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An Equilibrium Model of the Malawian HIV/AIDS Epidemic

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Abstract

An equilibrium search model of the Malawian HIV/AIDS epidemic is presented. Individuals engage in different types of sexual activity, which vary in their riskiness. When choosing a sexual activity, such as short-term sex without a condom, a person rationally considers its risk. A simulated version of the model is parameterized to match some salient facts about the Malawian epidemic. Some topical policies (e.g., male circumcision, treatment of other STDs, and promoting marriage) are studied and found to have potential to backfire: Moderate interventions may actually increase the prevalence of HIV/AIDS, due to shifts in human behavior and equilibrium effects.

Keywords: Bayesian learning, circumcision, condoms, disease transmission, HIV/AIDS, homo economicus, Malawi, marriage, policy intervention, sex markets, search, STDs

Preliminary and Incomplete–Comments welcome

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1 Introduction

HIV/AIDS is a major cause of death, currently killing about 2 million people worldwide each year. The number of new infections is even higher than 2 million, suggesting an even more severe problem in the future. The most affected continent is Africa which hosts about two thirds of all HIV/AIDS infected people. Within Africa most transmissions occur through heterosexual sex. Further, more than half the HIV positive population is female, compared to less than one third in most developed countries. While much recent attention has been devoted to access to treatment (such as anti-retroviral therapy), Canning (2006) argues that prevention policies are by far more cost effective. The obvious question that follow is, what are the most effective prevention policies. While many different prevention strategies are advocated by organizations such as UNAIDS, there is still little consensus regarding what works best, or, in some cases, whether they work at all.

The current paper contributes to this literature by analyzing several different prevention policies in a novel theoretical framework. The set-up is a model of sexual behavior and HIV/AIDS that allows for behavioral adjustments in response to policy changes as well as general equilibrium effects. The model explicitly models men and women, thereby allowing for gender asymmetries in disease transmission. The model is applied to the case of Malawi. Malawi is an African country with a relatively high HIV/AIDS rate and very good data related to HIV/AIDS and sexual behavior. The main finding of the analysis is that most policies have the potential to backfire. In particular, small policy changes may lead to an increase in the HIV/AIDS rate caused by the behavioral responses.

The framework used to study the Malawian HIV/AIDS epidemic is an equilibrium search model that accounts for the main decision margins by which a person can affect the spread of the epidemic. The main benefit that economics can bring to the field of epidemiology is homo economicus. Obvious economic decisions that affect the aggregate prevalence of the disease are: the frequency of sexual activity; whether this activity is with the same partner or with varying ones; whether protective measures such as condoms are used. In the homo economicus framework, these choices are made by rational agents that take into account

the risk associated with different types of sexual activities. Moreover, they are able to infer that a partner who seeks casual sex might carry a different risk than somebody who seeks marriage.

These decision margins are captured in an otherwise simple equilibrium model with three key ingredients. First, there are "markets" for different sexual activities. These markets allow people to have different search behavior towards long- and short-term relationships. Additionally, the market for short-term relationships is split into two: one where condoms are used and the other where they are not. People have differing tastes over various types of relationships. They can choose how intensively to search for a partner on a particular market (or even to abstain). They do this recognizing that some of these relationships will be riskier than others. For example, a short-term relationship using a condom is safer than one that does not. Yet, condoms may fail and hence are not perfectly safe. Associated with each market for a relationship is a price or transfer payment that one of the partner makes to the other. The price adjusts to clear the market. For example, there may be more men desiring to engage in short-term unprotected sex than women. To attract women into this risky market men may have to make some form of transfer payment. This market structure eliminates any joint decision problem between partners about whether or not to use a condom. They have the same desires.

Second, in the analysis a person's past sexual history is private information. Still, the fact that someone does not want to use a condom in a short-run relationship may signal something about their past sexual behavior. In particular, it may indicate a proclivity to engage in risky behavior. Hence, in the analysis, people form rational forecasts about the likelihood of a partner having HIV/AIDS based upon the type of relationship that they are seeking. A person can then forecast the odds of getting HIV/AIDS if s/he engages in a particular type of relationship.

Third, the odds of having HIV/AIDS depends on one's past sexual history. In the analysis an individual rationally updates the odds that s/he has HIV/AIDS on the basis of their own behavior. This influences their current decisions about how to direct their search. People

who believe that they have HIV/AIDS may be more likely to participate in risky behavior than those that do not because they think that they have little to lose. This channel will worsen the health of participants on the short-term market for unprotected sex and amplify the risk of participation.

To address the HIV/AIDS epidemic in Malawi, the model is analyzed numerically. This is done in two steps. First, a benchmark simulation is constructed that displays features that are broadly consistent with the Malawian case. In particular, the simulated model has a HIV/AIDS infection rate that corresponds with the Malawian data, the proportion of sexual encounters that is casual is approximately the same, and the fraction of these encounters that use a condom is reasonable. It should be noted up-front, given the complex nature of the model, that an informal benchmarking procedure is adopted. Second, the benchmark model is then used to analyze some topical policy experiments, such as circumcision or the promotion of marriage. At this early stage in the research program using computational general equilibrium models, the upshot of these experiments cannot be taken as evidence in favor or against any particular policy; to do so on such an important issue would be reckless. They do show that the impact of policy interventions may be very complex. In particular, the HIV/AIDS rate may increase in response to a proposed policy intervention due to the changes in human behavior that they induce and the associated general equilibrium effects.

For example, circumcising males may make them less prone to catching the HIV virus. The lower risk could cause men to engage in more risky sexual behavior. As a consequence, the rate of HIV/AIDS in the female population may actually rise. This indeed occurs with non-trivial magnitudes in our benchmark model. Due to the equilibrium nature of these effects, they will not be picked up in field experiments unless a substantial mass of the male population in the location of the experiment are circumcised. Likewise, policies promoting marriage may have unintended consequences if risky types are brought into matrimony. This lowers the relative safety of marriage. It also decreases the relative risk of casual sex. As a consequence, unprotected sex increases. It turns out that the overall rate of HIV/AIDS in society may rise. Yet, at the extreme, if *everyone* married and remained faithful there

could not be a HIV/AIDS epidemic. Therefore, the response to such a policy may exhibit nonmonotocities depending on the strength of the marriage effect.

The remainder of this paper is organized as follows. The next subsection discusses the theoretical literature on HIV/AIDS. Section 2 provides background information on sexual behavior and HIV/AIDS in Malawi. Section 3 sets up the economic environment, while Section 4 defines the equilibrium. Section 5 describes the benchmark parametrization of the model, and Section 6 presents the results of the policy experiments. Section 7 offers some concluding remarks.

1.1 Relationship to the Literature

The work-horse epidemiological model of disease transmission is the susceptible-infected (SI) model with random mixing; see, e.g., Anderson and May (1992). In such a model people are in either one of two states: infected or susceptible. If a person is infected he transmits the disease to susceptible (non-infected) people until he leaves the sexually active population. In models of HIV/AIDS there is no stage of recovery.[1] In these models people encounter other individuals in the population randomly. Most epidemiological models take the number of encounters, i.e., the number of sexual partners, as exogenously given. The assumption that individuals do not change their behavior in response to their environment–in particular in response to the overall prevalence of the disease–is problematic for human populations. Surveys suggest that people react to higher presence of HIV/AIDS by adjusting the number of partners, the type of sexual relationships, and the protective measures that they use [e.g., Wellings et al. (1994)].

Kremer (1996) augments the basic SI model by determining the number of partners endogenously as a choice by rational agents that depends on the prevalence of the disease. He focusses on the theoretical implications of such endogeneity. He shows that a reduction

[1] For other infectious diseases recovery and, either resistance against further infection, or the possibility of reinfection, are explicitly modeled. See Hethcote (2000) for a comprehensive overview of mathematical modeling of infectious disease.

in the transmission probability can increase the overall prevalence rate because individual agents behave in a more risky fashion. Moreover, in a heterogeneous population an increase in the prevalence in the pool of potential partners might make people with low sexual activity more cautious while making high activity people fatalistic, since the latter expect such a high probability of contracting the disease that the marginal increase in infection due to an additional sexual act is small. This might in turn lead to a higher overall prevalence rate.

To derive these predictions theoretically, Kremer (1996) abstracts from a number of issues. People only consider the life-time probability of not contracting HIV/AIDS, but do not care about the exact time they contract the disease. They choose the same behavior in every period of their life and do not update their behavior based on their sexual experience. Condom use is not explicitly modeled, which abstracts from the joint decision problem in using a condom. All relationships have the same length, so a distinction between long-term partners and short-term sex is not possible. And all agents meet randomly. The current paper shows how these richer elements can be modeled, and evaluates quantitatively the impact of these various channels for disease transmission, but at the cost of additional complexity.

Kremer and Morcom (1998) abstract from homo economicus but introduce selective mixing instead of random mixing: Individuals have a higher probability of meeting people like themselves as opposed to others. This idea also features prominently in this paper, but is applied to the type of sexual behavior that a person seeks. For example, an individual that seeks a long-term relationship can search in a way that makes it particularly likely to meet a partner that also seeks a long-term relationship. The special case where people exclusively meet people that are seeking the same type of relationship allows for selective mixing and avoids modeling any conflict of interest in relationship formation. This case will be the focus in this paper. As in Kremer (1996) the current work assumes that other characteristics such as age, past sexual activity and sexual preferences are private information and abstracts from the issue that these characteristics are (imperfectly) observable in real life. Additionally, as in Kremer (1996), attention is restricted to steady states, with the justification that

prevalence rates in Malawi have remained roughly constant in recent years.

Magruder (2008) develops a Jovanovic (1979) style matching model of marital search to model the HIV/AIDS epidemic in South Africa. The idea is that partners enter trial marriages and explore whether or not they are good matches. During this period couples have sex. There is undoubtedly truth to this story. In his setting there is no decision about whether or not to use a condom during these trial marriages. Also, the analysis is not general equilibrium in nature. The decision about whether to accept or reject a partner is not affected by the prevalence rate of HIV/AIDS in society, or by any beliefs that the individual may have about whether or not s/he has the virus based upon their past sexual history. This may be important because healthy (young) individuals might self-select into the safety of marriage while those who believe to be infected have less to gain from safety and might opt for more risky alternatives.

2 Families, Sexual Behavor, and HIV/AIDS in Malawi

The Republic of Malawi serves as a focal country to which the analysis is applied. Therefore, this section briefly describes some information on the HIV/AIDS epidemic in Malawi, together with details about sexual behavior and family life. This background will be useful in guiding the modeling choices. For example, it is argued that it is reasonable to ignore in the analysis mother-to-child transmissions and homosexual sex.

The Republic of Malawi is a country in southeast Africa. It has a population of 14 million people and a land mass of 118,000 square km, making it one of the most densely populated nations in the world. Malawi is heavily affected by HIV/AIDS.[2] Twelve percent of the adult population is currently infected. This is well above the average within Sub-Saharan Africa (SSA), which has an adult prevalence rate of about 7.2%–see Canning (2006). It is also well below the HIV rate of the most affected countries, such as Botswana with an adult prevalence rate of 37%, or South Africa where 22% of all adults are infected. The Malawian

[2] Unless noted otherwise, information on HIV prevalence and patterns of sexual behavior are from the 2004 Demographic and Health Survey's (DHS) Final Report for Malawi.

HIV rate has been roughly constant (ranging between 12-14%) since the mid 1990s, yielding some indication that the disease dynamics have settled into steady-state.

