A Quantitative Theory of HIV Diffusion

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(PRELIMINARY AND INCOMPLETE)

Abstract

This paper develops a model of HIV diffusion among heterosexual individuals who choose whether to engage in sex—the source of potential infection—and the nature of their relationship as measured by the rate of destruction and the presence of extra-marital partners (i.e., concurrency). We study the general equilibrium of this economy where the distribution of the population is endogenously determined in stationary and nonstationary environments. We use a calibrated version of the model to analyze the quantitative implications of policies that target AIDS treatment and HIV prevention. Our preliminary results look promising. First, the theory is able to generate high and low aggregate HIV prevalence scenarios associated with mature epidemics. Second, we find that changes in policy parameters generate large aggregate and distributinal changes in the long-run patterns of HIV infection and prevalence. For example, free universal access to antiretrovirals generates an increase in aggregate HIV prevalence from 9.7% to 34.4%; increasing the stability of sexual partnerships by 2 years substantially reduces HIV prevalence; reductions in the level of infectiousness per sexual act succed in lowering the aggregate HIV prevalence; and, reductions in the efficiency of the matching technology reduce HIV prevalence by a factor of .8. Distributional effects across gender, marital status and type of sexual partner (which includes the possibility of serodiscordant couples) are also analyzed.

1 Introduction

Current HIV policies implemented by UNAIDS largely favor the allocation of funds toward AIDS treatment or its consequences (e.g. distribution of antiretrovirals, and care of orphans) rather than HIV prevention. This can be explained by the lack of policy guidance on the effects of prevention policies on sexual behavior change and hence on the evolution of the epidemic. Our paper works toward closing this gap.

We develop a simple model of the endogenous diffusion of AIDS. We consider a population that is heterogeneous in terms of gender and utility from sexual contact. We study the full information case and show that there are balanced growth paths in which some individuals choose to practice safe sex, which in the context of the model corresponds to having sex only with healthy individuals, while others are willing to risk the possibility of infection. This differential behavior, as well as a number of shocks that we take as exogenous in this first version, determine the steady state distribution of infection [1] The model is calibrated to match several data moments of risky sexual behavior in Sub-Saharan Africa, and provide quantitative normative policy assessments.

In future work, we plan to study the impact of adding a "highly infectious period", incorporate extra-marital partners, as well as the consequences of letting people endogenously destroy their partnerships (divorce) which will allow the study of conurrency and the factors that influence it. To keep the model simple, we have abstracted away from income differences, even though we suspect that the differential ability to attract sex partners can explain the observed relationship between income (and education) and prevalence of AIDS. We also plan to explore the nonstationary properties of our model, in particular, how much it can replicate the evolution of the HIV epidemic, and in that context study the consequences of policy at different stages of the epidemic. In doing so, we will take into account nonstationary aspects of the education gradient of HIV: More-educated individuals change faster their sexual behavior (number of extramarital partners) in

[1] The discussion on related literature on sexual behavior, Kremer [1994], Kremer [1996], Oster [2005], Greenwood et al. [2010] and Fernández-Villaverde et al. [2010], needs to be completed. A discussion of the literature on the welfare implications of HIV will be provided as well: Young [2005] and Santaeulàlia-Llopis [2011]

response to the epidemic than less-educated individuals.

2 Basic facts of the HIV Epidemic

Figure 1 shows the evolution of the HIV epidemic for a subset of Sub-Saharan countries.[2] The HIV prevalence time path follows an assymetric bell shape in all countries starting in early and mid-1980s, with a fast rise and a slow decline. There is, however, a large degree of heterogeneity across countries: At any given year there is a large variation of aggregate prevalence rates across countries (e.g. in 2010 the HIV prevalence is 1.5% in Burkina Faso, 5.2% in Cameroon, 12.5% in Malawi, and 23.3% in Lesotho); the HIV peak level and year also differ across country (e.g. Cameroon peaked in 1993 at 7.1%, Zimbabwe in 1997 at 29.1%, Malawi in 1998 at 13.8%, and Guinea has not yet peaked in 2010); and the speed at which each country moves toward and away from the peak from their respective peak is also different.

Table 1 shows the HIV prevalence by gender, marital status and education groups in Malawi using the Malawi Diffusion and Ideational Change Project (MDICP 2006).[3] First, in a a country with a mature epidemics such as Malawi, the HIV prevalence in females is larger than that of males, respectively 9.9% and 7.8%. Married individuals have an HIV prevalence of 6.0% (6.1% for females, 5.4% for males) lower than that of singles divorced 19.2% and widowed 12.5%. Again, divorced females have a prevalence larger than that of men, 20.5% against 12.5%, and widowed females also have a prevalence larger than that of men, 12.5% against 13.3%. Finally, the never married are the demographic group with the lowest HIV prevalence, an average of 1.5% with women having a prevalence of virtually 0%. Last, more-educated individuals have an HIV prevalence larger than less-educated individuals: The HIV prevalence rate is 6.1% for individuals with less than primary schooling, 7.5% with primary schooling, and 8.9% with secondary schooling. Further, this education gradient of HIV is more evident for females than for men.

[2] This section relies heavily on the discussion of the relationship between education, HIV status, and risky sexual behavior over the stages of the HIV epidemic in Iorio and Santaeulàlia-Llopis [2011].

[3] MDICP collects fairly representative panel data for rural Malawi biannually since 1998. Individuals are tested for HIV since 2004.

Table ?? shows an additional a set of additional figures for other countries SSA computed using the Demographic and Health Surveys for men (we will add women shortly). There we see differences in the sign of the education gradient of HIV across countries [to be completed]. In this context, Iorio and Santaeulàlia-Llopis [2011] find empirical evidence on the education gradient of HIV being nonstationary over stages of the epidemic.

3 The Model

Assume a heterogeneous population where the utility associated with having sexual intercourse (unprotected for now) is given by u, where u is drawn from a distribution F. The utility of not having sex is normalized to zero. Assume that the occurrence of death is associated with a Poisson counter that depends on the individual's health status: An individual with health status $k \in \{h, b\} = \{\texttt{healthy,infected}\}$ and gender $j \in \{m, f\} = \{\texttt{male, female}\}$ faces a process with arrival rate δ_k^j. The utility cost of death is $-D$, with $D > 0$. Healthy individuals of gender j who are paired with infected individuals can become infected at the rate η^j.

We assume that the rate at which single individuals find a partner is driven, in part, by a matching function. For a given individual, characterized by the pair $(j, k) = (\texttt{gender, health status})$, not all matches are acceptable. In particular, we assume that some healthy individuals will choose to accept to be matched with other healthy individuals and, hence, can guarantee themselves a zero probability of infection, while other will accept all available matches. We take the rate at which partnerships are destroyed as exogenous (and independent of health status). To be precise, we model destruction (or divorce) as a Poisson process that, if initiated by an individual of gender j has parameter β^j. The total rate at which partnerships end is then $\beta = \beta^m + \beta^f$.

