men is driven by relatively inelastic demand for sex (in which case total acts might remain constant, or decline only as other paths for sexual activity have a higher price) or by a more elastic supply of consenting partners (in which case, the number of sexual acts might change significantly).

Some literature suggests that the demand-side model is more relevant than the supply-side model, or equivalently that total number of sexual acts among an unaffected group will remain constant as one group of potential partners changes their behavior. For example, Dupas (2006) shows that an intervention which encouraged young girls to have less sex with married men 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, sociological research from Kenya by Luke (2001) has demonstrated 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. An anthropological surgery from Nigeria in the late 1980s inquiring about male sexuality among 1,749 men and 1,976 women found that over half of all men and one third of all women believed one woman was unable to satisfy a man's sexual needs; and that a man's nature entitles him to extramarital relations. One eighth of all wives in the survey responded that men were justified in seeking alternative sexual partners during the post-partum period of abstinence (Orubuloye et al., 1997).

We thus assume that the total number of coital acts for men does not change when young girls delay debut, and equivalently that the total number of sexual acts among women does not change when men have less sex outside of marriage.

A. Delayed sexual debut

We start by considering the impact of delayed sexual debut among young women. A delayed age of debut lowers the number of coital acts attributed to young women and thus changes who men have sex with as well.

The amount of time women had premarital sex declined in Uganda over the relevant time period. In the 1988 DHS, 11.5 percent of Ugandan women reported never having had sex. In 1995, the share was about the same, 10.5 percent. However, the length of time women had been sexually active before marriage fell. Among 15-19 year old women, the duration of premarital sexual activity fell from nearly 2 years to 1 year. The decline was somewhat smaller among women aged 20-24 and actually increased among women aged 25-29, reflecting the fact that their premarital years predated the epidemic. We see evidence of this in pregnancy trends. In the Central region of Uganda (where Kampala is located), the pregnancy rate to 15-19 year old women was essentially unchanged, but the share of pregnancies among never married women fell from 29 percent in 1989 to 12 percent in 1995 (figure 7). Figure 8 shows that the percentage of young, never married women who waited to have sex until marriage increased by 12 percentage points in Uganda between 1988 and 1995. Uganda's nearest neighbors; however, did not experience such a dramatic shift (yet, had relatively less sex before marriage than Ugandans in 1988). Similarly, the duration of premarital sexual activity of young women in Uganda decreased when compared to their neighbors (figure 9).

Delayed sexual activity reduces the number of sex acts that women in this age group have and changes who they have it with. Appendix B shows the change in sexual mixing rates implied by delayed sexual initiation. Relative to the base case in table 7, 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 prostitutes, though we do not model HIV positive rates among men.

Table 9 shows the impact of delayed sexual initiation on predicted HIV rates. Delayed sexual activity predicts an 8.0 percentage point reduction in HIV positive rates among teenage girls, roughly 57 percent of the observed decline. The decline is 1.8 percentage points among women aged 20-24, or 26 percent of the observed change. Reflecting the longer length of time prior to marriage, the impact of abstinence is negligible women aged 25 and older.

B. 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. In the UDHS 1995, men were asked whether they had changed their sexual practices to prevent AIDS and, if so, how. Only 11 percent of married men admitted to becoming monogamous,[23] and 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). Appendix B shows the change in the mixing probabilities. Given the small change in faithfulness reported in the survey, the impact on mixing probabilities

[23] Some men are polygamous. Even excluding those, however, the share rises to only 12.5 percent. Using this percentage does not change the results.

is small as well. And this translates into a small change in HIV positive rates. Interestingly, since many men are HIV positive, increased faithfulness actually increases slightly the share of women we observe who are HIV positive. Table 9 shows that HIV positive rates increase by about 0.1 percentage points as a result of this change.

C. Increased Condom Use

Figure 10 shows the number of condoms sold in Uganda in the 1990s. Condoms were introduced into Uganda in 1991, and even then remained scarce until 1997. The men's DHS survey in 1995 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.

Table 10 shows the responses for condom use. About one-third of unmarried men in 1995 report using a condom in their last sexual encounter. The rate is lower among married men, especially with their spouse and higher among older, never married men. Interestingly, condom use in 1995 was not much higher in Uganda than in Tanzania or Kenya (figure 11).

The fact that condoms were used most prominently among highest risk individuals means that the impact will be greater than the average rate of condom use would suggest. We assume condom use in extramarital sex acts exhibited linear growth from zero in 1991 to 40 percent in 1995. We also assume that condoms were used correctly.

Using these assumptions and correcting for the impact of condom use on fertility, we derive ANC-based HIV rates with the increase in condom use and compare those to the observed outcomes in the mean of the three years around 1995. The results are reported in Table 9.

Among women aged 15-19, increased condom use explains a 4.2 percentage point reduction in predicted HIV positive rates. This is about one-third of the actual 14 percentage point reduction in rates between 1990-92 and 1994-96. Thus, greater condom use explains slightly less than one third of the overall predicted decline in HIV prevalence among the youngest cohort of pregnant women.

Condom use becomes less important for explaining the pattern of HIV as women age. For women ages 20-24 and 25-29, condom use accounts for 20 and 13 percent of the total reduction in HIV prevalence, respectively. For older women, exposure to HIV positive men mainly occurs in the marriage, a context where condom use was not widely acceptable.

D. Death of HIV Positive Men

Those that died predominately over this time period were primarily the high risk males, either through combat, aging or AIDS. Since older men were infected early by prostitutes, they would be the first ones in the generalized epidemic to succumb. Using sibling reports of mortality from UDHS 1995, the crude death rate (CDR) for men over the age of thirty in Uganda jumped from 12 to 17 per 1000 between the years 1989 and 1992; whereas the CDR for women over age 25 stayed constant at 7 per 1000. In addition, studies on the natural history of HIV have shown that age is an important determinant of the survival: the older one is, the shorter the incubation period and faster the time to death (Coutinho, 2000).[24]

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

[24] The CASCADE (2000) study brought together data from 38 studies in Australia, North America and Europe and included data on 13,030 HIV -1 infected individuals whose date of conversion could be reliably estimated. Median survival time ranged from 12.5 years for those who serconverted in the age group 15-24 to 7.9 years for those who seroconverted in the age group 45-54.

(defined as a CD4 cell count <200 cells/cubic millimeter).[25] According to early studies of the natural progression of the disease in the developed world, approximately 30 percent of individuals with such profound lymphopenia would die within a year (Post et al., 1996). However, time from seroconversion to full blown AIDS may be faster in the developing world due to lack of treatment for opportunistic infections and the endemicity of other infectious diseases (such as diarrheal diseases, malaria and TB). A study of the natural history of HIV-1 infection in Thai military recruits found that approximately 15 percent of HIV positive Thai recruits died within five years of diagnosis and 30 percent had died by 6 years (Rangsin et al. 2004). Since we do not know the exact date of seroconversion for each member of the α cohort, we experimented with several possible values. Specifically, we modeled death as a 15, 22.5 or 30 percent mortality rate among alpha males over a horizon of 5 to 10 years. Each rate directly impacts the prevalence of HIV in the alpha group and necessitates a recalculation of sexual mixing probabilities. The best fit appeared to be a 15 percent mortality rate.[26]

As men with AIDS die, there are fewer partners for young women to have sex with. As above, we assume that total sexual acts for women remain the same. Implicitly, this means that young women have more sex with younger men, who are less likely to have AIDS.