The principal mode of HIV transmission in Malawi is through heterosexual sex. Mother-to-child transmissions are also important, accounting for about 10% of all new HIV infections. However, we abstract from this mechanism in our analysis because most people born with HIV die before they reach sexual maturity (about half of all babies infected during the perinatal period die before their fifth birthday), and therefore do not add to the propagation of HIV. Like in the rest of SSA, more than half of the HIV-infected population in Malawi is female. This is in contrast to two thirds of the infected being male in the Western world–see World Development Indicators (2009). In the West, HIV is largely a disease of homosexuals and drug users. In SSA, by contrast, HIV is a disease that disproportionately affects married (and divorced/widowed) women. The HIV rate among adult women is currently about 13%, compared to 10% among men, suggesting important gender differences. Women are also affected by HIV much earlier in life. For example, 3.7% of women aged 17-19 are HIV+, compared to just 0.4% for men in the same age category.

A rational model of HIV makes only sense if people know what HIV is, and in particular know how it gets transmitted and how to avoid it. This seems largely to be the case in Malawi. Almost 100% of the surveyed Malawians had heard of HIV or AIDS. About 57% of women and 75% of men correctly identified the use of condoms as a means to protect against HIV infection. Finally, an overwhelming majority of adults in Malawi–74% for women and 86% for men–know of a source to get condoms. Finally, Delavande and Kohler (2009) document that people in Malawi are relatively good in assessing their own probability of being infected with HIV. Thus, a rational model of risky sexual behavior seems a reasonable approximation for the Malawian epidemic.

Sexual behavior that allows the spread of the disease is relatively common in Malawi. Condoms are used by less than half of all respondents in their last sexual act–38.7% of women reported using a condom during their last sexual activity, compared to 46.3% of the men. Interestingly, Malawian women have sex at earlier ages than Malawian men. Large age gaps

in sexual relationships are quite common. It is also considered normal for unmarried people to change partners often. A female teenager said in an interview that "[Boys say] 'Do you just eat vegetables daily? Sometimes, you change [your diet]' ... Girls say, 'You don't need to have one cloth [outfit] only.' " (Undie et al 2007). Further, divorce is relatively common. Reniers (2003) reports that 45% of marriages end in divorce within 20 years. The quantitative evidence from the DHS seems to suggest that men engage in more risky sexual behavior than women. However, this difference might be partially due to a social bias as to what is acceptable behavior. Miller, Zulu, and Watkins (2001) analyze gender differences in survey responses in Malawi and find that when husband and wife survey responses contradict each other, the wife is more likely to have said 'no' while the husband is more likely to have said 'yes.' In other words, gender differences in reported sexual behavior have to be interpreted with extreme caution. Several other forms of risky behavior will be abstracted from in our paper. For example, the model does not have concurrent relationships, such as extra-matrial affairs or polygyny, both of which are relatively common in Malawi. For example, in 2004, 8.3% of all married men admitted to having had an affair in the last year. Women admit to much fewer affairs. Note that polygyny is also fairly common in Malawi where, as recent as 2004, 10% of all men had more than one wife. The model abstracts from concurrent relationships to keep it tractable. Future work should include these phenomena.

The high prevalence of risky behavior does not necessarily imply that people are uninformed or irrational: it is more likely due to the trade-off between increased safety versus less pleasure. For example, in an interview about protected sex, a Malawian female said that "You can't eat [candy] while it's in the wrapper. It doesn't taste [good]." (Undie et al 2007). In Malawi, condom use within marriage is essentially non-existent (Chimbiri 2007). One reason is that marital sex is often aimed at reproduction. Furthermore, using a condom in marriage may be interpreted as a signal of infidelity. Bracher, Santow and Watkins (2004) write that "in essence, using condoms within marriage is a sign that it is 'not a real marriage' " and quote a Malawian saying "she does not protect herself with her husband, for it is marriage." Note also that while using a condom lowers the transmission

risk substantially, it does not decrease the risk to zero. Bracher, Santow and Watkins (2004) cite a study which finds that for new condoms, the average breakage rate is 4%; this rate jumps to 19% for condoms that are 7 years old. The higher breakage rate may, in fact, be the more relevant figure in the context of Malawi since condom quality degrades faster in tropical climate where the temperature often exceeds the recommended storage temperature of 25 degrees Celsius.

Poulin (2007) documents that money and gift transfers in sexual partnerships are part of the courting practices of young Malawian women and men. In addition to the expression of love and commitment, she argues that these transfers are a way of acquiring sex for men and about meeting their financial needs for women. A gift might be in the form of sugar or soap, but also in cash. Men who give gifts expect to be receiving sex, and they expect it sooner rather than later. Transfers are not made directly before or after sex (as with prostitution), but rather gift giving is an integral part of a relationship that may depend on the need (e.g. for soap) of the recipient as well as the availability of cash for the giver. Similar evidence is also given in Swidler and Watkins (2007). The model we develop will allow for such transfers between men and women in sexual relationships.

Finally, note that both testing and treatment (such as anti-retroviral drugs) have been fairly uncommon in Malawi until very recently. Testing was introduced in 2004 within the context of the MDICP. This has lead to 2,686 women and 2,581 men being tested (as of the publication of the 2004 DHS Final Report on Malawi), providing the first national population-based HIV prevalence estimate for Malawi.

3 Economic Environment

Imagine a world populated by males and females. Males and females desire relationships with the opposite sex. There are two types of relationships, viz short-term and long-term ones. Within a relationship individuals engage in sex. Sex is risky because of the presence of the HIV/AIDS virus in society. There are two types of sex, protected and unprotected. Protected sex offers a better defense against the transmission of HIV/AIDS across partners.

It provides less enjoyment, though. Individuals interested in a short-term relationship must decide what kind of sex they desire. Put simply, they must weigh the extra momentary utility associated with unprotected sex against the increased odds of being afflicted with the HIV/AIDS virus in the future. Sex is always unprotected in long-term relationships. Suppose that a person can only engage in one relationship at a time.

Denote the utility from unprotected sex by u and the utility from protected sex by p, with $u \geq p > 0$. Additionally, there is a utility flow from a long-term relationship that will be denoted by l, which may be negative. Individuals also realize utility from the consumption of goods. Let this utility be given by $c^{1-\sigma}/(1-\sigma)$, where c is consumption and $\sigma \geq 0$. Each period a person receives income in the amount y. There is no borrowing or lending in the economy. An individual discounts the future at a stochastic rate that takes two values, viz ι and β with $\iota \leq \beta$. Some individuals start off life with the low rate ι. This low rate reflects the impatience of youth, which may lead to a predilection to engage in risky behavior. A person may then switch permanently to the high rate with probability η. These two discount factors reflect the probability of dying from a natural death each period given by δ; that is, $\iota = \widetilde{\iota}(1-\delta)$ and $\beta = \widetilde{\beta}(1-\delta)$ where $\widetilde{\iota}$ and $\widetilde{\beta}$ are the underlying subjective discount factors. The values of l, p, u, and y may differ across individuals of a given gender in accordance with some underlying type parameter denoted by x.

At the beginning of each period an unattached individual may search for a long-term partner. The odds of finding a partner on the long-term market are denoted by π_l. The individual can pick these odds at an increasing cost in terms of lost utility. These costs are given by $C(\pi_l) = \omega[\pi_l/(1-\pi_l)]^{\kappa+1}$, where $\kappa \geq 0$ and $\omega > 0$. Observe that $C(0) = 0$ and $C(1) = \infty$. A long-term relationship may break up (at the end of) each period with exit probability ξ. If the person is unsuccessful at finding a long-term mate s/he then enters the short-term market. Note that an individual who does not want a long-term relationship can set $\pi_l = 0$. If the person wants a short-term one, then s/he must decide whether to have one involving protected or unprotected sex. Let π_p and π_u represent the odds of finding a partner in protected and unprotected markets for short-term relationship, which will be

choice variables. The cost of searching in each market is given by $C(\pi_p)$ and $C(\pi_u)$, so that the total cost of searching for a short-term partner will be $C(\pi_p) + C(\pi_u)$. Assume that individual will not simultaneously draw a partner on both markets. The odds are therefore constrained by $\pi_p + \pi_u \leq 1$, and an individual will be abstinent with probability $\pi_a \equiv 1-\pi_p-\pi_u$. Also, observe that individuals can choose abstinence by picking $\pi_p = \pi_u = 0$.

Associated with each market is a transfer payment, t, that is made between the two partners. For the person receiving the transfer, t will be positive, while it will be negative for the individual making it. Think about the people receiving the transfers as supplying relationships on the market, and those paying transfers as demanding them. Interpret the transfer as representing the inputs into a relationship: affection, entertainment, gifts, etc. The magnitude of this transfer is determined in competitive equilibrium. The size of it will depend upon the demand and supply for a given type of relationship by each gender. This will hinge on the utility that each gender realizes from a partnership in the various markets and the riskiness of participating in them.

A person enters a period with a prior belief about the likelihood of not being infected with HIV/AIDS. Denote this prior belief by ϕ, which is private information. The person then may have a relationship involving either protected or unprotected sex. The risk of catching HIV/AIDS from an infected person is different for the two kinds of sex. If the individual has sex with an HIV/AIDS infected person then the virus will get transmitted with probability $1-\gamma$, where γ differs across the types of sex. The transmission probability is lower for protected sex vis à vis unprotected sex. A person who is inflicted with HIV/AIDS will typically not show symptoms for a while, in which case he cannot distinguish his health state from a person who is not infected. If symptoms occur, they mark the severe part of the illness. The person and others in his surroundings will know that he is ill. Assume that an infected person will develop symptoms each period with probability α. At the end of a period, a person updates his prior in Bayesian fashion depending upon: (i) the type of relationship he was in; (ii) whether or not he observed symptoms in himself or his partner. Let the expected lifetime utility for a person with the symptoms of HIV/AIDS

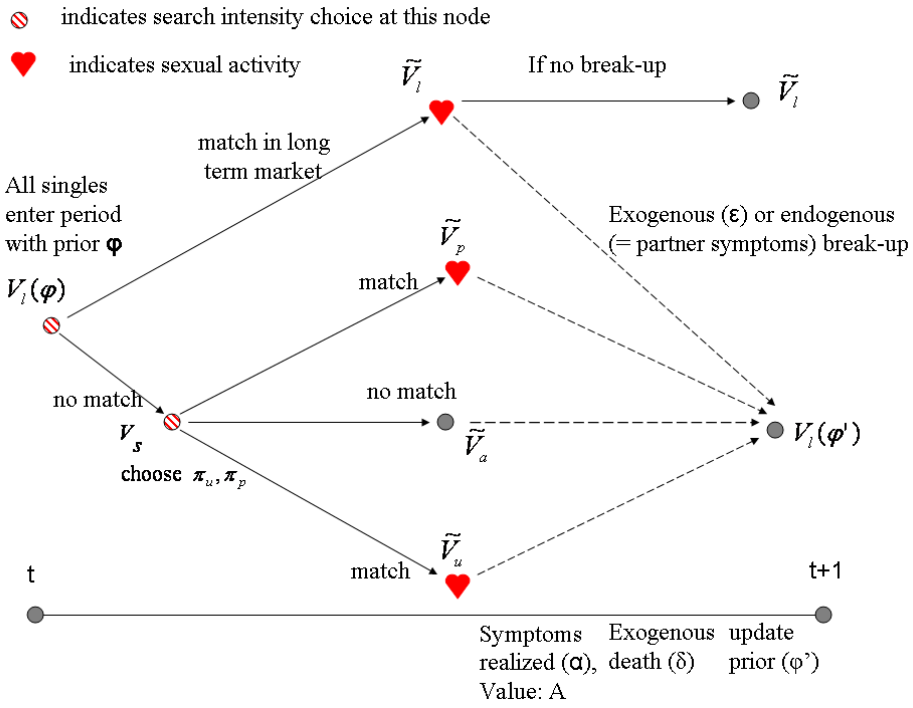

Figure 1: Timing of events.

be represented by A. Assume that a person stricken with HIV/AIDS symptoms engages in no further relationships. The probability that a person displaying symptoms dies is δ_2. Since person with HIV/AIDS symptoms engages in no further activity, δ_2 does not appear in the value functions. However, it is relevant for computing the average HIV/AIDS rate in socity. Note that in the framework there is an attrition in the population each period both due to natural death and to HIV/AIDS. This loss is replenished by an inflow each period of newly born males and females. Recall that x denotes the set of permanent characteristics for an individual, say l, p, u, and y. People also differ by gender. Gender will be suppressed unless it is specifically needed and then it will be represented by the subscript g (for $g = f, m$) attached to a function or variable. Assume that μ type-x individuals are born at the beginning of each period. Figure 1, portrays the timing of events.