At this point we do not consider the possibility of abstinence, although it is relatively simple to add this feature. In the context of the model, if the cost of finding a partner is sufficiently small or the infimum of the support of the cdf F sufficiently higher than zero, all individuals will choose to look for a partner.

We study the case in which the rate at which paired females with health status k give birth to individuals of gender j, denoted by $\bar{\gamma}_h^j$, is exogenous

We assume that single individuals search for partners and the rate at which they find a partner of the opposite gender is given by a matching function as in Mortensen and Pissarides (XXXX). Let $\tilde{M}(\bar{N}^m(s), \bar{N}^f(s))$ be the total number of matches when $\bar{N}^m(s)$ and $\bar{N}^f(s)$ females are searching. We assume that $\tilde{M}(\bar{N}^m(s), \bar{N}^f(s))$ is homogeneous of degree one. Define $\theta = \bar{N}^m(s)/\bar{N}^f(s)$, and $q^m(\theta) = \tilde{M}(\bar{N}^m(s), \bar{N}^f(s))/\bar{N}^m(s) = \tilde{M}(1, \theta^{-1})$, a decreasing function of θ. Similarly, let $q^f(\theta) = \tilde{M}(\bar{N}^m(s), \bar{N}^f(s))/\bar{N}^f(s0 = \tilde{M}(\theta, 1)$, an increasing function of θ.

We look for an equilibrium in which there are cutoff points (u^m, u^f) such that that healthy persons of gender j with utility level $u \leq u^j$ choose to match only with other healthy individuals, while the rest, i.e. those with $u > u^j$, find all candidates acceptable sex partners.

3.1 Individual Problem

We assume for simplicity that history does not matter. Thus, an individual can become infected only if currently paired with an infected individual. We also assume —this is not essential— that only paired individuals can give birth. For now, we take the birth rate as exogenous but, in future work, we plan endogeneize it. We assume that individuals are risk neutral and that the discount rate is r. We denote by $V^j(k)(u)$ the value (lifetime utility) of a single individual of gender j, who is in state k and has utility u, and by $M^j(k, k')(u)$ the value of a matched individual of gender j, who is in state k, matched to an individual who is in state k' and has utility u.

In some cases, we need to distinguish two between high and low utility individuals as this endogenously determines who they accept as partners. Consider first the relevant HJB equation corresponding to single individuals

1. $[u \geq u^j]$

$$\begin{aligned} rV^j(h)(u) &= -c^j + q^j(\theta)[\mu^{j'} M^j(h,h)(u) + \\ &\quad (1-\mu^{j'})M^j(h,b)(u) - V^j(h)(u)] \\ &\quad +\delta_h^j[-D - V^j(h)]. \end{aligned} \tag{1}$$

Here, c^j is the cost of searching for a partner and $\mu^{j'}$ is the fraction of single individuals of the opposite sex who are healthy. The "cost" of death is denoted by D. This equation simply says that the flow lifetime utility for a single individual who accepts all possible matches is given by the cost of search plus that arrival rate of death, δ_h^j, times the "gain" associated with dying (the change in the value of utility), plus the rate at which this individual finds a partner, $q^j(\theta)$, times the expected gain from having found a partner. This expected gain depends on the lifetime utility of being paired with a healthy individual, $M^j(h,h)(u)$, weighted by the probability of finding a healthy member of the opposite sex in the singles pool, $\mu^{j'}$, and the lifetime utility of being matched to an infected individual, $M^j(h,b)(u)$, times the fraction of infected agents in the singles population.

2. $[u \leq u^j]$

$$\begin{aligned} rV^j(h)(u) &= -c^j + q^j(\theta)[\mu^{j'} M^j(h,h)(u) - V^j(h)(u)] \\ &\quad +\delta_h^j[-D - V^j(h)(u)]. \end{aligned} \tag{2}$$

The second branch is very similar to the first. The main difference is that this low flow utility individual accepts only healthy partners.

In the case of infected individuals, they have no incentive to distinguish between healthy or infected agents as they do not care for the utility of their partners. However, they are not an acceptable match for some of the people they meet. In particular, if they meet a single person of the opposite sex who has a low u $(u \leq u^{j'})$ this person will refuse to be matched.

In order to discuss this case we need to develop some notation. Let $N^j(p,k,k')(u)$ denote the number of matched (and hence not searching) individuals of gender j, who are in state k and matched to an individual in state k', whose utility is less than or equal to u. If we normalize, as we will, the total population (males plus females) to one, $N^j(p,k,k')(u)$ is a cumulative distribution with total mass given by $\bar{N}^j(p,k,k') = \lim_{u\to\infty} N^j(p,k,k')(u)$. Similarly, we denote by $N^j(s,k)(u)$ the number of individuals with singles in state k and, as before, we let $\bar{N}^j(s,k) = \lim_{u\to\infty} N^j(s,k)(u)$.

It turns out to be convenient to develop specific notation for some subpopulations. We let $\bar{N}^j(p,k) = \bar{N}^j(p,k,h) + \bar{N}^j(p,k,b)$ be the total number of individuals of gender j and state k who are paired, and $\bar{N}^j(s) = \bar{N}^j(s,h) + \bar{N}^j(s,b)$ the total number of singles irrespective of their health status. Note that this with this notation

$$\mu^j = \frac{\bar{N}^j(s,h)}{\bar{N}^j(s)}.$$

The utility of a single infected individual satisfies

$$\begin{aligned} rV^j(b)(u) &= -c^j + q^j(\theta)[\frac{\bar{N}^{j'}(s,h) - N^{j'}(s,h)(u^{j'})}{\bar{N}^{j'}(s)} M^j(b,h)(u) \\ &\quad +(1-\mu^{j'})M^j(b,b)(u) - V^j(b)(u)] + \delta_h^j[-D - V^j(b)(u)]. \end{aligned} \tag{3}$$

Here, the term $\bar{N}^{j'}(s,h) - N^{j'}(s,h)(u^{j'})$ is the number of healthy potential partners who are willing to be matched with an infected individual. Thus, $[\bar{N}^{j'}(s,h) - N^{j'}(s,h)(u^{j'})]/\bar{N}^{j'}(s)$ is probability of finding a healthy partner. The other terms are standard.

Next we consider the lifetime utility of a paired individual. In the case of a healthy person matched to another healthy person the HJB equation is

$$rM^j(h,h)(u) = u + \beta[V^j(h)(u) - M^j(h,h)(u)] + \delta_h^j[-D - M^j(h,h)(u)] \quad (4)$$
$$\delta_h^{j'}[V^j(h)(u) - M^j(h,h)(u)].$$

The interpretation is straightforward. The first term is the utility of being matched. The second captures the gain associated with breaking up the partnership, the third is the gain (loss) of dying, and the last one is the gain corresponding to the change in status (from matched to single) triggered by the death of a spouse. Note that individuals in this type of partnership never become infected.