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,

[25] We chose not to model AIDS-related deaths as the excess adult mortality in countries heavily impacted by HIV/AIDS versus those not heavily affected, as in Oster (2008). This is due to the possibility that the AIDS crisis may impact non-AIDS related health services. Case and Paxson (2009) use multiple waves of the DHS in fourteen sub-Saharan countries between 1988-2005 and find that regions of countries that have high HIV prevalence show the greatest erosion of health services. Grépin (2009) also found a negative association between a country's receipt of funding for HIV/AIDS and measures of quality of health care.

[26] If the decline is 30 or 22.5 percent, we overpredict the drop in HIV prevalence among young women by a significant amount; using 15 percent we predict 102 percent of the overall decline among 15-19 year old women, the subgroup with the most dramatic decline and 86 percent of the reduction overall.

although the magnitude is not enormous. The direct effect of fewer older being HIV positive is the greater impact.

Table 9 shows that death was an important component to the HIV decline. The selective death of the bridge population between prostitutes and young women helped to break the transmission of HIV and explained approximately 16 percent of the predicted decline among women ages 15-19. Unlike condom use, death explains a larger share of the (albeit modest) decline in HIV prevalence as women age.

Summary

All told, we estimate that we can explain about 86 percent of the reduction in HIV positive prevalence. Delayed sexual debut, increased condom use, and death were all contributing factors. The most important of the three, accounting for 41 percent of the total decline was primary abstinence among young Ugandan women. Death and condom use contributed 20 and 26 percent, respectively. Faithfulness actually increased the HIV prevalence among women by 1 percent, due to high prevalence men having more sex with their wives.

Importantly, the cause of the decline varied by age group. The behavior of younger women appears to be more *prevalence elastic*, perhaps because many of them were unmarried and therefore had greater choice in whether to have sex or use a condom. On the other hand, older women (almost all of whom were married) exhibited little preventive behavioral changes; their protection from HIV came almost exclusively from the mortality of high-risk men or the choice on the part of men to use condoms. These women may have appeared inelastic in their behavior due to societal norms, lack of education or the desire for children.

VI. Why Did Women Delay Their Sexual Debut?

The fundamental question that arises from our analysis is why women delayed their sexual debut – and to a lesser extent why condom use increased so much. It may be that men demanded less sex; thus young women could find no willing partners when attempting to lose their virginity or obtain funds for schooling or other needs. Alternatively, it may be that the supply of sex by young women was elastic to the ABC campaign.

The natural test of the demand and supply theories is to determine whether women's compensation for extramarital sex increased or decreased over the relevant time period. If demand for sexual contact among men fell, the compensation for sex with young women should fall as well. If supply contracted, in contrast, prices should rise. No data systematically examines gift exchange for sexual contacts among young women. But scattered evidence from anthropological data suggests that compensation did increase over the 1990s: from 0.4 to 4 USD in 1995 (Pickering et al., 1997) to 1 to 11 USD in 1999 (Gysels et al., 2002). Whether this is due to reduced supply or to a higher risk premium for casual sex as knowledge of AIDS expanded is not known.

Other studies also show that the sexual decisions of young women are responsive to income. For example, Dupas (2010) shows that the age of sexual partners of young women is responsive to information about HIV. Similarly, a recent paper by Barid et al. (2009) shows that providing cash to young women to stay in school curtailed their sexual activity. We thus suspect, although cannot prove definitively, that reduced sexual activity among teenage girls was a result of reduced supply of willing female partners.

VI. Conclusion

The decline in HIV in Uganda is concentrated among young women. Among 15-19 year old pregnant women, for example, HIV positive rates fell from 31 percent in the early 1990s to 14 percent in the mid 1990s.

Our results suggest three reasons for this decline. First, abstinence, as proxied by delayed sexual debut, is a major factor behind the decline, explaining about 41 percent of the total. Condom use among selective sex acts surprisingly also played a major role, as did the death of the first wave of older infected men, who were presumably replaced by less HIV positive and more condom-friendly counterparts. As a whole, these three explanations account for 102 percent of the reduction in HIV among young women, though a considerably smaller share of the reduction among slightly older women. There is still much to be explained, and the data we have on men is scant.

It would be useful to extend our analysis to the later 1990s to explain the second phase (albeit much less dramatic) of Uganda's HIV decline. Preliminary analyses suggests that there may have been a backslide in terms of abstinence and fidelity; however, this may have been tempered by the increasing availability of condoms and the decrease in transmission due to viral suppression from the national expansion of antiretroviral therapy.

References

Ainsworth, Martha, Chris Beyrer, and Agnes Soucat, "AIDS and public policy: the lessons and challenges of 'success' in Thailand," *Health Policy*, 64 (2003), 13-37.

Asiimwe-Okiror, Godwil, Alex Oprio, Joshua Musinguzi et al., "Change in sexual behavior and decline in HIV infection among young pregnant women in urban Uganda," *AIDS*, 11 (1997), 1757-1763.

Baird, Sarah, Ephraim Chirwa, Craig McIntosh et al., "The Short-Term Impacts of a Schooling Conditional Cash Transfer Program on the Sexual Behavior of Young Women," World Bank Policy Research Working Paper 5089 October 2009.

Boily, Marie-Claude, Rebecca Baggaley, Lei Wang et al., "Heterosexual risk of HIV-1 infection per sexual act: systematic review and meta-analysis of observational studies," *The Lancet Infectious Diseases*, 9 (2009) 118-129.

Case, Anne, and Christina Paxson, "The Impact of the AIDS Pandemic on Health Services in Africa: Evidence from Demographic and Health Surveys," NBER Working Paper No. 15000 (2009).

Collaborative Group on AIDS Incubation and HIV Survival including the CASCADE EU Concerted Action, "Time from HIV-1 seroconversion to AIDS and death before widespread use of highly-active antiretroviral therapy: a collaborative re-analysis," *The Lancet*, 355 (2000), 1131-1137.

Centers for Disease Control and Prevention, (2007). Downloaded from http://www.cdc.gov/hiv/topics/women/overview_partner.htm on 6/30/09.

Coutinho, Roel, "Some aspects of the natural history of HIV infection," *Tropical Medicine and International Health,* 5 (2000) A22-A25.

Dupas, Pascaline, "Do Teenagers Respond to HIV Risk Information? Evidence from a Field Experiment in Kenya," *American Economic Journal: Applied Economics*, forthcoming. Downloaded from http://www.econ.ucla.edu/pdupas/HIV_teenagers.pdf on 5/09/10.

Fenton, Lynda, "Preventing HIV/AIDS through poverty reduction: the only sustainable solution?" *The Lancet*, 364 (2004), 1186-1187.

Fortson, Jane, "HIV/AIDS and fertility," *American Economic Journal: Applied Economics*, 1; (2009) 170-94

Foss, Anna, Rachel von Simson, Cathy Zimmerman et al., "HIV/AIDS and rape: modeling predictions of the increase in individual risk of contracting HIV from forced sex in South Africa." In: *Program and abstracts of the XVII International AIDS Conference*, (2008); Mexico City, Mexico, Abstract TUPE0650.

FRONTLINE, *The Age of AIDS* ©PBS (2006). Downloaded from http://www.pbs.org/wgbh/pages/frontline/aids/ on 06/27/09.

Gray, Ronald, Maria Wawer, Ron Brookmeyer et al., "Probability of HIV-1 transmission per coital act in monogamous, heterosexual, HIV-1-discordant couples in Rakai, Uganda," *The Lancet*, 357 (2001), 1149–53.