Before proceeding on to the formal analysis some notation will be defined. An individual

will be indexed by his prior that he is healthy, ϕ, his discount factor, d, and his exogenous type x. Let $\widetilde{V}_r^d(\phi, x)$ denote that lifetime utility for a person with prior ϕ, a discount factor $d = \iota, \beta$, and an exogenous type x who just found a partner for a relationship of type $r = a, l, p, u$ (abstinent, long-term, short-term protected and short-term unprotected). Similarly, $V_r^d(\phi, x)$ will represent the expected lifetime utility for a person who is currently searching for a partner in a type-r relationship (for $r = l, s$ where s denotes short term), but has not found one yet. Attention will now be directed toward the determination of the functions $\widetilde{V}_r^d(\phi, x)$ and $V_r^d(\phi, x)$. The focus will be on studying a stationary equilibrium for this setting.

3.1 Short-term Relationships

3.1.1 Abstinence

The case of abstinence is the easiest to analyze. So, start here. To this end, imagine a type-x person with a high discount factor, β, who has failed to match on the short-term sex markets. Thus, he will be abstinent for the current period. The individual's discount factor will now remain high forever.

The recursion for a type-x individual with prior ϕ who is currently abstinent is given by

$$\begin{aligned} \widetilde{V}_a^\beta(\phi, x) &= \frac{y^{1-\sigma}}{1-\sigma} \\ &\quad +[1-(1-\phi)\alpha]\beta V_l^\beta(\phi', x) + (1-\phi)\alpha\beta A, \end{aligned} \tag{1}$$

with

$$\phi' = \Phi_a(\phi).$$

The first term on the righthand side of (1) gives the person's momentary utility from his current consumption. Two things can happen next period, as the next two terms illustrate. Now, even though the individual doesn't have sex in the current period, and is symptom free, he may still develop the symptoms of HIV/AIDS next period because of past relationships. He starts the current period with a prior, ϕ, about his probability of being non-infected. Therefore, he believes that he will become infected next period with probability $(1-\phi)\alpha$.

In this event the person will realize an expected utility level of A, which is discounted at rate β. This explains the third term on the righthand side of (1). Likewise, he will remain healthy next period with odds $1-(1-\phi)\alpha$. In this situation, the person will enter the long-term market next period and search for a mate. Also, when the individual does not suffer the symptoms of HIV/AIDS, he updates his prior to ϕ' in line with the function (2). The discounted expected utility from searching on the long-term market next period with prior ϕ' is given by $V_l^{\beta}(\phi', x)$. This accounts for the second term in the equation. The function $\Phi_a(\phi)$ specifies how the individual will update his prior for next period, contingent upon not observing the symptoms of HIV/AIDS in the current period. Note that if one shows the symptoms of HIV/AIDS in the current period then it is known with certainty that one has the virus. The form of the updating function is discussed now.

The function for updating the prior, $\Phi_a(\phi)$, is given by Bayes rule:

$$\begin{aligned} \Phi_a(\phi) &= \frac{\Pr(\text{not being infected this period}|\phi)}{\Pr\left(\text{not observing any symptoms this period}|\phi\right)} \\ &= \frac{\phi}{\phi+(1-\phi)(1-\alpha)} = \frac{\phi}{1-(1-\phi)\alpha}. \end{aligned} \tag{2}$$

The prior probability of not being infected is ϕ. The probability of not observing any symptoms has two components: the odds of not having HIV/AIDS, ϕ, and the odds of being infected but not showing symptoms, $(1-\phi)(1-\alpha)$. As will be seen, the formula for updating can become complicated when one has sex. Here, the odds of transmission from the partner must be taken into account. Additionally, in a long-term relationship there is valuable information contained in a partner's health status.

Next, consider the case of an abstinent person with a low discount factor, ι. The discount factor may switch next period to the high value, β, with probability η, or remain at the low one, ι, with probability $1-\eta$. It is easy to see that the recursion for a type-x person with a low discount factor, ι, and a prior ϕ, will now read

$$\begin{aligned} \widetilde{V}_a^{\iota}(\phi, x) &= \frac{y^{1-\sigma}}{1-\sigma} + [1-(1-\phi)\alpha]\iota[\eta V_l^{\beta}(\phi', x) + (1-\eta)V_l^{\iota}(\phi', x)] \\ &\quad +(1-\phi)\alpha\iota A, \end{aligned} \tag{3}$$

with $\phi' = \Phi_a(\phi)$.

3.1.2 Sexual Relationships

Now, suppose that the individual is matched in a short-term relationship. Again, start with the situation where the person has a high discount factor β. If $s = p$ then the person will realize the utility p from his relationship. If $s = u$ the individual will enjoy u. Define the indicator function $I(s)$ to return a value of 1 when $s = p$, and a value of 0 otherwise. Thus, the joy from a short-term sexual relationship can be written as $pI(s) + u[1 - I(s)]$. The cost of sex on the two markets differs for two reasons. First, the transmission risk of catching HIV/AIDS from an infected person differs across markets. Specifically, the transmission risk in the protected market, $1 - \gamma_p$, is lower than in the unprotected one, $1 - \gamma_u$. Second, the average level of healthiness in the pool of participants in the two markets will in general differ. The fact that person doesn't want to use a condom signals something about their past tendencies to engage in risky behavior. In light of this, $\bar{\phi}_s$ gives the odds that a randomly drawn partner on the short-term market s (for $s = p, u$) does not have the HIV/AIDS virus. Given his prior, ϕ, the individual believes that he will suffer the symptoms of HIV/AIDS next period with probability $\alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)]$. Symptoms can arise from two potential sources. The person could already have the virus and the symptoms materialize. The odds of this event are $\alpha(1-\phi)$. Or, the person can catch the virus from his current partner, and then the symptoms appear, an event that occurs with probability $\alpha\phi(1 - \bar{\phi}_s)(1 - \gamma_s)$. The odds of not suffering the symptoms of HIV/AIDS next period are then just $1-\ \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)]$.

The recursion for a type-x individual with prior ϕ who is currently having a short-term relationship in market s is given by

$$\begin{aligned} \widetilde{V}_s^{\beta}(\phi, x) &= \frac{(y - t_s)^{1-\sigma}}{1 - \sigma} + pI(s) + u[1 - I(s)] \\ &\quad + \{1 - \alpha[(1 - \phi) + \phi(1 - \bar{\boldsymbol{\phi}}_s)(1 - \gamma_s)]\}\beta V_l^{\beta}(\phi', x) \\ &\quad + \alpha[(1 - \phi) + \phi(1 - \bar{\boldsymbol{\phi}}_s)(1 - \gamma_s)]\beta A, \end{aligned} \tag{4}$$

with

$$\phi' = \Phi_s(\phi), \text{ for } s = p, u. \tag{5}$$

The function $\Phi_s(\phi)$ specifies how the individual will update his prior after having short-term sex, contingent upon not observing the symptoms of HIV/AIDS in the current period. The form of this function is discussed below. Recall that the person's exogenous type, x, determines his tastes for protected sex, unprotected sex, short-term relationships, and the level of income. The dependence of p, u, and y on x in the recursions (1) and (4) is suppressed for convenience. Similarly, the gender of the individual has also been omitted. This will be indicated later on by a subscript g for $g = f, m$ (male or female) attached, when relevant, to a variable or function.

Bayes rule now says that the prior should be updated according to the formula

$$\begin{aligned}\Phi_s(\phi) &= \frac{\Pr(\text{not being infected this period}|\phi)}{\Pr\left(\text{not observing any symptoms this period}|\phi\right)}, \text{ for } s = p, u, \\ &= \frac{\phi\bar{\boldsymbol{\phi}}_s + \phi(1-\bar{\boldsymbol{\phi}}_s)\gamma_s}{\phi\bar{\boldsymbol{\phi}}_s + (1-\phi)(1-\alpha) + \phi(1-\bar{\boldsymbol{\phi}}_s)[(1-\gamma_s)(1-\alpha)+\gamma_s]}.\end{aligned}$$

There are two reasons why the individual might not have HIV/AIDS. Perhaps neither him nor his partner have it. The odds of this are $\phi\bar{\phi}_s$. Or, maybe his partner does have it, but it fails to transmit. This will happen with probability $\phi(1-\bar{\phi}_s)\gamma_s$. This explains the numerator. Turn now to the denominator. The individual will show no symptoms in both of these cases. He may also be symptom free even though he actually has the virus. He could initially have the virus yet no symptoms appear, an event that occurs with probability $(1-\phi)(1-\alpha)$, or he could catch it from his current partner but the symptoms fail to materialize, the odds of which are $\phi(1-\bar{\phi}_s)(1-\gamma_s)(1-\alpha)$. Note that this formula presumes that a short-term relationship ends before an individual can observe whether or not his partner is afflicted by the symptoms of HIV/AIDS at the end of the current period.

Again, consider the case when the person has a low discount factor, ι. The discount factor may switch next period to the high value, β, with probability η, or remain at the low one, ι, with probability $1-\eta$. Therefore, the analogue to (4) is

$$\begin{aligned}\widetilde{V}_s^{\iota}(\phi, x) &= \frac{(y-t_s)^{1-\sigma}}{1-\sigma} + pI(s) + u[1-I(s)] \\ &\quad +\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_s)(1-\gamma_s)]\}\iota[\eta V_l^{\beta}(\phi', x) + (1-\eta)V_l^{\iota}(\phi', x)] \\ &\quad +\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_s)(1-\gamma_s)]\iota A,\end{aligned} \tag{6}$$

again with $\phi' = \Phi_s(\phi)$ – for $s = p, u$.

Last, upon entering the market for short-term relationships a person must decide how much effort to expend searching in each market; that is, he must choose π_p and π_u. This is done in accordance with the problem outlined below.

$$V_s^d(\phi, x) = \max_{\substack{0 \leq \pi_u^d, \pi_p^d \leq 1, \\ \pi_u^d + \pi_p^d \leq 1}} \{\pi_p^d \widetilde{V}_p^d(\phi, x) + \pi_u^d \widetilde{V}_u^d(\phi, x) + (1 - \pi_p^d - \pi_u^d)\widetilde{V}_a^d(\phi, x) \tag{7}$$

$$-C(\pi_p^d) - C(\pi_u^d)\}, \text{ for } d = \iota, \beta.$$

The function $V_s^d(\phi, x)$ gives the ex ante value for a type-x individual, with prior ϕ, of entering the market for short-term sex. The solution for search effort is represented by the function $\pi_s^d = \Pi_s^d(\phi, x)$.