Consider next the value of a healthy individual matched to an infected one. It is given by (omitting the argument u when there is no risk of confusion)

$$rM^j(h,b) = u + \beta[V^j(h) - M^j(h,b)] + \delta_h^j[-D - M^j(h,b)] \quad (5)$$
$$\delta_b^{j'}[V^j(h) - M^j(h,b)] + \eta^j[M^j(b,b) - M^j(h,b)].$$

The first three terms are identical to the previous case. The fourth captures the gain (loss) associated with the healthy individual becoming infected. The other value functions are very similar, and we report them for completeness.

$$rM^j(b,h) = u + \beta[V^j(b) - M^j(b,h)] + \delta_b^j[-D - M^j(b,h)] \quad (6)$$
$$\delta_h^{j'}[V^j(b) - M^j(b,h)] + \eta^{j'}[M^j(b,b) - M^j(b,h)].$$

and

$$\begin{aligned} rM^j(b,b) &= u + \beta[V^j(b) - M^j(b,b)] + \delta_b^j[-D - M^j(b,b)] \\ &\quad \delta_b^{j'}[V^j(b) - M^j(b,b)]. \end{aligned} \tag{7}$$

In order to solve for the value functions it suffices to know the cutoff points (u^m, u^f), the sex ratio in the singles market, θ, and some terms of the distribution of the population. We turn next to the description of the evolution of each population category.

3.2 The Evolution of the Population

Population evolves endogenously. In this section we describe the laws of motion of the number of individuals of a given gender, in a given state and status. We consider (for now) only balanced growth paths. Thus for example, the number of individuals of gender j, in state k that are paired with an individual in state k' at time t whose utility value is less than or equal to u is denoted $\tilde{N}^j(t,p,k,k')(u)$. The balanced growth assumption is equivalent to specifying that there exists a γ such that

$$\tilde{N}^j(t,p,k,k') = e^{\gamma t} N^j(p,k,k'). \tag{8}$$

It then follows that

$$\frac{\partial \tilde{N}^j}{\partial t}(t,p,k,k') = \gamma \tilde{N}^j(t,p,k,k') = \gamma e^{\gamma t} N^j(p,k,k').$$

Since the term $e^{\gamma t}$ will appear on both sides of the dynamic evolution equations, it can be omitted. The relevant laws of motion are:

1. $u \geq u^j$

$$\begin{aligned} \gamma N^j(s,h)(u) &= [\bar{\gamma}_h^j \bar{N}^f(p,h) + \bar{\gamma}_b^j \bar{N}^f(p,b)]F(u) - \delta_h^j N^j(s,h)(u) \\ &\quad -q^j(\theta)[\mu^{j'} N^j(s,h)(u^j) + N^j(s,h)(u) - N^j(s,h)(u^j)] \\ &\quad +\beta[N^j(p,h,h)(u) + N^j(p,h,b)(u)] \end{aligned} \tag{9}$$

The interpretation is simple. The term $\bar{\gamma}_h^j \bar{N}^f(p,h) + \bar{\gamma}_b^j \bar{N}^f(p,b)$ is the total number of individuals of gender j born (all in state h) and $F(u)$ is the fraction that have utility less than u. The term $\delta_h^j N^j(s,h)(u)$ represents the flow of deaths from this category, and $\beta[N^j(p,h,h)(u) + N^j(p,h,b)(u)]$ those that become single (while healthy) because their matches (either to a healthy or infected partner) are destroyed.

The number of individuals who leave this population because they get matched is given by $q^j(\theta)[\mu^{j'} N^j(s,h)(u^j) + N^j(s,h)(u) - N^j(s,h)(u^j)]$. The first term inside the brackets captures the size of the population that gets paired (after being matched) and who have "low" values of u. Thus, these individuals only accept healthy partners, as captured by the term $\mu^{j'}$. The rest of the population, $N^j(s,h)(u) - N^j(s,h)(u^j)$, accepts all matches.

2. $u \leq u^j$.

$$\begin{aligned} \gamma N^j(s,h)(u) &= [\gamma_h^j \bar{N}^f(p,h) + \gamma_b^j \bar{N}^f(p,b)]F(u) \\ &\quad -\delta_h^j N^j(s,h)(u) - q^j(\theta)\mu^{j'} N^j(s,h)(u) \\ &\quad +\beta[N^j(p,h,h)(u) + N^j(p,h,b)(u)]. \end{aligned} \tag{10}$$

The interpretation of the evolution equation for the "low utility" individuals is similar to the previous one. The only noteworthy difference is that since they only accept healthy individuals as potential partners, the rate at which they leave singlehood is smaller than for those in the high utility category.

In the case of infected single individuals they law of motion satisfies

$$\begin{aligned}\gamma N^j(s,b)(u) &= -q^j(\theta)[\frac{\bar{N}^{j'}(s,h) - N^{j'}(s,h)(u^{j'})}{\bar{N}^{j'}(s)} + (1-\mu^{j'})]N^j(s,b)(u) \\ &\quad +\beta[N^j(p,b,h)(u) + N^j(p,b,b)(u)] - \delta_h^j N^j(s,b)(u).\end{aligned}$$

The first term measures the number of single infected individuals who find a partner. Even though infected agents are willing to be paired with anyone, some of their potential partners are not willing to accept them. Specifically, only infected partners, a fraction $1-\mu^{j'}$ of the singles, and high utility healthy individuals, a fraction $[\bar{N}^{j'}(s,h) - N^{j'}(s,h)(u^{j'})]/\bar{N}^{j'}(s)$, acquiesce to a match. The previous expression can be simplifies to

$$\begin{aligned}\gamma N^j(s,b)(u) &= -q^j(\theta)[1 - \frac{N^{j'}(s,h)(u^{j'})}{\bar{N}^{j'}(s)}]N^j(s,b)(u) \\ &\quad -\delta_b^j N^j(s,b)(u) + \beta[N^j(p,b,h)(u) + N^j(p,b,b)(u)].\end{aligned} \tag{11}$$

There are four categories of "matched" populations, and we describe them now. Consider first the healthy pairs, i.e. $N^j(p,h,h)$.

$$\begin{aligned}\gamma N^j(p,h,h)(u) &= (\beta + \delta_h^j + \delta_h^{j'})N^j(p,h,h)(u) \\ &\quad q^j(\theta)\mu^{j'}N^j(s,h)(u)\end{aligned} \tag{12}$$

Note that, under our conjecture, $N^j(p,h,b)(u) = 0$, for all $u \leq u^j$ as a healthy individual with this level of utility refuses to be matched with an infected agent. Thus, the only relevant branch for this subpopulation is the high utility. Formally,

$$\begin{aligned}\gamma N^j(p,h,b)(u) &= q^j(\theta)(1-\mu^{j'})[N^j(s,h)(u) - N^j(s,h)(u^j)] \\ &\quad -(\beta + \delta_h^j + \delta_b^{j'})N^j(p,h,b)(u) - \eta^j N^j(p,h,b)(u).\end{aligned} \tag{13}$$

The last term is the flow of healthy individuals in this group who become infected and hence "leave" this subpopulation.