Green, Edward, "Uganda's HIV prevention success: the role of sexual behavior change and the national response," *AIDS Behavior*, 10 (2006), 335-346.

Ghanotakis, Elena, Susannah Mayhew, and Charlotte Watts, "Tackling HIV and gender-based violence in South Africa: organisational responses to PEPFAR", In: *Program and abstracts of the XVII International AIDS Conference*, (2008); Mexico City, Mexico, Abstract THAE0404.

Grépin, Karen, "Too much of a good thing? The effects of HIV/AIDS funding on African health systems," Harvard University, manuscript (2009).

Gysels, Marjolein, Robert Pool and Betty Nnalusiba," Women who sell sex in a Uganda trading town: life history, survival strategies and risk," *Social Science & Medicine*, 54 (2002), 179-192.

Hallett, Timothy, John Aberle-Grasse, George Bello et al., "Declines in HIV prevalence can be associated with changing sexual behavior in Uganda, urban Kenya, Zimbabwe and urban Haiti," *Sexually Transmitted Infections*, 82 supp (2006), i1:i18.

Jejeebhoy, Shireen, "Women's education, autonomy and reproductive behavior, evidence from developing countries," (Oxford University Press, USA: NY, 1995).

Juhn, Chinhui, Sebnem Kalemli-Ozcan and Belgi Turan, "HIV and Fertility in Africa: First Evidence from Population Based Surveys," NBER Working Paper No. 14248 (2008).

Kaplan, Edward, "Model Based Representations of Human Sexual Behavior," in *Sexual Nature Sexual Culture*. Paul Abramson and Steven Pinkerton, eds. (University of Chicago Press, IL: Chicago, 1995).

Karim, Quarraisha, Sengeziwe Sibeko and Cheryl Baxter, "Preventing HIV in Women: A Global Health Imperative" *Clinical Infectious Diseases*, 50 supp (2010), s122-s129.

Kiregyera, Ben, "2002 Uganda Population and Housing Census," Downloaded from http://www.ubos.org/onlinefiles/uploads/ubos/pdf%20documents/2002%20Census%20Final%20Reportdoc.pdf on 6/26/09.

Kiwanuka, Noah, Oliver Laeyendecker, Merlin Robb, et al., "Effect of human immunodeficiency virus Type 1 (HIV-1) subtype on disease progression in persons from Rakai, Uganda, with incident HIV-1 infection," *Journal of Infectious Diseases*, 197 (2008), 638-639.

Kremer, Michael and Charles Morcom, "The effect of changing sexual activity on HIV prevalence," *Mathematical Biosciences,* 151 (1998), 99-122.

Konde-Lule, Joseph , Seth Berkley and Robert Downing, "Knowledge, attitudes and practices concerning AIDS in Ugandans," *AIDS,* 3 (1987), 513-518.

Leclerc, Pauline, and Michel Garenne, "Inconsistencies in age profiles of HIV prevalence: A dynamic model applied to Zambia," *Demographic Research*, 16 (2007), 121–140.

Luke, Nancy. "Confronting the 'Sugar Daddy' Stereotype: Age and Economic Asymmetries and Risky Sexual Behavior in Urban Kenya," *International Family Planning Perspectives*, 31 (2001), 6-14.

Macro International, Demographic and Health Surveys. Uganda 1988, 1995, 2000/1, Tanzania 1991, 1996, 1999 and Kenya 1989, 1993, 1998. Downloaded from http://www.measuredhs.com/aboutsurveys/dhs/start.cfm on 06/30/09.

May, Robert Lewis, and Roy Anderson, "Infectious diseases of humans: dynamics and control," (Oxford University Press, UK: Oxford, 1991).

Montana, Livia, Vinod Mishra, and Rathavuth Hong, "Comparison of HIV prevalence estimates from antenatal care surveillance and population-based surveys in sub-Saharan Africa," *Sexually Transmitted Infections*, 84 (2008), i78-i84.

Morris, Martina, David Serwadda, Mirjam Kretzschmar et al.,"Concurrent partnerships and HIV transmission in Uganda," In *Program and abstracts of the XI International AIDS Conference*, (1996); Vancouver, Canada. Abstract Tu.D.473.

Moss, Gerald, David Clemston, Lourdes D'Costa et al., "Association of cervical ectopy with Heterosexual Transmission of Human Immunodeficiency Virus: Results of a Study of Couples in Nairobi, Kenya," *Journal of Infectious Diseases,* 164 (1991), 588-591.

Myer, Landon, Thomas Wright, Lynette Denny et al., "Nested case-control study of cervical mucosal lesions, ectopy, and incident HIV infection among women in Cape Town, South Africa," *Sexually Transmitted Diseases*, 11 (2006), 683-687.

Okunozi, Sam, and Helen Epstein, "Pragmatic safe sex, not abstinence or faithfulness, was key in Uganda's decline," *Health Policy and Development*, 3 (2005), ii-iii.

Okware, Samuel, John Kinsman, Saul Onyango, et al., "Revisiting the ABC strategy: HIV prevention in Uganda in the era of antiretroviral therapy," *Postgraduate Medical Journal*, 81 (2005) 625-628.

Orroth, Kate, Esther Freeman, Roel Bakker et al., "Understanding the differences between contrasting HIV epidemics in east and west Africa: results from a simulation model of the Four Cities Study," *Sexually Transmitted Infections,* 83 (2007), i5–i16.

Orubuloye, I.O, John Caldwell and Pat Caldwell, "Perceived Sexual Needs and Male Sexual Behavior in Southwest Nigeria," *Social Science & Medicine*, 44(1997), 1195-1207.

Oster, Emily, "Estimating HIV Prevalence from Mortality Data," Downloaded from http://faculty.chicagobooth.edu/emily.oster/papers/hivestimates.pdf on 12/07/09.

Parkhurst, Justin, and Louisiana Lush, "The political environment of HIV: lessons from a comparison of Uganda and South Africa," *Social Science & Medicine*, 59 (2004), 1913-1924.

Pettifor, Audrey, Michael Judgens, Brooke Levandowski et al., "Highly efficient HIV transmission to young women in South Africa," *AIDS*, 21 (2007), 861–865.

Philipson, Tomas, "Economic epidemiology and infectious diseases," In *Handbook of Health Economics*, A. J. Culyer & J. P. Newhouse eds. (Elsevier, London, 2000).

Pickering, H., M. Okongo, B. Nnalusiba et al., "Sexual networks in Uganda: Casual and Commercial Sex in a Trading Town," *AIDS Care*, 9 (1997) 199-207.

Piot, Peter, Michael Kazatchkine, Mark Dybul et al., "AIDS: lessons learnt and myths dispelled," *The Lancet*, 374 (2009), 260-263.

Post, Frank, Robin Wood, and Gary Maartens, "CD4 and total lymphocyte counts as predictors of HIV disease progression," *Quarterly Journal of Medicine*, 89 (1996), 505-508.

Quinn, Thomas, Maria Wawer, Nelson Sewankambo et al., "Viral load and heterosexual transmission of human immunodeficiency virus type 1," *New England Journal of Medicine*, 342 (2000), 921-929.

Rangsin, Ram, Joseph Chiu, Chirasak Khamboonruang et al., "The natural history of HIV-1 infection in young Thai men after seroconversion," *Journal of the Acquired Immune Deficiency Syndrome,* 36 (2004), 622-629.