3.2 Long-term Relationships

Imagine a person who is currently in a long-term relationship. In a long-term relationship there are no choices to make: there are no affairs, all sex is unprotected, the partnership endures until some form of exogenous breakup occurs, and there is no bargaining over the size of the transfer payment to be made. Suppose that the person entered this relationship n periods ago with prior ϕ. At the end of period n this relationship can breakup for three basic reasons (besides natural death).[3] First, it could transpire that both people are symptom free and that the relationship terminates due to an exogenous breakup. Second, it could end because the individual's partner becomes afflicted with HIV/AIDS symptoms. Third, the person may develop symptoms and the relationship stops. How should the individual update his prior in each of these three cases?

3.2.1 Updating in a Long-term Relationship

Take the case where the individual is exiting a long-term relationship at the end of period n where both partners are symptom free (or appear to be healthy, h). Here, the individual

[3] For simplicity assume that both partners die together; i.e., with probability δ the pair dies, and with probability $(1 - \delta)$ both survive.

should update his prior according to the rule

$$\begin{aligned}\Phi_l^h(\phi, n) &= \frac{\Pr(\text{not being infected } \textit{and} \text{ partner showing no symptoms at end of period } n|\phi)}{\Pr(\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi)} \\ &= \frac{\phi\bar{\phi}_l + \phi(1-\bar{\phi}_l)\gamma_u^n(1-\alpha)^n}{\Delta^h}, \end{aligned} \tag{8}$$

with

$$\begin{aligned}\Delta^h \equiv\ & \phi\bar{\phi}_l + (1-\phi)(1-\bar{\phi}_l)(1-\alpha)^{2n} \\ & +[\phi(1-\bar{\phi}_l) + (1-\phi)\bar{\phi}_l](1-\alpha)^n[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j} + \gamma_u^n].\end{aligned}$$

where $\bar{\phi}_l$ is the (time-invariant) probability that a randomly drawn partner on the long-term market does not have the HIV/AIDS virus.

The first term in the numerator is the chance that neither individual in the relationship initially had the HIV/AIDS virus, which occurs with probability $\phi\bar{\phi}_l$. The second term gives the odds that: (i) the person starts the marriage healthy but that his partner initially had the HIV/AIDS virus, the odds of which are $\phi(1-\bar{\phi}_l)$; (ii) the virus fails to transmit despite having n periods of unprotected sex, which has a likelihood of γ_u^n; (iii) the partner never shows symptoms, which can occur with probability $(1-\alpha)^n$. The denominator includes these two terms in addition to the possibility that either person may have caught the HIV/AIDS virus, but the symptoms have not appeared yet. There are three possibilities to consider here. First, perhaps both partners initially had the virus but no symptoms have occurred yet, which is reflected by the term $(1-\phi)(1-\bar{\phi}_l)(1-\alpha)^{2n}$. Second, there is the situation where the person catches the virus from his partner in any of the n periods, but the symptoms don't emerge in either individual. This occurs with probability $\phi(1-\bar{\phi}_l)(1-\alpha)^n(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}$, where $\phi(1-\bar{\phi}_l)(1-\alpha)^n(1-\gamma_u)\gamma_u^j(1-\alpha)^{n-j}$ is the likelihood that the individual catches the disease exactly j periods after marriage but neither partner shows any symptoms until this period. Third, there is the possibility that the individual did have the virus, initially, while his partner didn't, and no symptoms have occurred. The odds of this happening are $(1-\phi)\bar{\phi}_l(1-\alpha)^n[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j} + \gamma_u^n]$. No symptoms may occur because the

virus didn't transmit (as reflected by the γ_u^n in the brackets) or because it it did transmit at some time but remains dormant [the $(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}$ term].

The second case where the individual's partner develops the symptoms of HIV/AIDS, a, at the end of period n is similar. If a long-term relationship ends because of sickness the individual will obviously observe this. This information should be used in his updating rule, which now reads

$$\begin{aligned}\Phi_l^a(n,\phi) &= \frac{\Pr(\text{not being infected } \textit{and} \text{ partner showing symptoms at end of period } n|\phi\ )}{\Pr(\text{no symptoms in oneself } \textit{and} \text{ symptoms in partner at end of period } n|\phi)} \\ &= \frac{\phi(1-\bar{\boldsymbol{\phi}}_l)\gamma_u^n(1-\alpha)^{n-1}\alpha}{\Delta^a}. \end{aligned} \tag{9}$$

with

$$\begin{aligned}\Delta^a \equiv\ & (1-\phi)(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{2n-1}\alpha + \phi(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{n-1}\alpha[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}+\gamma_u^n] \\ & +(1-\phi)\bar{\boldsymbol{\phi}}_l(1-\alpha)^n\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-1-j}.\end{aligned}$$

Once again focus on the numerator first. The individual can only be healthy, while his partner shows symptoms, if the former didn't have HIV/AIDS initially while the latter did. Furthermore, the virus must have failed to transmit after n periods of unprotected sex. The odds of this happening are $\phi(1-\bar{\phi}_l)\gamma_u^n(1-\alpha)^{n-1}\alpha$. The denominator can be explained in similar fashion to the one in equation (8), with due alternation. The first term, $(1-\phi)(1-\bar{\phi}_l)(1-\alpha)^{2n-1}\alpha$, gives the odds that both people initially had the HIV/AIDS virus, but just the partner shows the symptoms after n periods. It could also happen that the individual doesn't have the virus initially, but his partner does. The chance of this happening, together with just the partner showing symptoms at the end of period n, are given by second term $\phi(1-\bar{\phi}_l)(1-\alpha)^{n-1}\alpha[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}+\gamma_u^n]$. The probability that the partner catches the virus from the individual, and just shows the symptoms at the end of n periods, is $(1-\phi)\bar{\phi}_l(1-\alpha)^n\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-1-j}$. Last, the third case is trivial. If the individual shows the symptoms of HIV/AIDS, then he must have the virus.

3.2.2 The Value of a Long-term Relationship

The value of entering into a long-term relationship, $\widetilde{V}_l^d(\phi, x)$, will now be specified. Start first with a person who has a high discount factor. Note that at the end of each period one of four things can happen: the match can sustain or it may break up for one of the three reasons mentioned above. From this observation it follows that $\widetilde{V}_l^\beta(\phi, x)$ can be written as

$$\begin{aligned}
\widetilde{V}_l^\beta(\phi, x) = & \frac{(y - t_l)^{1-\sigma}}{1-\sigma} + u + l \\
& + \sum_{n=1}^{\infty} \beta^n (1-\xi)^n \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\
& \times [\frac{(y - t_l)^{1-\sigma}}{1-\sigma} + u + l] \\
& + \sum_{n=1}^{\infty} \beta^n (1-\xi)^{n-1} \xi \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\
& \times V_l^\beta(\Phi_l^h(\phi, n), x) \\
& + \sum_{n=1}^{\infty} \beta^n (1-\xi)^{n-1} \Pr[\text{symptoms just in partner at end of period } n|\phi] \\
& \times V_l^\beta(\Phi_l^a(\phi, n), x) \\
& + \sum_{n=1}^{\infty} \beta^n (1-\xi)^{n-1} \Pr[\text{symptoms in person at end of period } n|\phi] \times A.
\end{aligned} \tag{10}$$

This expression looks more complicated than it really is. It will now be explained.

The term on the right-hand side of the first line gives the current utility from the match. The next two lines give the discounted expected utility accruing when the match sustains over the next n periods. For a match to sustain until period $n+1$ there cannot be an exogenous breakup prior to this date. This occurs with probability $(1-\xi)^n$. Additionally, neither party can show symptoms before this date. The formula for the odds that neither partner will show symptoms before period n is presented in Section 9.1 of the Appendix, as are the formulae for the other probabilities shown. The rest of the expression enumerates the expected utilities associated with breakup events in period $n+1$. In particular, the fourth and fifth lines reflects separations due to exogenous breakups. An exogenous breakup will

occur at the end of period n with probability $(1-\xi)^{n-1}\xi$. When this happens the individual enters into single life again. At that time he updates his prior according to the function $\Phi_l^h(\phi, n)$, as given by (8). The individual may also enter into single life because his partner develops HIV/AIDS symptoms in some period n. This is accounted for by the next two lines, six and seven. In this situation the individual will update using (9). Finally, HIV/AIDS may manifest itself in the individual at the end of some period n. This is captured by the last line. Finally, note that the odds of dying from a natural death are incorporated into β.

The value of a long-term relationship for a person with a low discount factor ι is determined analogously. One now must take into account that the discount factor may switch at some future date from ι to β. The expression for $\widetilde{V}_l^i(\phi, x)$ is now slightly more complicated. It is developed in Section 9.2 of the Appendix – see (21). The ex ante value of a long-term relationship for a type-(ϕ, x) person with discount factor d is given by

$$V_l^d(\phi, x) = \max_{\pi_l^d} \left[\pi_l \widetilde{V}_l^d(\phi, x) + (1-\pi_l)\widetilde{V}_s^d(\phi, x) - C(\pi_l)\right], \text{ for } d = \iota, \beta \tag{11}$$

The solution for search effort, π_l^d, is represented by the function $\pi_l^d = \Pi_l^d(\phi, x)$.

4 Stationary Equilibrium

A stationary equilibrium for the developed framework will now be formulated. This involves two steps. First, the equilibrium type distributions for singles will specified. Second, the market-clearing conditions for the various types of relationships will be cast. After this is done, the analysis will conclude with a formal definition of the equilibrium.

4.1 Equilibrium Type Distributions

At the beginning of each period there will be a certain number of single type-x individuals that have a prior ϕ and discount factor d. Let $S^d(\phi; x)$ represent the non-normalized stationary distribution over the prior ϕ for single type-x individuals that holds at the beginning of a period for people with a discount factor d. Some of these people will find a match in the long-term market and exit single life. Others will find a partner in one of the short-term

markets. The rest will remain abstinent. At the end of the period, all singles who either don't die or experience the symptoms of HIV/AIDS will update their priors according to the sexual experiences they just had. Additionally, there will be a flow in of new arrivals. The new arrivals will be made up of two groups: the newly born, and older people whose long-run relationships have broken up. Let $L^d(\phi';x)$ denote the distribution over the prior ϕ' for type-x individuals with discount factor d who exit long-term relationships at the end of a period.

For simplicity, $S^d(\phi;x)$ and $L^d(\phi';x)$ will be represented by discrete distributions on some grid of values for ϕ, even though theoretically speaking they could be continuous ones.[4] The process just described above, which maps this period's singles distribution into the next period's one, will be represented by the transition operator T. In a steady state these distributions will be determined by the fixed point of this operator:

$$(\mathcal{S}^\beta, \mathcal{L}^\beta, \mathcal{S}^\iota, \mathcal{L}^\iota) = \mathbf{T}(\mathcal{S}^\beta, \mathcal{L}^\beta, \mathcal{S}^\iota, \mathcal{L}^\iota). \tag{12}$$

The operator T is characterized fully in Section 9.3 of the Appendix–see equations (22) to (28).