The rest of the laws of motion are

$$\begin{aligned}\gamma N^j(p,b,h)(u) &= q^j(\theta)[\frac{\bar{N}^{j'}(s,h) - N^{j'}(s,h)(u^j)}{\bar{N}^{j'}(s)}]N^j(s,b)(u) \\ &\quad -(\beta + \delta_b^j + \delta_h^{j'})N^j(p,b,h)(u) - \eta^{j'}N^j(p,b,h)(u),\end{aligned}$$

or

$$\begin{aligned}\gamma N^j(p,b,h)(u) &= q^j(\theta)[\mu^{j'} - \frac{N^{j'}(s,h)(u^j)}{\bar{N}^{j'}(s)}]N^j(s,b)(u) \\ &\quad -(\beta + \delta_b^j + \delta_h^{j'})N^j(p,b,h)(u) - \eta^{j'}N^j(p,b,h)(u).\end{aligned} \tag{14}$$

and

$$\begin{aligned}\gamma N^j(p,b,b)(u) &= q^j(\theta)(1-\mu^{j'})N^j(s,b)(u) - (\beta + \delta_b^j + \delta_b^{j'})N^j(p,b,h)(u) \\ &\quad +\eta^j N^j(p,h,b)(u) + \eta^{j'}N^j(p,b,h)(u).\end{aligned} \tag{15}$$

Note that, for $u \leq u^j$ the last term is zero.

The next step is to use equations (8)-(15) to determine the total number of individuals in each category. In order to ease notation define

$$\bar{\gamma}^j = \frac{\gamma_h^j \bar{N}^f(p,h) + \gamma_b^j \bar{N}^f(p,b)}{\bar{N}^j(s)}. \tag{16}$$

This gives the total number of births of gender j relative to the number of single individuals of that gender.

Then, from equation (9) it follows that

$$(\gamma+\delta_h^j+q^j(\theta))\mu^j = \bar{\gamma}^j+q^j(\theta)[(1-\mu^{j'})\frac{N^j(s,h)(u^j)}{\bar{N}^j(s)}] + \beta\frac{\bar{N}^j(p,h)}{\bar{N}^j(s)}. \tag{17}$$

From equation (11)

$$\left[\gamma+\delta_b^j+q^j(\theta)[1-\frac{N^{j'}(s,h)(u^{j'})}{\bar{N}^{j'}(s)}]\right](1-\mu^j)=\beta\frac{\bar{N}^j(p,b)}{\bar{N}^j(s)}. \tag{18}$$

From equation (12)

$$(\gamma+\delta_h^j+\delta_h^{j'}+\beta)\frac{\bar{N}^j(p,h,h)}{\bar{N}^j(s)}=q^j(\theta)\mu^{j'}\mu^j. \tag{19}$$

From equation (13)

$$(\gamma+\beta+\delta_h^j+\delta_b^{j'}+\eta^j)\frac{\bar{N}^j(p,h,b)}{\bar{N}^j(s)}=q^j(\theta)(1-\mu^{j'})[\mu^j-\frac{N^j(s,h)(u^j)}{\bar{N}^j(s)}]. \tag{20}$$

From equation (14)

$$(\gamma+\beta+\delta_b^j+\delta_h^{j'}+\eta^{j'})\frac{\bar{N}^j(p,b,h)}{\bar{N}^j(s)}=q^j(\theta)(1-\mu^j)[\mu^{j'}-\frac{N^{j'}(s,h)(u^j)}{\bar{N}^{j'}(s)}]. \tag{21}$$

From equation (15)

$$(\gamma+\beta+\delta_b^j+\delta_b^{j'})\frac{\bar{N}^j(p,b,b)}{\bar{N}^j(s)} = q^j(\theta)(1-\mu^j)(1-\mu^{j'})+ \eta^j\frac{\bar{N}^j(p,h,b)}{\bar{N}^j(s)}+\eta^{j'}\frac{\bar{N}^j(p,b,h)}{\bar{N}^j(s)}. \tag{22}$$

3.3 Equilibrium

Here we define the equilibrium and provide an algorithm for its computation. As of now, we focus on stationary equilibria.

3.3.1 Definition

Definition 1 (Stationary equilibrium)**.** A stationary equilibrium is a set of functions that include all the value functions and the distributions of the population and a vector $[\theta, u^m, u^f]$ such that the following (among others) conditions are satisfied:

1. [Equilibrium in the matching market]:

$$\begin{aligned} \bar{N}^m(p,h,h) &= \bar{N}^f(p,h,h) \\ \bar{N}^m(p,b,b) &= \bar{N}^f(p,b,b) \\ \bar{N}^m(p,h,b) &= \bar{N}^f(p,b,h) \\ \bar{N}^m(p,b,h) &= \bar{N}^f(p,h,b) \end{aligned} \tag{23}$$

   It is straightforward to check that our specification of the laws of motion of the population and the assumption that the matching function is homogeneous of degree one [Need to explain this better] implies that all the conditions in equation (23) are automatically satisfied. Thus, these conditions do not restrict our choices.

2. [Population normalization]

$$\sum_{j\in\{m,f\}} [\bar{N}^j(s) + \bar{N}^j(p,h) + \bar{N}^j(p,h)] = 1. \tag{24}$$

3. [Consistency in the matching market]

$$\theta = \frac{\bar{N}^m(s)}{\bar{N}^f(s)}. \tag{25}$$

Given the assumption that the function $\tilde{M}$ is homogeneous of degree one, it follows that

$$q^f(\theta) = \theta q^m(\theta)$$

4. [Utility maximization] Let the lifetime utility of a low utility individual be (this follows from equation (1))

$$V^j(h)(u) = \frac{-c^j + q^j(\theta)\mu^{j'} M^j(h,h)(u) - \delta_h^j D}{r + q^j(\theta) + \delta_h^j} \tag{26}$$

while his utility if he were to accept all partners would be

$$\begin{aligned} \hat{V}^j(h)(u) \quad = \quad & [-c^j + q^j(\theta)(\mu^{j'} M^j(h,h)(u) + \\ & (1-\mu^{j'}) M^j(h,b)(u)) - \delta_h^j D]/ \\ & [r + q^j(\theta)(1-\mu^{j'}) + \delta_h^j]. \end{aligned} \tag{27}$$

Then, in the equilibrium that we conjecture it must be the case that

$$V^j(h)(u) \geq \hat{V}^j(h)(u), \quad \text{for all } u \leq u^j.$$

Conjecture 2. *The value functions are affine functions of u. For example, $M^j(k,k')(u)$ is of the form $M_0^j(k,k') + M_1^j(k,k')u$ for some constants $M_0^j(k,k')$ and $M_1^j(k,k')$.*

Remark 3. *One concern is that we have one variable to satisfy two equilibrium conditions. Existence?*

Definition 4 (Nonstationary equilibrium)**.** Nonstationary equilibrium.