Rothenberg, Richard, David Gisselquist, and John Potterat, "A simulation to assess the conditions required for high level heterosexual transmission of HIV in Africa," *International Journal of STD and AIDS*, 15 (2004), 529-532.

Schoepf, Brooke G., "Uganda: Lessons for AIDS control in Africa," *Review of African Political Economy*, 30 (2003), 553-572.

Sharp, Paul M., and Beatrice Hahn, "Prehistory of HIV-1," *Nature*, 455 (2008), 605-606.

Sindling, Steven, "Does 'CNN' (Condoms, Needles and Negotiation) work better than 'ABC' (Abstinence, Being Faithful and Condom Use) in attacking the AIDS epidemic?" *International Family Planning Perspectives*, 31 (2005), 38-40.

Sittitrai, Werasit, Praphan Phanuphak, Jean Barry et al., "Thai sexual behavior and risk of HIV infection: A report of the 1990 survey of partner relations and risk of HIV infection in Thailand," Program on AIDS, Thai Red Cross Society and Institute of Population Studies, Chulalongkorn University (1992), Bangkok.

Slutkin, Gary, Sam Okware, Warren Naamara, et al., "How Uganda reversed its HIV epidemic," *AIDS Behavior*, 10 (2006), 351-356.

Stoneburner, Rand and Daniel Low-Beer, "Population-level HIV declines and behavioral risk avoidance in Uganda," *Science*, 304 (2004), 714-718.

UNAIDS, *AIDS Epidemic Update* (Geneva, UNAIDS, 2007).

UNAIDS, *Report on the global HIV epidemic* (Geneva, UNAIDS, 2008).

UNAIDS, *Condom social marketing: selected case studies.* (Geneva, UNAIDS, 2001).

UNAIDS/WHO, *Epidemiological Fact Sheet on HIV/AIDS Updates for Botswana, Kenya, Tanzania, South Africa, Thailand and Uganda* (Geneva, UNAIDS, 2004, 2006 & 2008).

United States Census Bureau. HIV/AIDS Surveillance Database. Downloaded from http://www.census.gov/ipc/www/hiv/ on 6/18/09.

USAID, *What happened in Uganda* (Washington, DC, USAID, 2002) Downloaded from http://www.usaid.gov/our_work/global_health/aids/Countries/africa/uganda_report.pdf on 06/27/09.

Vasan, Ashwin, Boris Renjifo, Ellen Hertzmark, et al., "Different rates of disease progression of HIV type 1 infection in Tanzania based on infecting subtype," *Clinical Infectious Diseases*, 42 (2006), 843-852.

Wawer, Maria, Ron Gray, David Serwadda, et al., "Declines in HIV Prevalence in Uganda: Not as Simple as ABC," In *12th Conference on Retroviruses and Opportunistic Infections*, (2005); Boston, USA, Abstract #27LB.

Wilcox, Amy, Clarice Weinberg, and Donna Bard, "Timing of sexual intercourse in relation to ovulation—effects on the probability of conception, survival of the pregnancy, and sex of the baby," *The New England Journal of Medicine*, 333 (1995), 1517-1521.

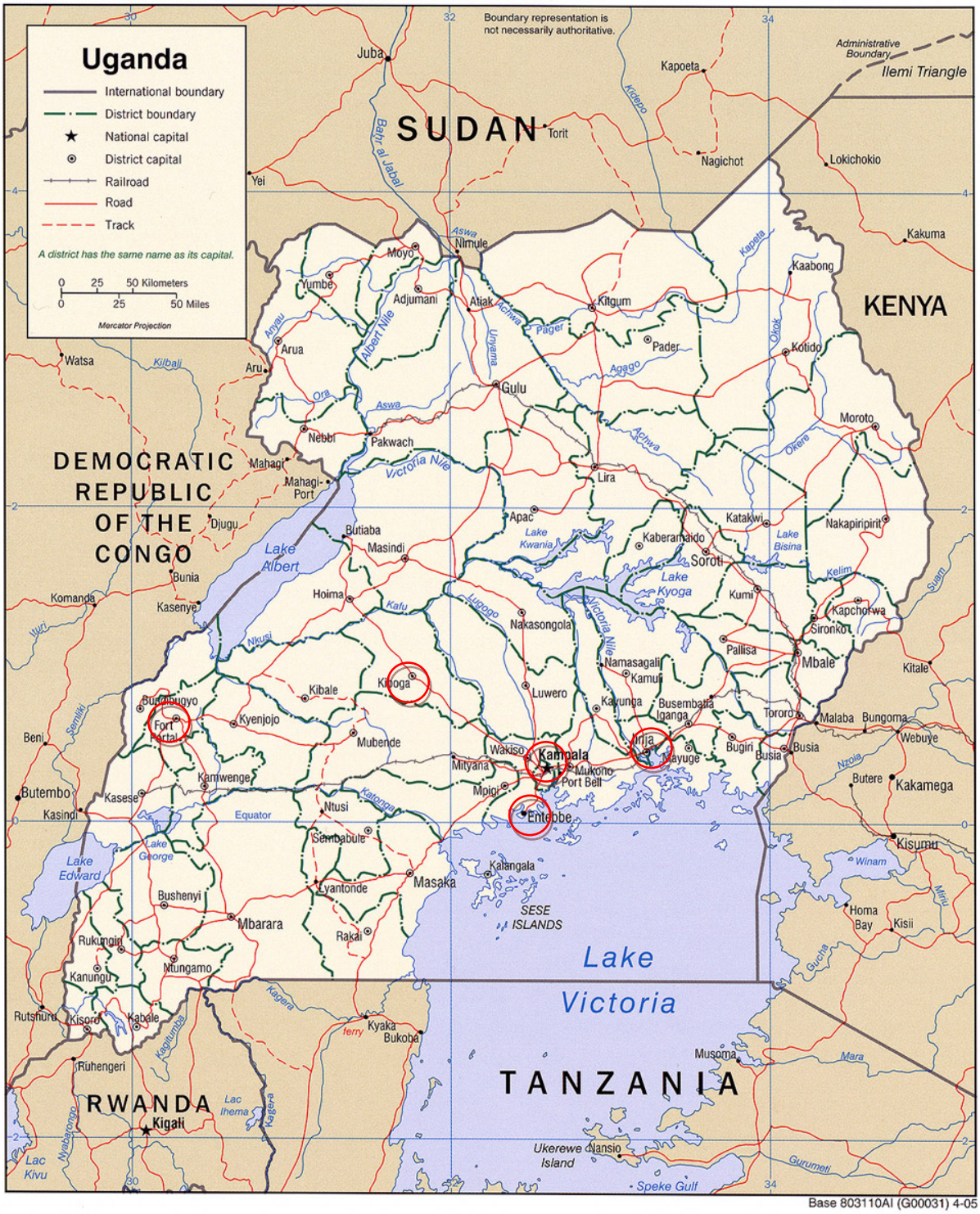
World Bank, *HIV epidemiology: A review of recent trends and lessons* (Washington DC, World Bank, 2006).

Worobey, Michael, Marlea Gemmel, Dirk Teuwen, et al., "Direct evidence of extensive diversity of HIV-1 in Kinshasa by 1960," *Nature*, 455 (2008), 661-664.

Young, Alwyn, "The Gift of the Dying: The tragedy of AIDS and the welfare of future African generations," *Quarterly Journal of Economics,* 120 (2005), 243-266.