For the probability that a randomly drawn person does not have HIV/AIDS it is now useful to introduce the subscript g (for $g = f, m$) to a function or variable to denote the gender of the person in question. For example, the relevant odds for men in the long-term market is the rate of nonprevalence in the associated pool of females, $\bar{\phi}_{f,l}$, while for females the pertinent odds refer to those in the pool of potential male partners, $\bar{\phi}_{m,l}$. It is readily apparent that the odds of a randomly drawn person of gender g $(= f, m)$ not having HIV/AIDS, who entered into relationship of type r $(= l, p, u)$, are given by

$$\bar{\boldsymbol{\phi}}_{g,l} = \frac{\sum_d \sum_x \sum_\phi \phi \Pi^d_{g,l}(\phi, x) \mathcal{S}^d_g(\phi; x)}{\sum_d \sum_x \sum_\phi \Pi^d_{g,l}(\phi, x) \mathcal{S}^d_g(\phi; x)}, \tag{13}$$

$$\bar{\boldsymbol{\phi}}_{g,p} = \frac{\sum_d \sum_x \sum_\phi \phi \Pi^d_{g,p}(\phi, x)[1 - \Pi^d_{g,l}(\phi, x)] \mathcal{S}^d_g(\phi; x)}{\sum_d \sum_x \sum_\phi \Pi^d_{g,p}(\phi, x)[1 - \Pi^d_{g,l}(\phi, x)] \mathcal{S}^d_g(\phi; x)},$$

[4] They will be countable in equilibrium, as only a finite number of types enter each period, and each type has with probability one only a finite number of possible experiences before exit due to exogeneous death.

and

$$\bar{\phi}_{g,u} = \frac{\sum_d \sum_x \sum_\phi \phi \Pi^d_{g,u}(\phi,x)[1-\Pi^d_{g,l}(\phi,x)]\mathcal{S}^d_g(\phi;x)}{\sum_d \sum_x \sum_\phi \Pi^d_{g,u}(\phi,x)[1-\Pi^d_{g,l}(\phi,x)]\mathcal{S}^d_g(\phi;x)}. \tag{14}$$

4.2 Market-Clearing Conditions

In equilibrium the number of females and males in each type of relationship must exactly balance. Again, using the subscript $g = f, m$ to indicate the gender of the person in question, this implies that the following market-clearing conditions hold:

$$\sum_d \sum_x \sum_\phi \Pi^d_{f,l}(\phi,x)\mathcal{S}^d_f(\phi,x) = \sum_d \sum_x \sum_\phi \Pi^d_{m,l}(\phi,x)\mathcal{S}^d_m(\phi,x). \tag{15}$$

$$\sum_d \sum_x \sum_\phi \Pi^d_{f,p}(\phi,x)[1-\Pi^d_{f,l}(\phi,x)]\mathcal{S}^d_f(\phi,x) = \sum_d \sum_x \sum_\phi \Pi^d_{m,p}(\phi,x)[1-\Pi^d_{m,l}(\phi,x)]\mathcal{S}^d_m(\phi,x),$$

and

$$\sum_d \sum_x \sum_\phi \Pi^d_{f,u}(\phi,x)[1-\Pi^d_{f,l}(\phi,x)]\mathcal{S}^d_f(\phi,x) = \sum_d \sum_x \sum_\phi \Pi^d_{m,u}(\phi,x)[1-\Pi^d_{m,l}(\phi,x)]\mathcal{S}^d_m(\phi,x). \tag{16}$$

Take the second condition, as an example. The left-hand side represents the number of females in short-term relationships involving protected sex. The right-hand side is the number of males. Additionally, a transfer paid by one gender on a market is a transfer earned by the other so that

$$t_{f,r} + t_{m,r} = 0, \text{ for } r = l, p, u. \tag{17}$$

4.3 Definition of Equilibrium

Definition 1 *A stationary equilibrium is described by a set of decision rules for search effort, $\Pi^d_{g,r}(\phi,x)$, a set of transfer payments, $t_{g,r}$, a set of stationary type distributions, $S^d_g(\phi;x)$ and $L^d_g(\phi',x)$, and a set of HIV/AIDS (non)prevalence rates for a partner on each market, $\bar{\phi}_{g,r}$, for all $d = \{\iota, \beta\}$, $g \in \{f, m\}$, $r \in \{l, p, u\}$, such that:*

1. *The decision rules for search intensities, $\Pi^d_{g,r}(\phi,x)$, satisfy the appropriately gender subscripted versions of the generic problems (7) and (11), taking as given transfer payments and HIV/AIDS prevalence rates;*

2. *The stationary type distributions, $S_g^d(\phi; x)$ and $L_g^d(\phi, x)$, solve the appropriately gender subscripted version of (12);*

3. *The prevalence rates for HIV/AIDS on each market, $\bar{\phi}_{g,r}$, are given by (13) to (14);*

4. *The transfer payments, $t_{r,g}$, are such that the markets for all types of relationships clear, or so that equations (15) to (16) hold. Additionally, the flow of transfers across the genders must balance as specified by (17).*

5 The Benchmark Simulation

To address the HIV/AIDS epidemic in Malawi, the model is analyzed numerically. This is done in two steps. First, a benchmark simulation is constructed that displays features that are broadly consistent with the Malawian case. In particular, the simulated model has a HIV/AIDS infection rate that corresponds with the Malawian data, the proportion of sexual encounters that is casual is approximately the same, and the fraction of the these encounters that use a condom is reasonable. It should be noted upfront, given the complex nature of the model, that an informal benchmarking procedure is adopted. Second, the benchmark simulation is then used to analyze some topical policy experiments, such as circumcision or the promotion of marriage. At this early stage in the research program using computational general equilibrium models, the upshot of these experiments cannot be taken as evidence in favor or against any particular policies proposals; to do so on such an important issue would be reckless. They do show that the impact of policy interventions may be very complex. For reasonable parameter values, the response of HIV/AIDS to a proposed policy intervention does not appear to be monotone due to the general equilibrium effects that they induce.

For example, circumcising males may make them less prone to catching the HIV virus. The lower level of risk could cause them to engage in more risky sexual behavior. As a consequence, the rate of HIV/AIDS in the female population may actually rise. This type of effect will not be picked in field experiments, unless a substantial mass of the male population in the location of the experiment are circumcised. Likewise, policies promoting marriage may backfire if risky types are brought into marriage. This lowers the relative safety of marriage. It also decreases the relative risk of casual sex. As a consequence, unprotected sex increases.

It turns out that the overall rate of HIV/AIDS in society may rise. Yet, at the extreme, if *everyone* married and remained faithful there couldn't be a HIV/AIDS epidemic. Therefore, the response to such a policy may exhibit nonmonotocities depending on the strength of the marriage effect. All of this is discussed in the policy analysis.

The analysis now proceeds in the following manner: First, a numerical benchmark will be constructed that captures the most important stylized features of sex, marriage, and HIV/AIDS in Sub-Saharan Africa, or more specifically Malawi. Second, after constructing this benchmark, some policy experiments will be conducted to see what the model predicts about the efficacy of various interventions aimed at curbing the prevalence of HIV/AIDS. These policy experiments are not dispositive in establishing the usefulness of any particular intervention; rather, they illustrate the complex nature of HIV/AIDS problem and uncover the more elusive general equilibrium channels through which countervailing effects reduce the efficacy of some interventions.

5.1 Parameterization

At this stage in the development cycle, the model is too complicated to estimate. It has many parameters. Plus, for a given set of parameter values the model takes some time to run. The solution process is often a bit temperamental. While the simulations recover only a single interior equilibrium some additional boundary equilibria may exist. This and some other algorithmic issues require monitoring when running the computer program and occasionally some intervention is required.[5] This makes an automated estimation process difficult. Therefore, a more informal approach is taken to construct the benchmark simulation. This is done in two steps. First, some parameters have direct data analogs and are thus easy to pin down. Second, the remaining parameters are chosen to match, roughly, some key observations related to the HIV/AIDS epidemic in Malawi. The data mostly obtains from the 2004 Demographic and Health Survey (DHS) that was conducted in Malawi. The facts

[5] Define an equilibrium to be interior if all markets attract a positive measure of agents. Call all others boundary equilibria. Clearly, only interior equilibria have a chance of matching the data.

garnered from Malawi are often complemented with data from other sources, as necessary. A complete list of the parameters and their benchmark values is given in Table 6 in the Appendix.

The most important parameter values for the simulation are those concerning HIV/AIDS. Fortunately, for the most part, these can be taken from medical literature. Take the period to be one quarter. The transmission risk obviously depends on whether or not a condom is used. Even when a condom is used, however, protection is far from perfect–Bracher, Santow and Watkins (2004). In line with Bracher, Santow and Watkins (2004) suppose that condoms are not used in marriage; i.e., all marital sex is unprotected.[6] Further, in accord with the medical evidence, females are assumed to have a higher risk of contracting HIV than males.[7] Concretely, set the quarterly non-transmission risks equal to $\gamma_p^m = 0.98$, $\gamma_p^f = 0.95$, $\gamma_u^m = 0.94$, and $\gamma_u^f = 0.91$, where γ denotes the non-transmission rate, the subscript indicates the type of sex, and the superscript the gender of the person facing the specified transmission risk. These numbers are in step with what is found by Grey et al (2001) who calculate an infection risk of 0.0011 per sexual encounter. They further report an average of 9 sexual activities per month (for a couple). This would imply a quarterly risk of 3%. Note that during the course of the studies condoms were distributed freely (and sometimes used), therefore the number has to be interpreted as a convex combination between protected and unprotected sex. Based on data from Uganda, Wawer et al (2005) report even higher numbers. Depending on the stage of the disease, they find transmission rates of 0.008, 0.0007, and 0.003 per sex act. Note that the higher male-to-female transmission risk is the only exogenous gender difference fed into the benchmark simulation described in Section 5.2. As is discussed in greater detail in Section 5.2, this single exogenous gender difference leads to a number of endogenous gender differences in the outcomes, including a higher female HIV prevalence,

[6] Bracher, Santow and Watkins (2004) use qualitative data from journals to argue that "using condoms within marriage is a sign that it is 'not a real marriage.'" For example, a married women commenting on her friend says "She does not protect herself with her husband, for it is a marriage."

[7] See Carpenter et al (1999), Bracher, Santow and Watkins (2004), Nicolosi et al (1994), and Padian, Siboski and Jewell (1991).

females contracting HIV in greater proportion than males at young ages, a gender reversal in HIV/AIDS prevalence at older ages, and females marrying earlier than males. Last, the average time from infection to the outbreak of symptoms is equal to 10 years (DHS 2004). Therefore, let $\alpha = 0.025$; i.e., 40 quarters. The average time from the outbreak of symptoms to death is 2 years (DHS 2004). Thus, pick $\delta_2 = 0.125$; i.e., 8 quarters.

Some other parameters values that can also be pinned down using a priori information. Set the quarterly divorce hazard equal to $\xi = 0.03$. Bracher, Santow and Watkins (2004) report that 26.4% of all marriages in Malawi end in divorce within the first five years. Assuming a constant annual divorce hazard, this would imply a quarterly risk of 1.56%.[8] There are two reasons why a rate is used that is twice this number. First, polygyny is fairly common in Malawi, from which the analysis abstracts. Second, extramarital affairs are relatively common as well. Therefore, interpret, for example, a 10-year marriage with one affair as two long term relationships with a third casual one in between. Section 6.3.2 explores what happens to equilibrium outcomes when the risk of divorce is lower. The quarterly (non-HIV related) death hazard is picked so that $\delta = 0.003$.

A study conducted by the U.S. Census Bureau (2004) estimates that the death probability due to non-HIV related causes is 1.2% annually in Malawi. This translates to a quarterly death probability of 0.3%. Let quarterly income per person be equal to \$300 so that $y = 300$. According to the 2003 World Development Indicators, gross national income per capita in Malawi in 2001 was \$560 (PPP adjusted). After adjusting for the fact that only about 50% of the Malawian population is of working age and also for the different time horizon (a quarter rather than a year), a quarterly income of \$300 seems reasonable.