3.3.2 Computatinal Algorithm

We compute the stationary equilibrium using the following steps:

1. Guess the cutoff utility levels $(u^m, u_i^f) \in [0, \bar{u}]^2$.

2. Given (i) (u^m, u^f), (ii) the set of population and technology parameters $(\delta_k^j, \eta^j, \beta, \phi_k^j, A, \alpha)$, and (iii) an initial distribution of the population, χ_0, then we can compute the invariant distribution, χ, by iterating forward the system (??)-(??), that is

$$\chi_{t+1} = \Omega \, \chi_t$$

   until $\chi = \chi_{t+1} = \chi_t$.

3. Given χ—hence, the population statistics θ, μ^j and μ_{acc}^j—solve for the value functions (??)-(??). Precisely, we conjecture our value functions belong to the collection of functions that can be written as a linear combination of a set of n-known linearly independent basis functions ψ_i, $i = 1, ..., n$,

$$\sum_{i=1}^{n} \omega_i \, \psi_i(u) \tag{28}$$

   and we solve for the n unknown ω_i coefficients for piecewise linear splines.

4. Check $V^j(h)(u) \geq \hat{V}^j(h)(u)$ for all $u \leq u^j$. If yes, STOP, otherwise, go back to STEP 1 and update our guess.

3.4 Calibration

Our choice of parameters accomodates for some general aspects of the HIV epidemic. Some of these parameters, such as life expectancy, singles/married ratios, and number of lifetime partners are country-specific. We will accomodate for such idiosincrasies shortly.

Parameter	Value	Commment
δ_h^j	.015	An expected life-time of 65 years
δ_b^j	.025	A somewhat lower life expectancy for infected individuals
β	.2	Partnerships gets destructed an average of every five years
η^m	.2	Female-to-male infection per coital act is about .0011[4]
η^f	.4	Male-to-female infection per coital act is twice as much
ϕ_h	.1	Children arrive every 10 years for healthy married women
ϕ_b	.08	Infected women have about 20% less children
A	.9	Match singles/married ratio of about .31
α	.5	Fixing the curvature of the matching function

4 Results

Table 2 shows the results for our baseline economy using the preliminary set of parameters in Section 3.4. The population ratios in the economy are such that there are .887 females per male, there are .692 single per single male, and the number of singles are less than one third the number of married.

The aggregate HIV prevalence of this economy is 9.7%. This prevalence is lower for females, 7.6%, than for males 11.5%, and for singles 7.2% than for married individuals 10.4%. In singlehood, females have less prevalence 5.4% than males 8.5%. Within marriage, females have less prevalence 8.2% than males 12.5%; the fraction of couples with healthy partners is 86.6%; the fraction of serodiscordant couples is 6.3%; and the fraction of household with both partners infected is 7.2%.

The model also uncovers patterns of new HIV infections. About .7% of the total population gets infected per period. Female new infection happens at a rate of 1% per period, and at rate .4% for males. In these models, all HIV infections happen in serodiscordant couples.

5 Policy Experiments

As of now, five policy experiments have been conducted under stationary environments.

5.1 Universal ARV Distribution: Increasing Life Expectancy

Describe Table 3 [To be completed]. Universal access to ARVs: ultimately this implies equating life expectancy between healthy and infected indviduals in the model. ARV Drugs increase prevalence rate by almost a factor of four. For an alternative benchmark choice of parameters that yields about 20% of HIV (South Africa), ARVs rise the long-run prevalence to about 80%. The long-run (new) infections raise substantially: more than 3% (1%) of females (males) get infected per period.

5.2 Reducing Infectiousness per Sexual Act

Reducing Infections per Sexual Act: Gels, 100% Condom use, an HIV Vaccine. Describe Table 4 [To be completed]. Introduction of gels (or other forms of prevention technologies) that reduce the infection per coital act to sthg close to zero. Reductions in η^j reduces the amount of infections per sexual act, but the total number of infections is given by the interaction between η^j and the number of sexual acts. In our context, one sexual act is identical to having one partner (per period). There is sexual behavior change as we observe the amount of people that accept sex with infected individuals increases.

5.3 Promoting Stability of Sexual Partnerships (and the Opposite)

Describe Table 5 [To be completed]. Promotion of long-stable relationships: this implies reducing the exogenous (for now) partnership destruction rate. In our context, Greenwood, Kircher and Tertilt (2010) is an extreme case that sets $\beta = .0$.

5.4 Reproductive Health

Describe Table 6 [To be completed]. Reducing mother to child transmission: this implies two things: increase in the survival probability of infected children and increase fertility. In order to analyze the effects of reductions (or increases) in mother-to-child transmission of HIV we need

to allow infected females to give birth to infected children. We will do so shortly.

5.5 Increasing the Efficiency of the Matching Technology

Describe Table 7 [To be completed]. Importantly, this suggests that that have a more efficient matching technology (better roads, trains, railroad network, bars...) have higher aggregate HIV prevalence. Hence, if higher income is associated with better roads, then a more efficienty matching technology can help to explain why richer regions in Sub-Saharan Africa have more HIV prevalence.

6 Conclusion

- We have showed a useful theory of HIV diffusion in which the epidemic arises endogenously.
- Our preliminary results look very promising. First, the theory is able to generate high and low aggregate HIV prevalence scenarios associated with mature epidemics. Second, we seem to have good identification properties as our equilibrium outcome responds to the parameter choice.
- There is still a long road ahead. Our to-do list includes.

  1. Endogenize two more sexual margins that we think are important:
     - First, partnership destruction (as per our results).
     - Second, introduce extramarital partners through two types of markets.

     This allows us to study issues like concurrency.
  2. Introduction of heterogeneity in income (consumption) that changes the incentives of partnership stability. Further, we can test if our theory is able replicate the education gradient of HIV. This implies further HIV policies that can be evaluated as we can specifically target populations by education level.
  3. Introduce different levels of infectiousness. See Appendix B.

4. Quantify the ability of income differentials in generating different HIV epidemics across countries (East, West and Southern Africa).