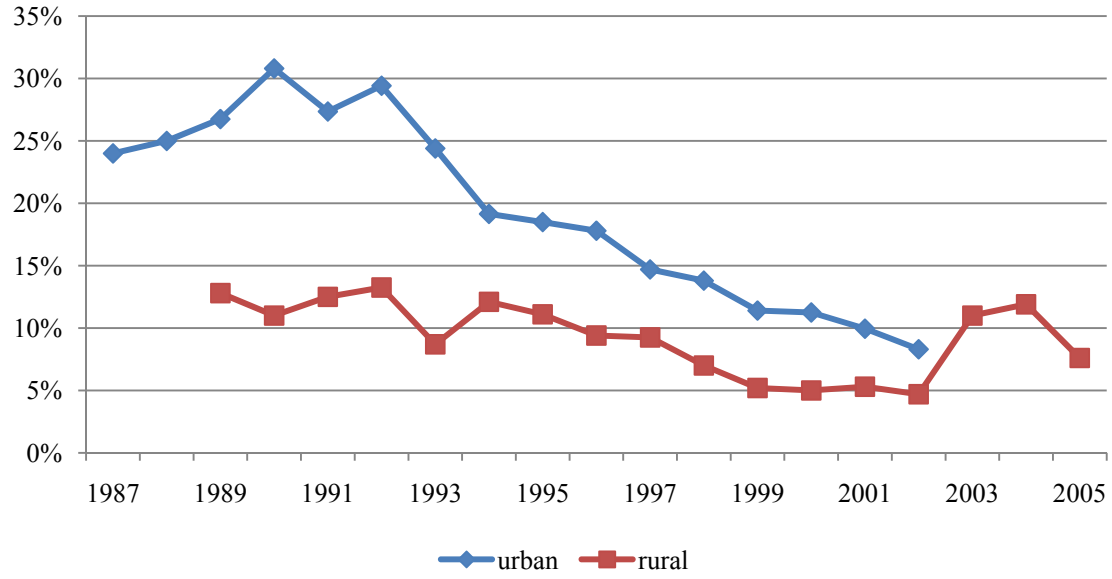
Young, Alwyn, "In sorrow to bring forth children: fertility amidst the plague of HIV," *Journal of Economic Growth*, 12 (2007), 283-327.

Figure 1: Map of Uganda, with antenatal clinic sites outlined in circles.



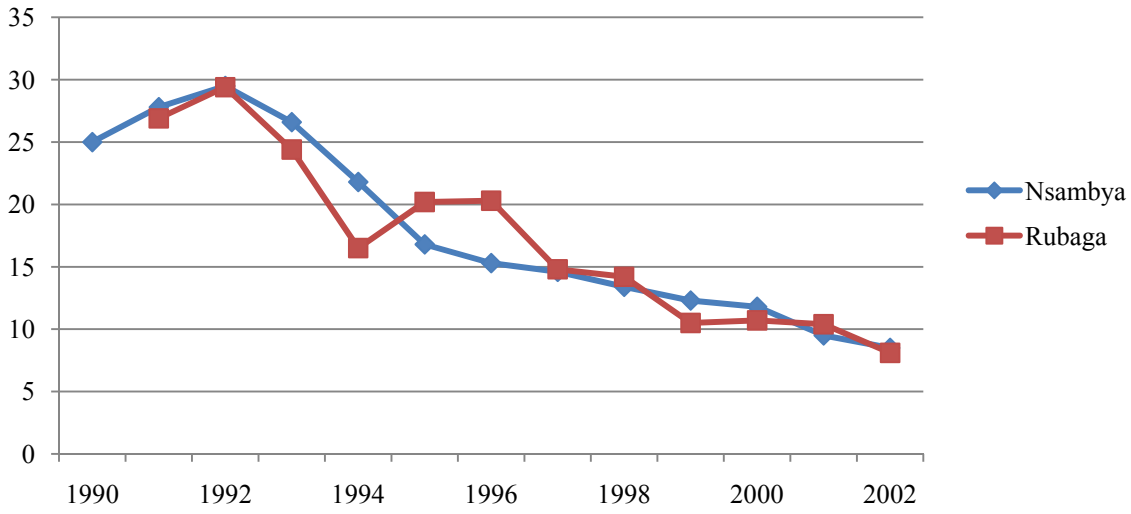
Source: Perry-Castañeda Library Map Collection at the University of Texas.

Figure 2: Median HIV Prevalence Among Pregnant Women in Uganda



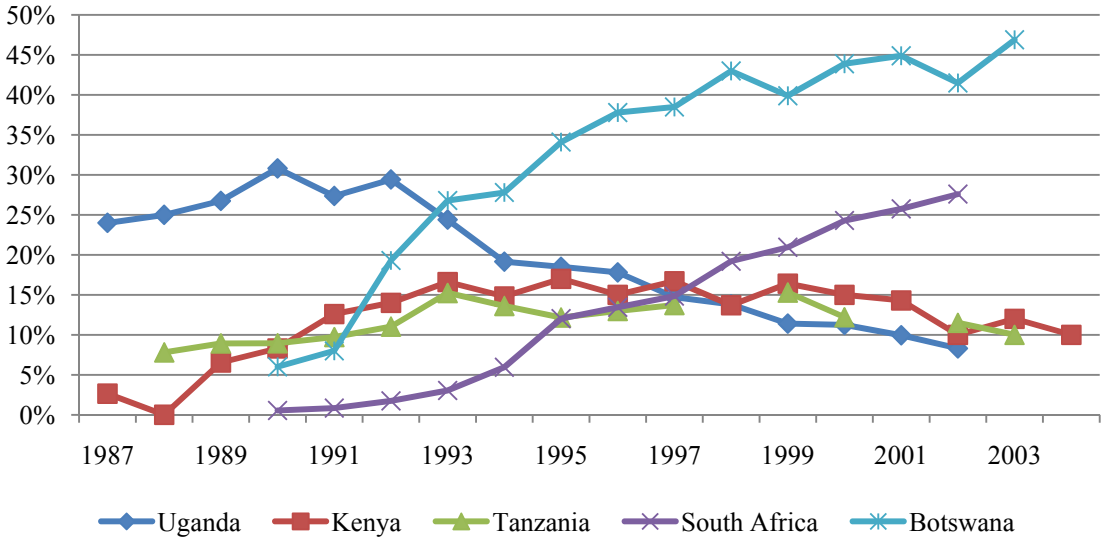
Notes: Data are from UNAIDS Uganda Epidemiological Fact Sheets on HIV/AIDS from 2004, 2006 and 2008. Median annual prevalence is from antenatal clinics that performed surveillance HIV testing.