The remaining parameters have no clear data analogs. These parameters are picked to match (via the eyeball metric) several facts related to sex, marriage and HIV/AIDS in Malawi. A list of the parameters and their values is given in Table 6. For example, utilities from the different types of sexual relationships are free parameters, constrained only by $p \leq u$; i.e., people enjoy unprotected sex more than protected sex. To ensure interior

[8] A similar number is also reported by Reniers (2003).

solutions, the model also specifies that people enjoy variety in their sexual partners, and so marriage (sex with one partner) decreases overall utility from unprotected sex ($l < 0$).

To keep things simple, assume that there are only two exogenous types of males and females. The only exogenous heterogeneity (in addition to the gender difference in transmission risk) is the degree of patience people have. Assume that young people are more impatient then old people and that there a small subset of the population that is extremely impatient. Therefore, there are four different discount factors for each combination of age and type. These are denoted by $\widetilde{\beta}$ and $\widetilde{\iota}$ in Table 6. The subscripts 1 and 2 stand for the two types. Assume that the smaller impatient group is only 10% of the population (this follows from $\mu_1 = 0.01$ and $\mu_2 = 0.001$). The values reported in the table are "pure" discount factors; i.e., net of mortality risk.

5.2 The Benchmark Model vs. The Data

So, what are the data targets and how well does the benchmark simulation match them? This question will now be addressed. The upshot of the analysis is that benchmark simulation delivers results that are qualitatively in line with some key features of the Malawian HIV/AIDS epidemic. To begin with, as can be seen from Table 1, the HIV/AIDS prevalence rate predicted by the model is 12.3%, close to the 11.8% that is reported in the data.[9]

Moreover, the experiment captures the gender difference in HIV/AIDS infection rates, with females experiencing an HIV/AIDS rate that is 2 percentage points higher than that for males (13.3% versus 11.4%). This captures a large fraction of the 3 percentage point difference seen in the data (10.2% versus 13.3%).

In addition to qualitatively matching moments on HIV/AIDS prevalence, the benchmark experiment is also broadly consistent with the data on other aspects of sexual activity. In the model casual or short-term sex as a fraction of all sexual encounters is 16%, very close to the 18% of sex that occurs outside of a union that is reported in the data. For those who engage in casual sex, the model predicts that a quarter of them uses a condom. This is less than

[9] All data sources for the figures and tables are discussed in the Data Appendix.

the 39% seen in the data, but is still close. In fact, as people have been found to overstate the amount of protected sex they have [see Allen (2003)], these two numbers may be closer in reality than first meet the eye. The next two rows in Table 1 report the fraction of singles who had casual sex in the last quarter and in the last year. These statistics are different from the fraction of all sexual activity that is casual both because (all) married people have sex and because some singles are abstinent. Singles in the model have more casual sex than their real-life counterparts, but again, it is possible that people systematically under-report their risky sexual behavior. Finally, the fraction of the population that die from natural causes is comparable across model and data (64 versus 54%).

What is probably not misreported is people's marital status. The next-to-last row in Table 1 lists the fraction of singles in the entire population. The model predicts this fraction to be 28%, close to 33% observed in the data. Figure 2 plots the percentage of the population that has ever been married by certain ages. Note that the benchmark experiment captures two stylized facts observed in the data. The first is that women marry much earlier than men–in the model, 85% of women are married by age 21, whereas only 40% of men are married by that age. This fraction in the data is approximately 88% and 48%, respectively. However, as is only reasonable, men eventually "catch up", and most of the population is married by their mid to late 20s. This may be interpreted as being reflective of the "sugar daddy" phenomenon seen in Malawi where young girls enter into relationships with older men.[10]

In summary, singles in the model are somewhat more likely to engage in risky sexual behavior than in the data–they are more likely to both have short-term sex and have such sex without using condoms. However, there are slightly fewer singles in the model than in the data. Consequently, the model-predicted HIV/AIDS infection rate is remarkably similar to that found in the data.

[10] See Dupas(2009) and Luke (2002) for documentation of such a phenomenon.

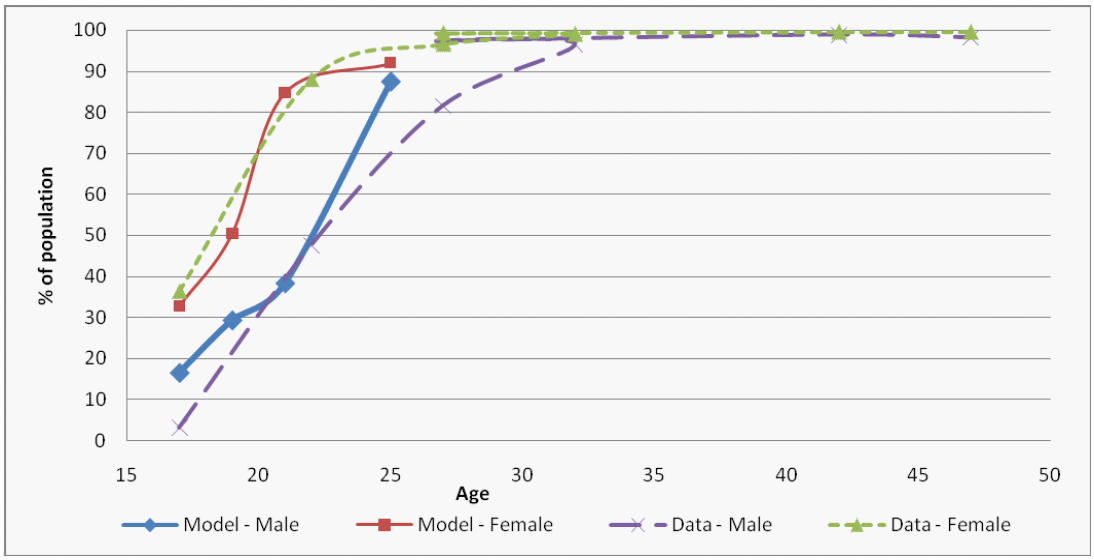

Figure 2: Fraction Ever Married, by Age and Sex–Data and Model

TABLE 1: STYLIZED FACTS ON HIV/AIDS IN MALAWI

Observation	*Data*	*Model*
HIV/AIDS rate	11	12
–Males	10	11
–Females	13	13
Fraction of all sex that is casual	18	16
Condom use for casual sex	39	25
% singles that have casual sex	16 (per month)	51 (per quarter)
% singles that have casual sex (1 year)	37	60
% singles	33	28
% married by age 21		
–Males	48 (20-24 yrs)	38
–Females	88 (20-24 yrs)	84
% people that die from natural causes	54	64

5.3 Non-Targeted Observations

The benchmark model generates some other predictions that were not targeted when picking values for parameters. Figure 3 plots HIV/AIDS prevalence by age.[11] Because agents in the model become sexually active earlier than is observed in the data, the model predicts a life-cycle HIV/AIDS infection pattern that is shifted left (to younger cohorts) compared to that seen in the data. The model and data do agree on a hump-shaped infection pattern, though. That is, HIV/AIDS prevalence initially rises as one moves from younger to older cohorts. Prevalence eventually peaks and then drops, however, so that the oldest cohorts have lower HIV/AIDS infection rates than younger cohorts. This pattern is explained by two observations. First, the rise in HIV/AIDS infection is due to the fact that older people have had more time to be sexually active, and so a larger percentage of the older cohort is infected with HIV/AIDS. People who are infected early in life will die before they make it to old age, though. Put differently, people who have made to it to old age must be those who have engaged in less risky sexual behavior and so are less likely to be infected with HIV/AIDS. This second fact explains the eventual drop in HIV/AIDS prevalence seen in the oldest cohorts. Figure 3 also illustrates the differentiated patterns of infection between the sexes. At younger ages, more females are infected with HIV/AIDS, while at older ages, men are the more infected group. Thus, there is a "crossing" in the prevalence rates for the sexes, and this is also observed in the data.

Recall that agents in the model have rational beliefs about their HIV/AIDS status. Figure 4 compares such beliefs against those reported by Delavande and Kohler (2009), who elicited responses on beliefs from rural Malawians. In both the data and model, there is a steep drop between the number of people who believe themselves to be almost certainly HIV/AIDS-negative–say less than a 5% probability of being infected–and the people who are only relatively sure that they are negative–say close to a 15% probability of being infected.

[11] In the model, agents' lives are simulated up to age 35.

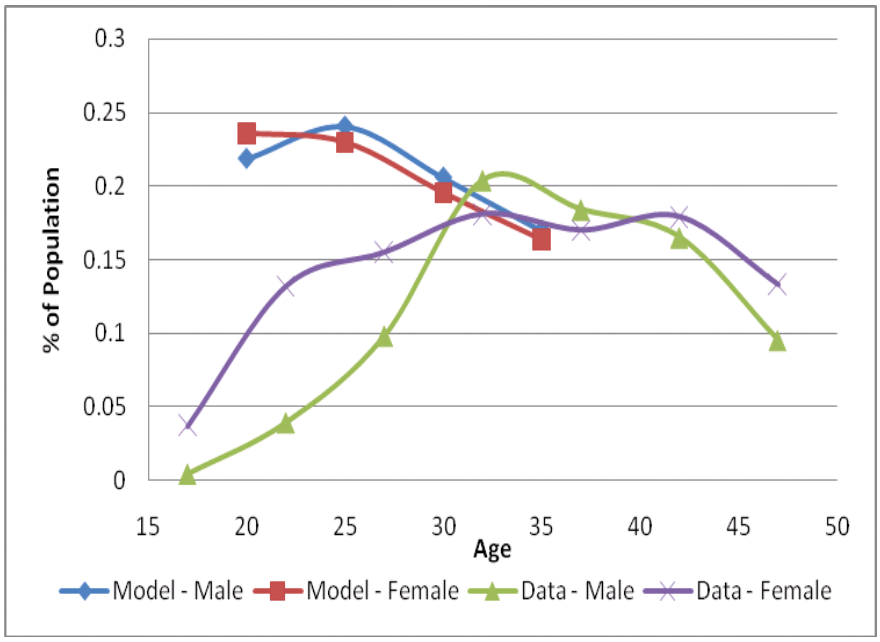

Figure 3: Fraction with HIV/AIDS, by Age and Sex–Data and Model.

After this drop, beliefs are flat throughout the rest of the distribution, a pattern confirmed by Delavande and Kohler (2009). Moreover, since Delavande and Kohler (2009) finds that the prevalence rates implied by the reported beliefs roughly match the actual prevalence rates, their work is supportive of the modeling assumption that agents are rational.