5. Solve for the nonstationary environment, and answer how much our economy is able to replicate the evolution of the HIV epidemic in Sub-Saharan Africa. Conduct the previous policy experiments in this nonstationary environment. Given heterogeneity in income, we will pursue to replicate some of the nonstationary aspects of the education gradient of HIV: More-educated individuals change faster their sexual behavior (number of extramarital partners) in response to the epidemic than less-educated individuals.

References

M.F. Chersich and H.V. Rees. Vulnerability of women in southern africa to infection with hiv: biological determinants and priority health sector interventions. *AIDS*, Suppl. 4(S27-40), 2008.

Jesús Fernández-Villaverde, Jeremy Greenwood, and Nezih Guner. From shame to game in one hundred years: An economic model of the rise in pre-marital sex and its de-stigmatization. Mimeo University of Pennsylvania, 2010.

R.H. Gray, M.J. Wawer, R. Brookmeyer, N.K. Sewankambo, D. Serwadda, F. Wabwire-Mangen, T. Lutalo, X. Li, T. vanCott, and T.C. Quinn. Probability of hiv-1 transmission per coital act in monogamous, heterosexual, hiv-1-discordant couples in rakai, uganda. *The Lancet*, 357 (9263):1149–53, 2004.

Jeremy Greenwood, Philip Kircher, and Michele Tertilt. An equilibrium model of the african hiv/aids epidemic. Mimeo, Stanford University, 2010.

Daniela Iorio and Raül Santaeulàlia-Llopis. Education, hiv status and risky sexual behavior: How much does the stage of the hiv epidemic matter?,. Mimeo, Universitat Autònoma de Barcelona and Washington University in St. Louis, 2011.

Michael Kremer. Can having fewer partners increase the prevalence of aids? NBER Working Paper, 4942, 1994.

Michael Kremer. Integrating ehavioral choice into epidemiological models of the aids epidemic. *Quarterly Journal of Economics*, pages 549–573, May 1996.

Vermund SH. Modjarrad K, Chamot E. Impact of small reductions in plasma hiv rna levels on the risk of heterosexual transmission and disease progression. *AIDS*, 22(16):2179–85, 2008.

A. Nicolosi, M.L. Correa-Leite, M. Musicco, C. Arici, G. Gavazzeni, and A. Lazzarin. The efficiency

of male-tofemale and female-to-male sexual transmission of the human immunodeficiency virus: A study of 730 stable couples. *Epidemiology*, 5(6):570–575, 1994.

Emily Oster. Sexually transmitted infections, sexual behavior and the hiv/aids epidemic. *Quarterly Journal of Economics*, pages 467–515, May 2005.

C.D. Pilcher, H.C. Tien, J.J. Eron, P.L. Vernazza, S.Y. Leu, P.W. Stewart, L.E. Goh, and M.S. Cohen. Brief but efficient: acute hiv infection and the sexual transmission of hiv. *Journal of Infectious Diseases*, 189(10):1785–92, 2004.

R. Santaeulàlia-Llopis. Aggregate effects of aids on development. Mimeo, Washington University in St. Louis, 2011.

M.J. Wawer, R.H. Gray, N.K. Sewankambo, D. Serwadda D, X. Li, O. Laeyendecker, N. Kiwanuka, G. Kigozi, M. Kiddugavu, T. Lutalo, F. Nalugoda, F. Wabwire-Mangen, M.P. Meehan, and T.C. Quinn. Rates of hiv-1 transmission per coital act, by stage of hiv-1 infection, in rakai, uganda. *Journal of Infectious Diseases*, 191(9):1403–9, 2005.

A. Young. The gift of the dying: The tragedy of aids and the welfare of future african generations. *Quarterly Journal of Economics*, 120(2):423–466, 2005.

Table 1: Cross-sectional facts of HIV prevalence: Malawi MDCIP 2006 Sample

HIV Prevalence	Total	Women	Men
All sample	9.1	9.9	7.8
by marital status groups:			
Never Married	1.5	.0	1.8
Divorced	19.2	20.5	12.5
Widowed	12.5	12.5	13.3
Married	6.0	6.1	5.9
by education groups:			
Less than primary schooling	6.1	6.1	6.0
Primary schooling	7.5	8.9	5.6
Secondary schooling	8.9	14.6	6.4

Source: Malawi Ideational Change Project, 2006.

Table 2: Baseline Results

Variable	Value
Population ratios:	
Females/Males	1.001
Single females/Single males	1.029
Singles/Married	.596
Married females/Married males	1.000
Population growth	.042
Patterns of HIV prevalence:	
HIV	.127
HIV females	.138
HIV males	.115
HIV singles	.125
HIV single females	.131
HIV single males	.119
HIV married	.128
HIV married females	.142
HIV married males	.113
Healthy couples/Total couples	.813
Serodiscordant couples/Total Couples	.117
Infected couples/Total couples	.070
Patterns of HIV infection:	
Total HIV infections/Healthy population	.011
Female HIV infections/Healthy females	.012
Male HIV infections/Healthy males	.009
Acceptance rates:	
Fraction of healthy females that accepts	.694
Fraction of healthy males that accepts	.823

Notes: HIV prevalence is the ratio of the stock of HIV infected individuals at period t in population group g, over the stock of population in group g at the beginning of period t; HIV infection is the ratio of the flow of HIV infections between period t and $t+1$ in population group g, over the stock of healthy population in group g at the beginning of period t.

Table 3: Policy I: Universal ARV Distribution: Increasing Life Expectancy

Variable	Value, given (δ_b^f, δ_b^m)		
	Baseline (.025,.025)	Low ARVs (.02,.02)	High ARVs (.008,.010)
Population:			
Females/Males	1.001	1.008	1.014
Single females/Single males	1.029	1.022	1.040
Singles/Married	.596	.608	.559
Married females/Married males	1.000	1.000	1.000
Population growth	.042	.039	.041
Patterns of HIV prevalence:			
HIV	.127	.280	.519
HIV females	.138	.304	.560
HIV males	.115	.256	.478
HIV singles	.125	.261	.467
HIV single females	.131	.278	.503
HIV single males	.119	.244	.429
HIV married	.128	.291	.548
HIV married females	.142	.320	.592
HIV married males	.113	.263	.504
Healthy couples/Total couples	.813	.595	.283
Serodiscordant couples/Total Couples	.117	.225	.336
Infected couples/Total couples	.070	.178	.380
Patterns of HIV infection:			
HIV infections/Total population	.011	.028	.059
Female HIV infections/Total population	.012	.029	.069
Male HIV infections/Total population	.009	.028	.050
Acceptance rates of infected partners:			
Fraction of healthy single females that accepts	.694	.758	1.000
Fraction of healthy single males that accepts	.823	.848	1.000