Figure 3: Median HIV prevalence in two selected ANC sites
(Nsambya and Rubaga)



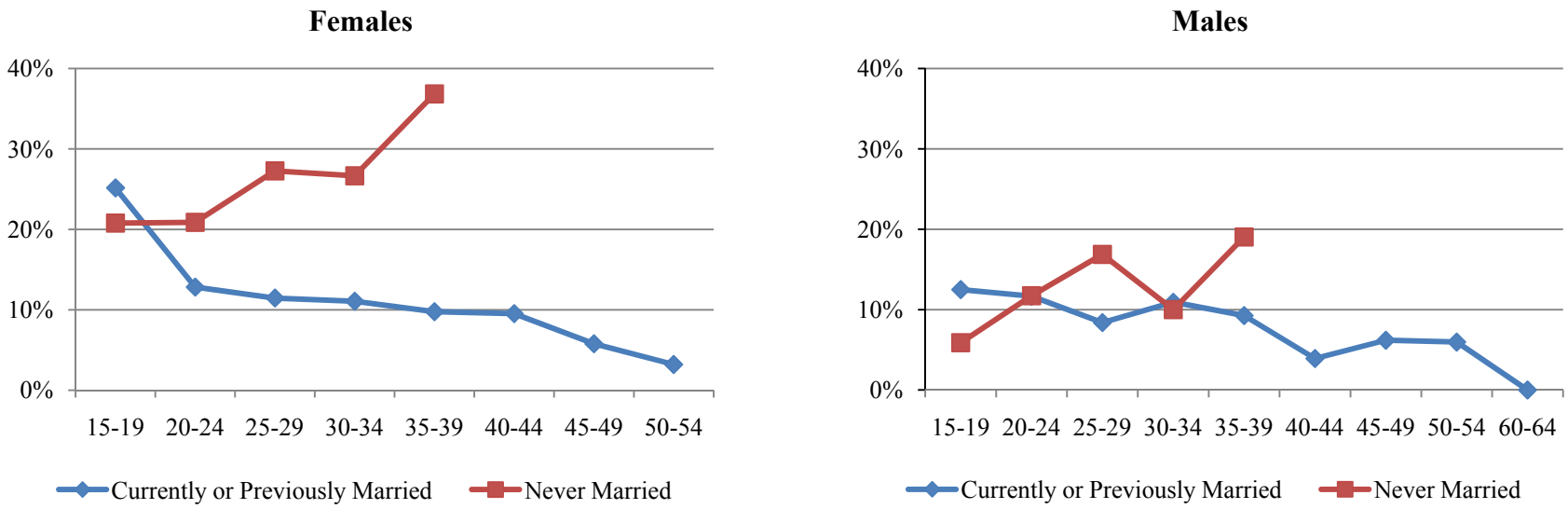
Notes: Data are from UNAIDS Uganda Epidemiological Fact Sheets on HIV/AIDS from 2004, 2006 and 2008. Nsambya and Rubaga are two hospital-based antenatal clinics located in Kampala with longitudinal data on HIV surveillance test results.

Figure 4: Median HIV Prevalence Among Pregnant Women in Urban Areas, Select Countries in Africa



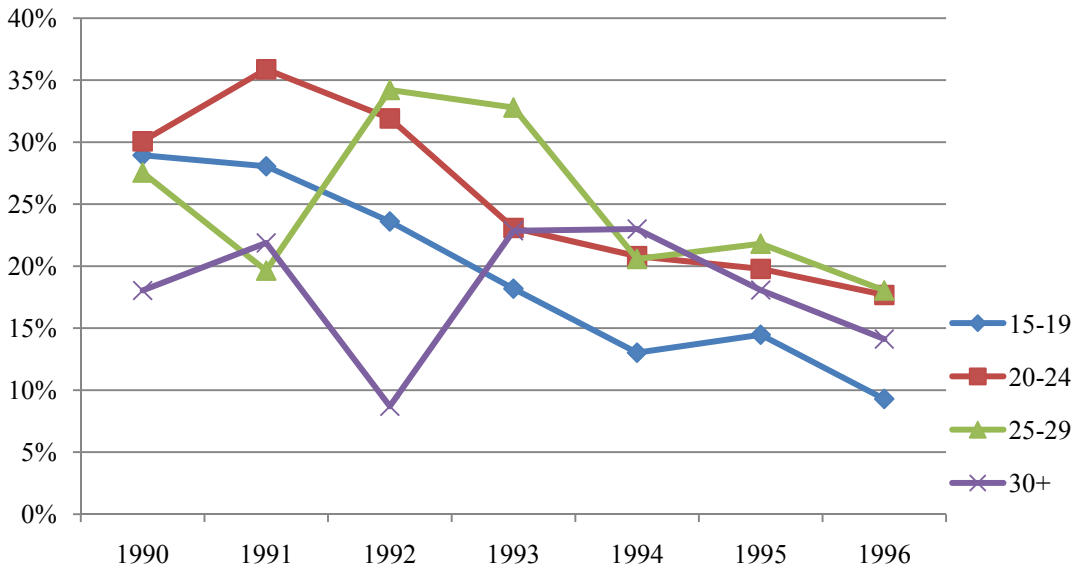
Notes: Data are from UNAIDS Epidemiological Facts Sheets on HIV/AIDS for the relevant countries from 2004, 2006 and 2008. Median annual prevalence is from urban antenatal clinics that performed surveillance HIV testing.

Figure 5: HIV Prevalence by Marital Status, Gender and Age (1987)



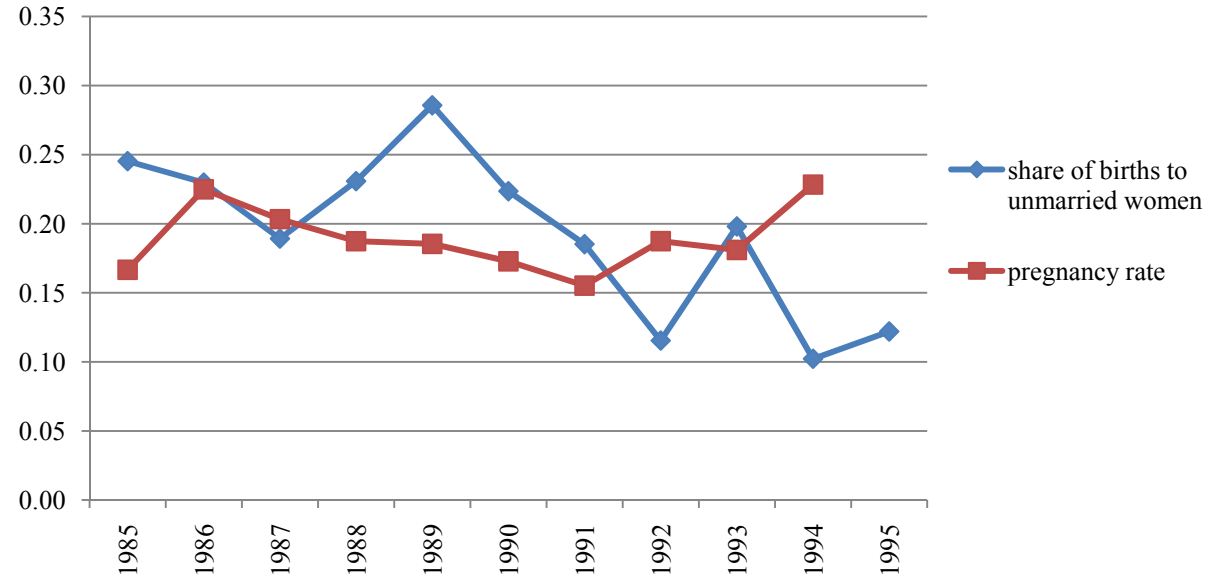
Notes: Data are from the 1987 Knowledge, Attitudes and Practices survey of 3928 Ugandans over the age of 15 in two subcounties approximately 10 kilometers from Kampala. The prevalence is the percent of individuals in each subgroup with a positive anti-HIV 1 antibody test. For never married women and men, after a certain age (45 for men, 40 for women), there are very few people. We omit those values.