6 Policy Experiments

The model is now ready to explore the effectiveness of various policies intended to curb the spread of HIV/AIDS. A caveat is in order before proceeding. Research using computational general equilibrium models to assess the implications that interventions might have on the spread of HIV/AIDS (or other diseases) is still in its infancy. The goal of obtaining hard numbers that can be used for policy analysis is still some way down the field. The simulations do illustrate potential pitfalls in the efforts to limit the disease, however. Specifically, as will be illustrated, moderate policy interventions have the potential to backfire due to the shifts

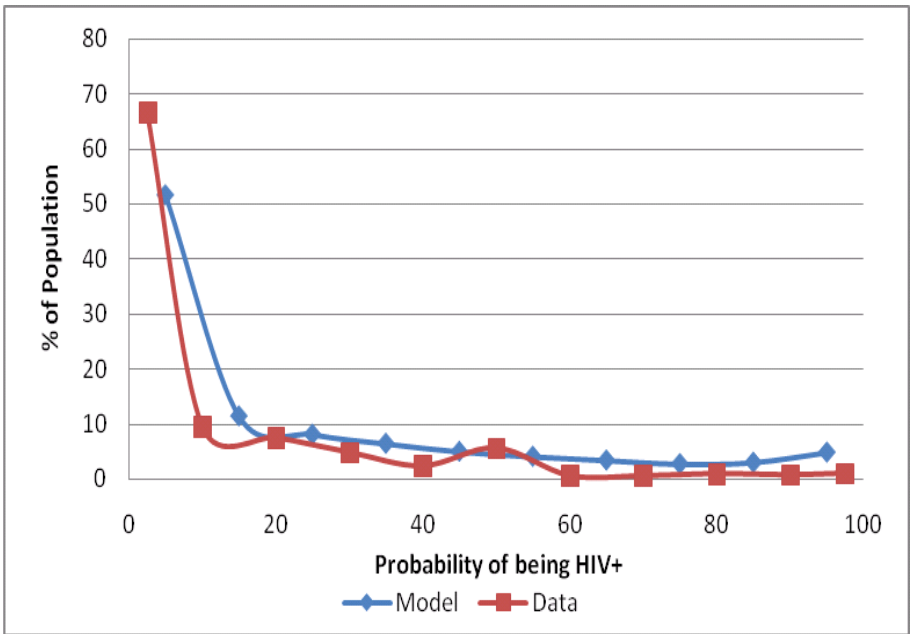

Figure 4: Distribution of Beliefs about the Odds of being Infected with HIV/AIDS–Data and Model.

in sexual behavior that they induce. These shifts in sexual behavior then feed back on the equilibrium rate of HIV/AIDS. In principle, computational general equilibrium models are well suited to analyze such effects. In practice, the best structure to be employed needs to be determined. A prototype structure is offered here. This section first gives a (non-exhaustive) overview of the policy-related HIV/AIDS literature and briefly discusses some aspects where equilibrium models can shed some additional light. Then, three different policies are explored using the model: male circumcision, the treatment of (other) STDs, and the promotion of marriage.

6.1 Literature

There are two broad classes of policies surrounding HIV: treatment and prevention–see Canning (2006) for an excellent survey. Treatment consists mostly of different generations of anti-retroviral drugs. Prevention includes condom distribution, reducing mother-to-child transmission, mass media campaigns, voluntary counseling and testing, and diagnosis and treatment of other sexually transmitted diseases. Overall, prevention is more cost-effective

than treatment–Canning (2006). Previous studies that analyze the effectiveness of a given policy are typically based on cross-sectional comparisons (e.g., different countries, different regions, or different ethnic groups), on epidemiological simulations, or on randomized field experiments. The approach taken in this paper, which focuses on simulating a choice-theoretic equilibrium model, may bring new angles to the debate. In particular, it allows the analysis of behavioral changes in response to an intervention and the analysis of general equilibrium feedback effects. The findings obtained may be useful for guiding future policy interventions. We now briefly discuss previous findings on policies related to the reduction of the transmission risk as well as the promotion of marriage and faithfulness.

A policy intervention that has received a lot of recent attention is male circumcision. Several studies find a decline in the female-to-male transmission rate for circumcised males. Based on this evidence, UNAIDS now lists male circumcision as one recommended strategy for HIV prevention.[12] The most widely cited evidence comes probably from Gray et al (2007) and Auvert at al (2005). Based on a randomized field experiment in Uganda, Gray et al (2007) find that the incidence of HIV/AIDS (over 24 months) in the treatment group was half the HIV incidence in the control group. Similarly, Auvert et al (2005) find a 60% reduction in female-to-male transmission for those who were circumcised in a randomized field experiment in South Africa. One thing that randomized experiments cannot measure are general equilibrium effects. If all men were circumcised, perhaps the entire village would feel safer and engage in riskier behavior (even if there was no difference in behavior between the treatment and control groups), which would crowd out some of the gains. In fact, Gray et al (2007) find that alcohol use with sexual intercourse (a measure of risky sexual behavior) before the study was 38%, and increased to more than 50% for both the intervention and control groups at the end of the study. Most important, what is typically not measured is the effect that male circumsion has on women. Given that only the female-to-male transmission risk is affected by the procedure, but not the reverse, if treated men change their behavior,

[12] See http://www.unaids.org/en/PolicyAndPractice/Prevention/MaleCircumcision/ accessed on October 7, 2009.

this might have adverse effects on women. The approach taken in the current paper is complementary to randomized field experiments. As will be seen, it allows researchers to assess the general equilibrium consequences of widespread circumcision, and to analyze the implications for both males and females.

Several other policies involve the reduction in transmission risk, albeit in a more symmetric fashion, i.e. affecting both men and women. For example, results of a new vaccine trial were made public very recently showing a 30% efficacy.[13] Another policy is the treatment of other sexually transmitted diseases (STDs) to decrease the transmission of HIV/AIDS. The idea is that the presence of other STDs makes a person more susceptible to contracting HIV. Treating other STDs to reduce transmission risk is in many ways similar to advocating male circumcision, and therefore measuring success is equally problematic. By comparing data from African countries and from the US and Western Europe, Oster (2005) reaches the conclusion that treating other STIs would be an effective policy. Using a "diff-in-diff" approach, she argues that the most likely cause for the different HIV rates are STDs–rather than behavioral differences. However, this conclusion is somewhat problematic. If currently sexual behavior in African countries (where the HIV rate is high) is similar to European countries (where the rate is low), then, this seems to suggest very different attitudes towards risky behavior in the two continents. If this was the case, then reducing the transmission risk by treating other STDs might lead to behavioral adjustments which might crowd out the gains from the reduced risk. Simulating people's behavior in response to the changed circumstances is one way of taking such behavioral responses into account and trying to assess the overall impact of such a policy.

Other policies have been aimed directly at social and behavior change. The so-called ABC approach stands for abstention, being faithful, and the use of condoms–Murphy et al (2006). It is somewhat less clear how such behavior changes can be achieved. One avenue that is pursued are large scale media campaigns aimed at disseminating information. More generally, the emphasis is often on information, education, and communication, which

[13] See Wall Street Journal, September 25, 2009, "Vaccine Shows Promise in Preventing HIV Infections."

is sometimes called the IEC approach. For example Gallant and Maticka-Tyndale (2004) study the effectiveness of education campaigns. One problem with such studies is that success is often measured as changes in attitudes and/or reported behaviors, while it is left open whether actual behaviors changed.[14] In our experiments we analyze the effects of "promoting marriage" where we have several interpretations of what promoting marriage might mean.

6.2 Medical Policies

We now analyze several medical policies that have been recommended recently. Several studies have documented that male circumcision reduces the female-to-male transmission rate but not the reverse–see Gray et al (2007). Other policies, such as the development of a vaccine against HIV and the treatment of other sexually transmitted diseases may reduce the transmission rate for both men and women. We now analyze what the implications of these transmission risk changes might be for the overall HIV prevalence rate, if one takes changes in sexual behavior and general equilibrium effects into account.

6.2.1 Male Circumcision

The numerical simulations point to a potential problem that would not be obviously apparent in studies using other methodologies. While male circumcision may make sex safer for men, it induces them to engage in more risky behavior, putting women at risk. In Table 2 the female-to-male transmission risk, $1 - \gamma_u^m$, is first decreased from 6% per quarter to 4% and then to 3%. As a result, the overall HIV/AIDS rate decreases for men, from 11.4% to 11.1% and then to 8%. As sex gets safer for men, however, prevalence first goes up for women, from 13.3% to 15.8%. As the transmission rate is cut even further the prevalence rate for women falls to 13.2%.

[14] This point is also made in Dupas (2009).

TABLE 2: MALE CIRCUMCISION

	Benchmark	*Safer*	*Safer Still*
γ_u^m	0.94	0.96	0.97
HIV/AIDS Rate–men	11.4	11.1	8
HIV/AIDS Rate–women	13.3	15.8	13.2
Fraction of sex that is casual	15.6	35.4	47.5
% Singles who have causal sex	51.3	68.2	78.6
% of causal sex with condom	24.8	15.4	29.2
% of men who are single	30.4	50.8	60
% of women who are single	26.1	41.4	49.6
Transfers (men to women)			
–protected	158	166	168
–unprotected	264	298	299
–long term	230	283	288

What causes the ∩-shaped prevalence pattern for women? As sex gets safer for men, they start changing their behavior. Specifically, in the experiment conducted, men engage in more risky behavior along all three dimensions of riskiness present in the model: (i) more men choose not to marry (i.e., there are more singles); (ii) singles are more likely to have sex (rather than remaining abstinent); (iii) when having casual sex, people are less likely to use a condom. Combined, these changes lead to a large increase in the percentage of sex that is casual (rather than in marriage). It rises from 15.6 to 35.4%–i.e., it more than doubles. Given this change in male sexual behavior, many more women are at risk now, because they are the sexual partners of these men. Why would women go along with this? After all, sex has not gotten any safer for women. The answer is simple. They get compensated for the additional risk they incur. Equilibrium transfer payments from men to women go up for all sexual activities.

Interestingly, as sex gets even safer for men, eventually women start to see the benefits as well. The last column in Table 2 shows that prevalence also declines for women if the female-to-male transmission risk becomes low enough. This happens even though men engage in even more risky behavior now. The reason is that there are two countervailing effects. On the one hand, a decrease in the female-to-male transmission rate will lead to a reduction in the HIV/AIDS rate, other things equal. On the other hand, men (and therefore women) engage in riskier behavior and this causes the prevalence rate to go up, ceteris paribus. In the beginning, the behavioral effect may dominate. Eventually, the reduction in transmission rate must take control, though, leading to an overall decline in the prevalence rate. This becomes most obvious in the extreme: If circumcision completely prevented the transmission of HIV/AIDS to males (which it does not) then the disease could not survive.

To conclude, male circumcision is not necessarily and unambiguously a policy that leads to a reduction in HIV/AIDS. Of course, the simulations only point to a potential problem. Establishing the presence and exact magnitude of this problem in a definitive manner would require further investigation. In particular, the strengths of the countervailing effects will depend on the amount by which the transmission risk is reduced by circumcision, which is an ongoing empirical question. The analysis also suggests that measuring the effect that circumcision has on the HIV/AIDS rate for men (e.g., randomized field experiments) might not be sufficient for answering this question. First, one would need to collect data both on men and women. Even if such data was collected, there are still potential general equilibrium effects that cannot be detected in field experiments unless feedback to and from the aggregate HIV/AIDS rate is allowed. Specifically, it could be the case that when a group of men is circumcised while a control group is not, that the infection rate for the circumcised men is lower than for the control group, and yet that it is higher for everyone, compared to a world where no one had been circumcised, due to higher risk-taking by parts of the population.