Table 4: Policy II: Reduction of Infectiousness

Variable	Value, given (η_b^f, η_b^m)		
	Baseline (.2,.4)	Medium (.1,.2)	Low (.01,.01)
Population:			
Females/Males	.887	.941	1.018
Single females/Single males	.692	.838	1.078
Married females/Married males	.957	.975	1.000
Singles/Married	.312	.310	.311
Patterns of HIV prevalence:			
HIV	.097	.057	.000
HIV females	.076	.044	.000
HIV males	.115	.069	.000
HIV singles	.072	.042	.000
HIV single females	.054	.032	.000
HIV single males	.085	.050	.000
HIV married	.104	.061	.000
HIV married females	.082	.048	.000
HIV married males	.126	.075	.000
Healthy couples/Total couples	.866	.920	1.000
Serodiscordant couples/Total Couples	.063	.038	.000
Infected couples/Total couples	.072	.042	.000
Patterns of HIV infection:			
HIV infections/Total population	.007	.004	.000
Female HIV infections/Total population	.010	.006	.000
Male HIV infections/Total population	.004	.002	.000
Acceptance rates:			
Fraction of healthy females that accepts	.946	.968	1.000
Fraction of healthy males that accepts	.915	.950	1.000

Table 5: Policy III: Promoting Stability of Sexual Partnerships

Variable	Value, given β		
	Baseline (.20)	Increase (.25)	Decrease (.15)
Population:			
Females/Males	.887	.661	1.015
Single females/Single males	.692	.285	1.075
Married females/Married males	.957	.895	1.000
Singles/Married	.312	.422	.258
Patterns of HIV prevalence:			
HIV	.097	.286	.000
HIV females	.076	.249	.000
HIV males	.115	.310	.000
HIV singles	.072	.239	.000
HIV single females	.054	.188	.000
HIV single males	.085	.254	.000
HIV married	.104	.305	.000
HIV married females	.082	.261	.000
HIV married males	.126	.345	.000
Healthy couples/Total couples	.866	.608	1.000
Serodiscordant couples/Total Couples	.063	.179	.000
Infected couples/Total couples	.072	.213	.000
Patterns of HIV infection:			
HIV infections/Total population	.007	.018	.000
Female HIV infections/Total population	.010	.001	.000
Male HIV infections/Total population	.004	.009	.000
Acceptance rates:			
Fraction of healthy females that accepts	.946	.812	1.000
Fraction of healthy males that accepts	.915	.746	1.000

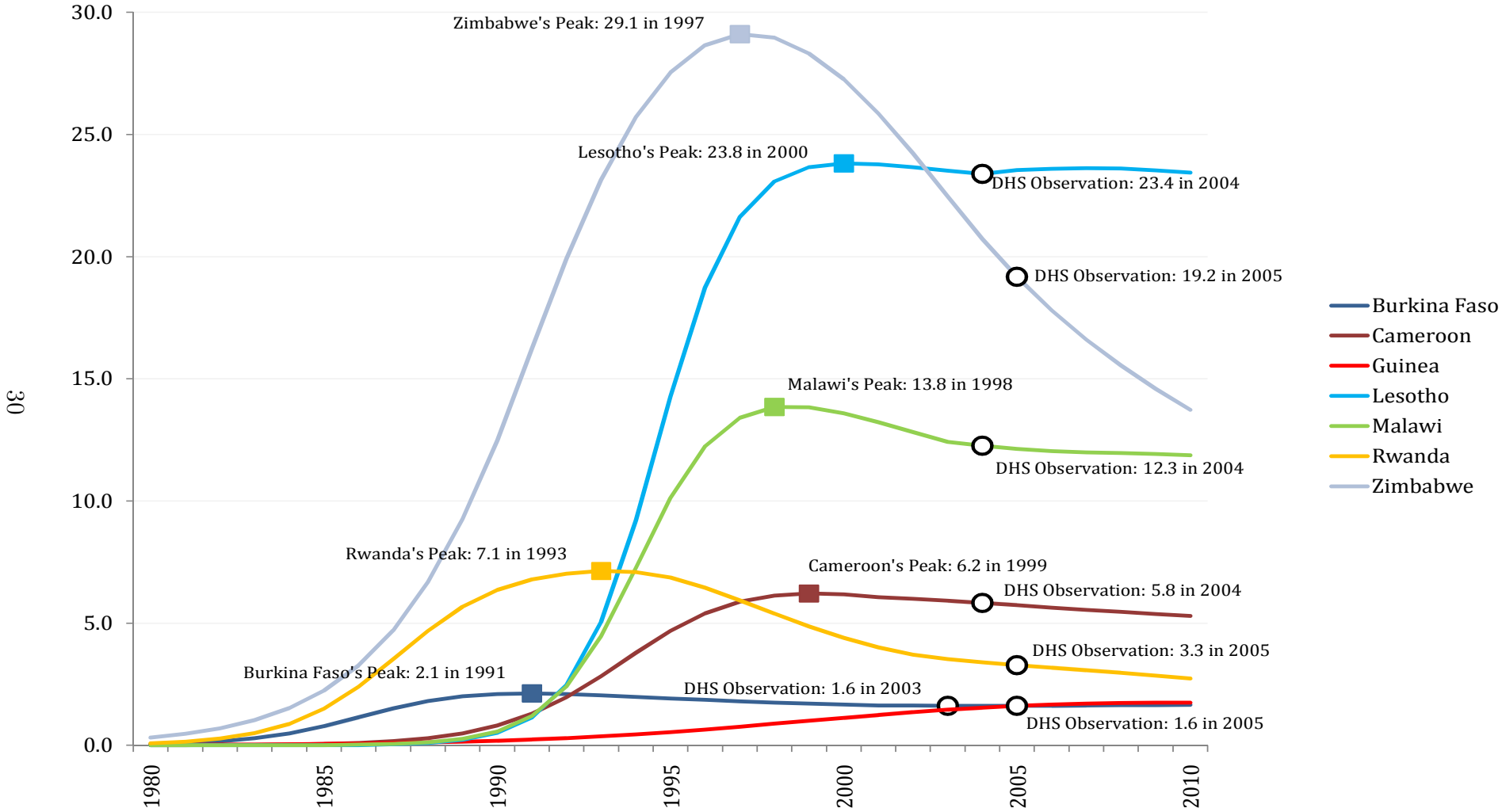
Table 6: Policy IV: Increasing Healthy Children per Infected Women