Figure 6: HIV Prevalence Among Pregnant Women by Age, Kampala



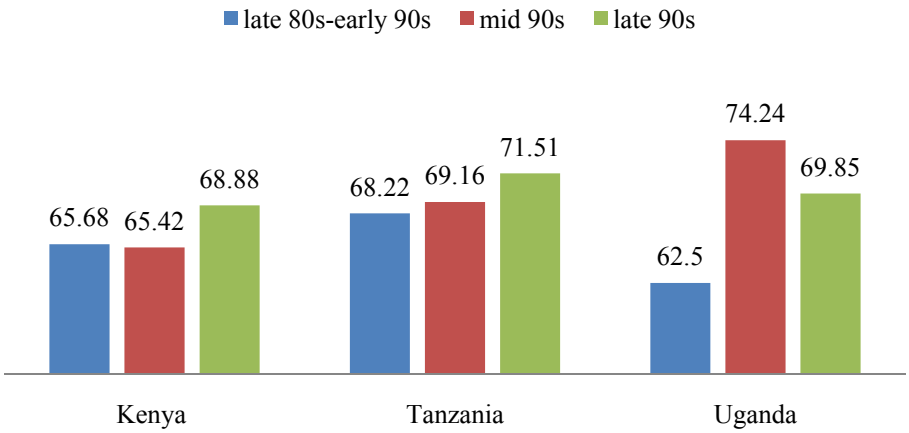
Note: Data from United States Census Bureau, HIV/AIDS Surveillance website.

Figure 7: Births to Women ages 15-19,
Central Uganda



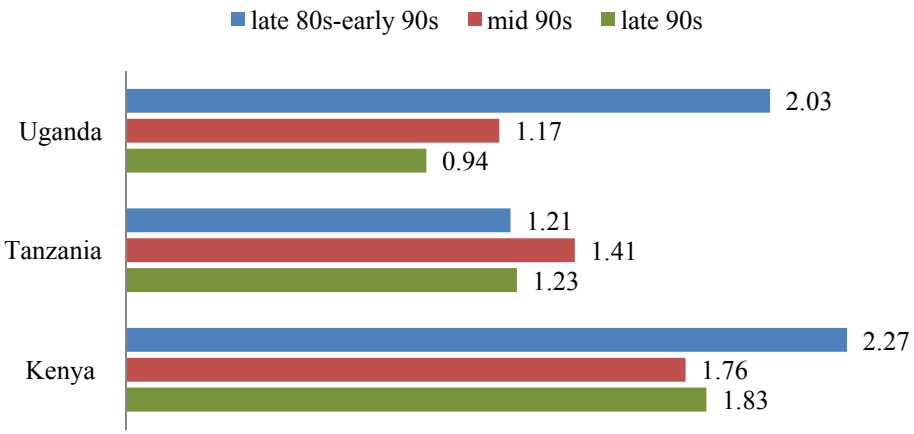
Notes: Data are from Uganda Demographic and Health Survey, 1988 and 1995. Pregnancy rate is the ratio of annual births to women aged 15-19 years to the total number of women in that age group, central region of Uganda.

Figure 8: Percent of Never Married Young Women Who Have Never Had Sex



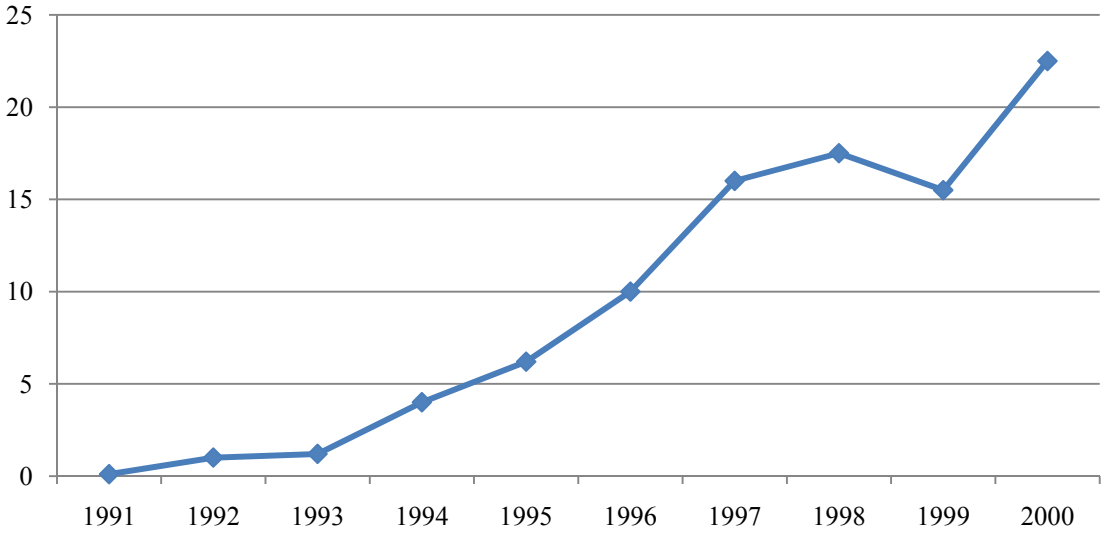
Notes: Data for Uganda are for 1988/89, 1995 and 2000/01. Data for Tanzania are for 1991, 1996 and 1999. Data for Kenya are for 1989, 1993 and 1998. All data are from Demographic and Health Surveys.

Figure 9: Average Duration of Premarital Sexual Activity, Young Women



Notes: Data for Uganda are for 1988/89, 1995 and 2000/01. Data for Tanzania are for 1991, 1996 and 1999. Data for Kenya are for 1989, 1993 and 1998. All data are from Demographic and Health Surveys.

Figure 10: Number of Social Marketed Condoms Sold in Uganda
(millions)



Note: Data are from Deloitte Touche Tohmatsu, Commercial Market Strategies, Uganda 2001 reproduced in the USAID publication, "What happened in Uganda".

Figure 11: Percent of all Men Who have Ever Used Condoms

Notes: Data are for Uganda 1995 and 2000/1, Kenya 1993 and 1998, Tanzania 1996 and 1999. All are from Demographic and Health Surveys.

Table 1: Knowledge about HIV/AIDS

	Uganda		Kenya			
	Men (1995)	Women (1995)	Men (1993)	Women (1993)	Women (1998)	Women (2003)
Ever heard of AIDS	99.9%	99.2%	99.4%	98.3%	99.2%	98.4%
Abstain sex	35	36	10	14	27	42
Limit partners	24	49	60	53	32	55
Use condoms	36	26	30	16	38	40
Don't know ways to avoid AIDS	7	11	15	22	19	16
Knows someone with and/or died of AIDS	93	89	41	43	71	74
Thinks they can catch AIDS	69	80	68	46	---	---
A healthy person can transmit AIDS	90	86	88	76	79	85
AIDS can be transmitted mother to child	84	87	90	86	86	88

Notes: Data are from the Demographic and Health Surveys for the indicated years. Men are aged 20-54 and women are aged 15-49.

Table 2: HIV Prevalence in Uganda, 1987

Group	Description	Percent 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	Prostitutes	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 Knowledge Attitudes and Practices Survey, Uganda 1987. The survey consisted of 3928 Ugandans over 15 years of age in two subcounties approximately 10 kilometers from Kampala. Prevalence is the percent of those in a given subgroup with a positive HIV 1 antibody test, except for prostitutes, who are assumed to have the HIV prevalence of older, never married females.

Table 3: 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 4: 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	7.0
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 Table 5. Coital frequency for women is predicted using the coefficients in Table 6. 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 extramarital partners is based upon responses in the Knowledge, Attitudes and Practices Survey, Uganda 1987.

In the last four weeks.

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

Table 5: Predicted Age of First Marriage and Age of Sexual Debut

Independent Variable	Females		Males	
	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)
N	1,154	1,287	423	568
R^2	0.05	0.05	0.10	0.03

Notes: Each column is an OLS regression with clustered, robust standard errors in parentheses. For females, the data are based on the 1988 DHS in Uganda, central region. For males, the data are based on the 1995 DHS in Uganda, central region. Only men who married or debuted at least 10 years prior to the survey were included in the analysis.

** Statistically significant at the 5 percent level.

*** Statistically significant at the 1 percent level.

Table 6: Predicted Coital Activity

	Number of coital acts per month		
	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**	-0.290
	(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	2,455	471	177
Vuong test	5.590	---	2.750

Notes: Regressions are based on the 1988 DHS in Uganda. Clustered, robust standard errors in parentheses. Depending on Vuong test results, we used either a zero inflated negative binomial or a negative binomial model.

** Statistically significant at the 5 percent level.

*** Statistically significant at the 1 percent level.

Table 7: Estimated Mixing Probabilities in the Baseline Model

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

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.

Table 8: Baseline Model Prevalence of HIV

Demographic group	Actual prevalence	Predicted prevalence	Percentage point difference	Percent difference
Women				
Total	13.1%	13.3%	0.1%	0.9%
Never married	22.5%	20.8%	-1.7%	-7.4%
Young married	17.5%	17.7%	0.2%	1.2%
Old married	8.3%	8.4%	0.1%	1.4%
Divorced/widowed*	9.5%	11.3%	1.8%	19.7%
Men				
Total	10.6%	10.7%	0.1%	0.4%
Young never married	5.9%	6.7%	0.8%	14.0%
Old never married	13.7%	14.5%	0.8%	5.5%
Young married	10.9%	10.0%	-0.9%	-8.9%
Old married	6.0%	5.9%	-0.1%	-0.9%
Divorced widowed*	20.7%	20.9%	0.2%	1.1%

*Assumed married for 1/2 time sexually active

Table 9: Simulated Impact of Abstinence, Faithfulness, Condom Use, and Death on HIV Positive Rates Among Pregnant Women

	Pregnant Women of Age			
Simulation	15-19	20-24	25-29	30+
Baseline Simulation				
Actual rate, 1990-1992	26.5%	26.8%	23.1%	15.7%
Predicted rate	26.5%	24.6%	24.7%	11.0%
Percent difference	0.1%	-8.3%	6.9%	-30.0%
Percentage point difference	0.0%	-2.2%	1.6%	-4.7%
Change in rate: 1990-92 to 1994-96	-14.0%	-6.8%	-2.3%	0.2%
Simulating Population Changes				
Impact of Abstinence				
Predicted rate	18.6%	22.9%	24.6%	10.9%
Percentage point change in predicted rate	-8.0%	-1.8%	-0.1%	-0.1%
Impact of Being Faithful				
Predicted rate	26.6%	24.7%	24.7%	11.0%
Percentage point change in predicted rate	0.1%	0.1%	0.0%	0.0%
Impact of Condom Use				
Predicted rate	22.4%	23.3%	24.4%	10.8%
Percentage point change in predicted rate	-4.2%	-1.3%	-0.3%	-0.2%
Impact of AIDS-Related Deaths				
Predicted rate	24.3%	23.1%	24.2%	10.7%
Percentage point change in predicted rate	-2.2%	-1.5%	-0.5%	-0.2%
Cumulative Impact				
Predicted change from abstinence, be faithful condom use, and death	-14.3%	-4.6%	-0.9%	-0.42%
Share of total decline	102%	67%	34%	---
Contribution of:				
Abstinence	57%	26%	0%	NA
Be faithful	-1%	-1%	0%	NA
Condom use	30%	20%	13%	NA
Death of α males	16%	23%	22%	NA

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

Table 10: Condom Use Among Men, 1995

	Males				
Females	Young, never married	Old, never married	Young, married	Old, married	Divorced/ widowed
Never married	34%	40%	23%	11%	33%
Young, married	34%	40%	4%	11%	33%
Old, married	34%	40%	23%	2%	33%
Divorced/widowed	34%	40%	23%	11%	33%

Notes: Data are from the 1995 Ugandan Demographic and Health Survey. 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.

Appendix A

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 prostitutes in our data, we omit $\pi_{\mu\alpha}$, $\pi_{\mu\beta}$, and $\pi_{\mu\delta}$ 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 prostitutes, this yields the equations:

6. $\pi_{\lambda\alpha}\,\Lambda = \pi_{\alpha\lambda}A$
7. $\pi_{\lambda\beta}\,\Lambda = \pi_{\beta\lambda}B$
8. $\pi_{\lambda\delta}\,\Lambda = \pi_{\delta\lambda}\Delta$
9. $\pi_{\omega\alpha}\Omega = \pi_{\alpha\omega}A$
10. $\pi_{\omega\beta}\Omega = \pi_{\beta\omega}B$
11. $\pi_{\omega\delta}\Omega = \pi_{\delta\omega}\Delta$

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 (prostitutes) is directly proportional to STI rates. Thus:

$\pi_{\omega\delta} = 1\text{-}d$
$\pi_{\omega\alpha} = (1- \pi_{\omega\delta})*(b/a)$
$\pi_{\omega\beta} = (1- \pi_{\omega\delta})*(1-b/a)$
$\pi_{\alpha\omega} = (d/a)\ \pi_{\delta\omega}$
$\pi_{\beta\omega} = (d/b)\ \pi_{\delta\omega}$
$\pi_{\beta\mu} = (b/a)\ \pi_{\alpha\mu}$
$\pi_{\delta\mu} = (d/a)\ \pi_{\alpha\mu}$

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. $\pi_{\alpha\lambda} + (d/a)\pi_{\delta\omega} + \pi_{\alpha\mu} = 1$
4. $\pi_{\beta\lambda} + (d/b)\pi_{\delta\omega} + (b/a)\pi_{\alpha\mu} = 1$
5. $\pi_{\delta\lambda} + \pi_{\delta\omega} + (d/a)\pi_{\alpha\mu} = 1$
6. $\pi_{\lambda\alpha}\ \Lambda = \pi_{\alpha\lambda}\ A$
7. $\pi_{\lambda\beta}\ \Lambda = \pi_{\beta\lambda}\ B$
8. $\pi_{\lambda\delta}\ \Lambda = \pi_{\delta\lambda}\ \Delta$
9. $\Omega = \pi_{\delta\omega}\ \{\Delta + (d/a)\ A + (d/b)B\}$

Appendix B

Estimated Mixing Probabilities Under Alternative Simulations

	Males		
Females	α	B	δ
Delayed Debut Among Young Women			
μ	NA / 36%	NA / 25%	NA / 17%
λ	83% / 34%	14% / 32%	2% / 21%
ω	17% / 30%	7% / 43%	76% / 62%
Reduced Out of Marriage Sexual Contact			
μ	NA / 29%	NA / 20%	NA / 14%
λ	85% / 41%	14% / 37%	2% / 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. Prostitutes are not in our sample; thus, we do not estimate the probability that they sexual contact with any particular set of men.