Variable	Value, given ϕ	
	Baseline (.08)	Increase (.1)
Population:		
Females/Males	.887	.916
Single females/Single males	.692	.771
Married females/Married males	.957	.966
Singles/Married	.312	.311
Patterns of HIV prevalence:		
HIV	.097	.004
HIV females	.076	.058
HIV males	.115	.089
HIV singles	.072	.055
HIV single females	.054	.041
HIV single males	.085	.065
HIV married	.104	.080
HIV married females	.082	.062
HIV married males	.126	.097
Healthy couples/Total couples	.866	.896
Serodiscordant couples/Total Couples	.063	.049
Infected couples/Total couples	.072	.055
Patterns of HIV infection:		
HIV infections/Total population	.007	.005
Female HIV infections/Total population	.010	.008
Male HIV infections/Total population	.004	.003
Acceptance rates:		
Fraction of healthy females that accepts	.946	.959
Fraction of healthy males that accepts	.915	.935

Table 7: Policy V: Increasing the Efficiency in the Matching Technology

Variable	Value, given A		
	Baseline (.9)	Increase (.5)	Decrease (.01)
Population:			
Females/Males	.887	.918	1.133
Single females/Single males	.692	.842	1.197
Married females/Married males	.957	.962	.996
Singles/Married	.312	.536	2.392
Patterns of HIV prevalence:			
HIV	.097	.080	.006
HIV females	.076	.062	.005
HIV males	.115	.096	.008
HIV singles	.072	.060	.005
HIV single females	.054	.046	.004
HIV single males	.085	.073	.007
HIV married	.104	.090	.009
HIV married females	.082	.070	.007
HIV married males	.126	.109	.011
Healthy couples/Total couples	.866	.885	.989
Serodiscordant couples/Total Couples	.063	.053	.005
Infected couples/Total couples	.072	.062	.006
Patterns of HIV infection:			
HIV infections/Total population	.007	.005	.000
Female HIV infections/Total population	.010	.007	.000
Male HIV infections/Total population	.004	.003	.000
Acceptance rates:			
Fraction of healthy females that accepts	.946	.954	.996
Fraction of healthy males that accepts	.915	.927	.993

Figure 1: The Evolution of the HIV Epidemic: A Sub-Saharan African Sample



circle on each HIV time-path displays the HIV prevalence at the year that the DHS data were collected.

A Some Notation

Here is the basic notation. A superscript j denotes gender. Almost all functions/quantities are indexed by gender. The relevant parameters of independent Poisson processes are:

1. $\delta_k^j =$ death rate of gender j and state k.
2. $\eta^j =$ rate at which individuals of gender j get infected (i.e. progress from h to b)
3. $\beta^j =$ rate at which an individual of type j destroys a partnership. Even though we index this by gender, all that matters is the total rate at which a partnership is destroyed. Since the Poisson processes are independent, all that matters is $\beta = \beta^m + \beta^f$. However, keeping the notation separate at this point will simplify the endogenous choice of intensity (if we ever do that).
4. $\mu^j =$ Fraction of single individuals of gender j that are healthy.
5. $\bar{\gamma}_k^j =$ Poisson parameter that determines the probability that a female in state k will give birth to an individual of gender j.
6. $V^j(k)(u) =$ value of a single individual of gender j, who is in state k and has utility u.
7. $M^j(k,k')(u) =$ value of a matched individual of gender j, who is in state k, matched to an individual who is in state k' and has utility u.
8. $N^j(\ell,k) =$ Number of individuals with status ℓ who are in state k.
9. $N^j(p,k,k') =$ Number of matched (and hence not searching searching) individuals who are in state k and matched to another individual in state k'. [Note $N^j(p,k) = N^j(p,k,h) + N^j(p,k,b)$]
10. $\tilde{M}(\bar{N}^m, \bar{N}^f) =$ Total number of matches when $\bar{N}^m$ and $\bar{N}^f$ females are searching. Here $\bar{N}^j = N^j(s,h) + N^j(s,b)$. We assume that $\tilde{M}(\bar{N}^m, \bar{N}^f)$ is homogeneous of degree one.

Define $\theta = \bar{N}^m/\bar{N}^f$, and $q^m(\theta) = \tilde{M}(\bar{N}^m, \bar{N}^f)/\bar{N}^m = \tilde{M}(1, \theta^{-1})$, a decreasing function of θ. Similarly, let $q^f(\theta) = \tilde{M}(\bar{N}^m, \bar{N}^f)/\bar{N}^f = \tilde{M}(\theta, 1)$, an increasing function of θ.

11. Let u^j be the cutoff utility level that separates those healthy single individuals who are willing to match with anyone, from those who are only willing to match with healthy individuals.

B Level of Infectiousness: A Review

The medical literature suggets the HIV viral load is the chief predictor of the risk of heterosexual transmission of HIV. [5] The average rate of HIV transmission is about 0.0011/coital act, see Gray et al. [2004]. By infection phases (see Wawer et al. [2005]):

Infection Phase	Time interval	Transmission Rate
Phase I (Acute HIV Infection)	$(0, 2.5]$ months	$.0082$/coital act
Phase II	$(2.5, 15]$ months	$.0015$/coital act
Phase III	$(15, AIDS)$ months	$.0007$/coital act
Phase IV (AIDS)	$(AIDS + 6 - 25)$ months	$.0028$/coital act

Table 8: HIV Transmission Rates per Infection Phases

Modjarrad K [2008] finds the likelihood of transmitting HIV by heterosexual contact increased, on average, by 20% with every 0.3 log10 increment in HIV RNA. A 0.5 log10 increment in HIV RNA was associated with 40% greater risk of heterosexual transmission and a 1.0 log10 increment in HIV RNA was associated with 100% greater risk of heterosexual transmission. Similar numbers (slightly higher) are associated ncreased risk of progression to AIDS or death. The overall probability of HIV-1 transmission per coital act was 0.0011 (Rakai, Uganda) and this transmission rate increased from 0.0001 per act at viral loads of less than 1700 copies/mL to 0.0023 per act at 38 500 copies/mL or more, Gray et al. [2004].

There is gender asymmetry. The male-to-female transmission/coital act (controlling for a bunch of factors including sexual frequency) is 2.3 times the female-to-male transmission, see Nicolosi et al. [1994] (and many others). Further, Pilcher et al. [2004] finds that in the acute HIV infection phase, men with average semen HIV-1 loads (and withouth STDs) are likely to

[5] This evidence is available for many countries: Gray et al. [2004], Modjarrad K [2008], Chersich and Rees [2008], and ?.

infect 7%-24% of susceptible female sex partners. [6] [7]

[6] This is the case for the U.S. and Europe. There is not much evidence in developing countries, and the little there is seems to point the opposite direction, see Gray et al. [2004].

[7] Some of these studies also stress age and genital ulceration Gray et al. [2004] Transimission rates in Rakai, Uganda, were 0.0041 with genital ulceration versus 0.0011 without. But how many have genital ulcerations? We do not know, however, how many individuals in the populations they study have genital ulcerations.