6.2.2 Policies that Reduce Transmission for Both Sexes

Another policy that has been advocated recently is the treatment of other sexually transmitted diseases (STDs). STDs often lead to open sores that make a person more susceptible to the transmission of the HIV/AIDS virus. Thus, reducing other STDs will decrease the transmission risk, both for men and women. For example, Grosskurth et al (1995) finds that improved STD treatment reduced HIV incidence by about 40% in rural Tanzania. Similarly, a vaccine has been developed recently that, according to prelimary findings, reduces the transmission rate by 30%.[15] Table 3 shows the simulation results for this policy. The experiment is similar to the one conducted in the previous section. Now the transmission risk declines for both men and women, though. Again, such a policy has potential draw-backs, but this time for both genders. As sex gets safer, the overall prevalence rate for HIV/AIDS first goes up and then down. The reason is related to the dramatic behavioral changes that occur. As sex gets safer, both men and women are more likely remain single, these singles in turn are now more likely to have sex, and conditional on having sex they are less likely to use a condom. When the change in quarterly transmission risk is small these three dimensions of increased riskiness lead to a 50% increase in the prevalence rate. As sex gets safer and safer the effect of the decreased transmission rate eventually dominates and the overall prevalence rate falls. Note that the last experiment roughly corresponds to the most recent vaccine study that finds a 30% reduction in the transmission risk. Our results may suggest that such a vaccine will lead indeed to a reducation in the HIV/AIDS rate. However, our findings also indicate that such a result may indeed be quite fragile, in the sense that only a slightly lower reducation may already lead to an increase instead.

Taking stock, the widespread treatment of other STDs (while probably a good policy for many reasons) will not necessarily lead to a decrease in the HIV/AIDS rate, even though it lowers the transmission risk from a one-time sexual encounter. The reason is that behavioral changes might lead to more risky behavior. Specifically, more unprotected sex with different

[15] Recent promising results of a new vaccine were for example reported in a Wall Street Journal article on September 25, 2009: "Vaccine Shows Promise in Preventing HIV Infections."

partners may occur, which increases the odds of catching the disease. Further empirical studies are necessary to understand better whether this is merely a theoretical possibility or is indeed relevant in Africa (and elsewhere). Again, conducting randomized field experiments on a small scale might not be enough to assess this. General equilibrium effects are present and they might be powerful.

TABLE 3: TREATING OTHER STDS

	Benchmark	*Small* Δ	*Medium* Δ	*Large* Δ	*Pattern*
$1-\gamma_u^m$	0.06	0.05	0.045	0.04	$\downarrow$
$1-\gamma_u^f$	0.09	0.08	0.075	0.07	$\downarrow$
HIV/AIDS rate,%	12.3	18.6	14.6	11	$\cap$
Fraction of sex that is casual, %	15.6	36.5	37.5	40.6	$\uparrow$
% of causal sex with condom	24.8	17	15	14.8	$\downarrow$
% Singles who have causal sex	51	73	73	74	$\uparrow$
% Men who are single	30.4	49.4	50.3	52.8	$\uparrow$
% Women who are single	26.1	42.6	43.7	46.6	$\uparrow$

6.3 Promoting Marriage

Another typical policy is to encourage change in people's behavior by promoting faithfulness and marriage. There are various ways to induce behavioral change, ranging from campaigns and subsidies to improve the perceived bliss of marriage, social and church activities to enable people to find a suitable marriage partner, and counselling and harsher social sanctions to increase the duration of marriage. In order to get a first insight regarding the efficacy of these various policies, in the model three experiments are undertaken. First, the utility from marriage is increased. Second, the search cost of finding a marriage partner is lowered. Third, the divorce hazard is reduced.

6.3.1 Increased Utility from Marriage

The implications from increased marital bliss might be quite complex, as Table 4 illustrates. To more easily follow the complexity, the last column of the table displays pictorially the change in behavior from the left to the right column for each of the variables. Experiments 1 through 4 gradually increase the value from marriage so that the final experiment has a utility value that is twice the benchmark. The prevalence rate displays interesting nonmontonic behavior: at first it falls, then it increases, and finally it falls again. That is, in the experiments, marriage is not unambiguously a good thing. The reason is that as more people enter marriage, it becomes a riskier activity. Initially the riskiest types have mostly casual sex and therefore don't do much damage in the marriage market. As marriage becomes more attractive, more of the riskier types (those with a low discount rate or those with a longer sexual history) enter marriage and the composition of the marriage pool changes dramatically. This is particularly apparent when comparing experiments 1 and 2. The prevalence rate in the marriage market goes up by more than 50%, for men from 10 to 16 percent, and for women from 11 to 19 percent. The HIV/AIDS rate in the casual markets falls. As a consequence, singles have a lot more sex (up by almost 50% from 47 to 67 percent. Moreover, singles become less careful: they are more likely to have unprotected sex: condom use decreases by almost a third, from 24 to 17 percent. Together, these changes cause a dramatic increase in the prevalence rate, from 11 to 16 percent.

As marriage becomes even more attractive, there are fewer and fewer singles. The prevalence rates start declining in all markets. In experiment 4 the number of singles is half that of the benchmark experiment. The remaining singles still have a lot more sex than in the benchmark (75% instead of 51% of singles have sex in a given period) and when they do, they are less likely to use a condom (condom use is down by half). However, the impact of the overall increase in faithfulness dominates and the societal HIV rate declines to 9.5% (from 12.3). Interestingly, the increase in marriage leads to a lower prevalence rate in all markets. The decline is larger in the riskiest markets. By comparing experiment 4 with the benchmark, observe that the difference in prevalance rates between the three markets have

significantly shrunk. In the benchmark experiment, a person having casual sex is about four times as likely to be matched with an infected partner than someone seeking marriage. This factor shrinks to less than two in experiment 4. In fact, the difference between seeking unprotected casual sex and a marriage partner has almost entirely disappeared in experiment 4. It is perhaps trite to say that if everyone married (and remained faithful) that the HIV/AIDS epidemic would end.

TABLE 4: PROMOTING MARRIAGE

	Bench	*Exp 1*	*Exp 2*	*Exp 3*	*Exp 4*	*Pattern*
Utility from marriage, l	5	5.5	7	8	10	$\uparrow$
HIV rate (society)	12.3	10.5	15.9	12.5	9.5	$\backsim$
Fraction of sex that is casual	15.6	12	17	13.3	9.3	$\backsim$
Condom use	24.8	23.7	16.8	14.7	12.9	$\downarrow$
% Singles who have casual sex	51.4	46.5	66.9	67.3	75.2	$\bigcup$
% Males who are single	30.4	26.3	29	23.9	15.5	$\backsim$
% Females who are single	26.1	22.8	23.2	19.2	14.9	$\backsim$
Transfers (from men to women)						
–Protected	158	159	185	191	159	$\bigcap$
–Unprotected	264	264	291	194	163	$\downarrow$
–Long term	230	233	281	189	137	$\bigcap$
Prevalence rates in different markets						
–Protected	40	41	29	22	17	$\bigcap$
–Unprotected	35	33	22	16	12	$\downarrow$
–Long term	10	9	16	14	11	$\backsim$
–Male, protected	48	50	34	26	20	$\bigcap$
–Male, unprotected	45	43	27	20	14	$\downarrow$
–Male, marriage	11	10	19	16	13	$\backsim$
–Female, protected	48	50	34	26	20	$\bigcap$
–Female, unprotected	45	43	27	20	14	$\downarrow$
–Female, marriage	11	10	19	16	13	$\backsim$

6.3.2 Marriage: Entry vs. Exit

Making marriage more attractive (as in Section 6.3.1) might be difficult to implement. A more realistic policy might be to either facilitate entry into marriage (by making search

easier) or impede exit from marriage (by making divorce harder). For example, social events organized by community or religious groups may facilitate searching for a spouse. Likewise, the provision of marriage councelling services may reduce divorce. Similarly, profamily tax codes could dissuade divorce. The impact effect of both strategies should be an increase in marriage. This has the potential to lower the HIV/AIDS rate. Concretely, these policies are operationalized by lowering the search cost of finding a marriage partner, ω_{LT}, and decreasing the odds of divorce, ξ. The corresponding experiments are depicted in Table 5. The findings indicate that a decrease in search cost is a lot more effective than a reduction in divorce risk. A small decrease in divorce risk lowers HIV/AIDS due to longer lasting marriages. There are fewer single males in society. A larger increase raises the rate of HIV/AIDS. Even though people stay longer sill in marriage, the fraction of people who are single remains roughly constant (or even declines), which means less people are entering into marriage. Further, single males choose to have more casual sex now. Together, these behavioral adjustments lead to a roughly constant HIV/AIDS rate.

Facilitating search, on the other hand, leads to a substantial decline in the HIV rate. When the search cost is lowered by a third (from 30 to 20), there are also almost a third less singles around (22.6 rather than 30.4% of all males are singles). Singles are also less eager to have casual sex. They know that marriage is safer and so the opportunity cost of getting infected in the short term market is higher. Interestingly, a decline in the search cost leads to a clearer separation of different types into different markets. The difference in prevalence rates across the three market increases substantially as the search cost falls. In the table, when the search cost is at its lowest value (i.e., the rightmost column), the overall prevalence amongst males desiring marriage is only 7%; i.e., substantially lower than the 10% in the benchmark. On the flip side, the average prevalence rate amongst males desiring casual protected sex increases from 40 to almost 50 percent. Given that more people sort into marriage, and that marriage is safer than before, the overall prevalence rate declines substantially as the search cost falls. The opposite seems to be the case for a decrease in the divorce risk, where markets get more similar and hence the HIV rate does not decline

despite a (slight) decrease in the number of singles.

Table 5: Marriage–Search Costs and Divorce Risk

	Benchmark	*Divorce risk* ↓		*Search cost* ↓	
Divorce risk, ξ	0.03	0.02	0.015	0.03	0.03
Marital search cost, ω_{LT}	30	30	30	28	20
HIV/AIDS rate	12.3	11.6	12.3	11.6	7.8
Fraction of sex that is casual, %	15.6	15.6	17.4	14.3	8.6
% Singles who have causal sex	51	57	64	50	41
% Men who are single	30.4	28.4	28.8	28.8	22.6
% Women who are single	26.1	23.1	23.3	24.6	18.5
Prevalence rates					
–Male, protected	40	40	38	41	49
–Male, unprotected	36	33	33	35	33
–Male, marriage	10	10	11	9	7

7 Conclusions

In Malawi about 11 percent of the population has the HIV/AIDS virus. Roughly 18 percent of sex is casual and a condom is used a quarter of the time. An equilibrium search model is constructed to analyze the Malawian HIV/AIDS epidemic. At the heart of the model is homo economicus. Specifically, it is presumed that the economic man (or woman) searches for the type of sexual activity that (s)he desires to engage in, while rationally taking into consideration the risks of this activity. Some people will select stable long-term relationships, others may choose more fleeting ones. Condoms may or may not be used in these more ephemeral encounters, depending on the participants' mutual desires. The number of such encounters is partially under people's control. All these of choices crucially affect the spread of HIV/AIDS in society.

The theoretical model that is developed is simulated to see whether or not it can capture some of the salient features of the Malawian HIV/AIDS epidemic, such as those mentioned above. It can. The benchmark simulation is then used to undertake some policy interventions that are discussed in the literature. The simulation results suggest that policy analysis of HIV/AIDS interventions may be very complicated. In particular, there is considerable scope for well-intentioned policies to backfire, due to induced changes in human behavior and general equilibrium effects. For example, treating sexually transmitted diseases may make people less susceptible to the HIV/AIDS virus. As the risk of contracting HIV/AIDS goes down more people may choose to engage in short-term unprotected sex. This change in behavior may actually cause the rate of HIV/AIDS to rise when the impact of STDs on the transmission of HIV/AIDS is relatively small. If curing STDs has a large impact on preventing the transmission of the virus then the rate of HIV/AIDS in society will fall. (Think about the case where the policy completely stops the transmission of virus.) Thus, the impact of this policy on the rate of HIV/AIDS in society may be nonmonotonic depending on the efficacy that curing STDs has on the transmission rate of HIV/AIDS. Future generations of computational general equilibrium models, in conjunction with empirical work, may help policy makers assess which equilibrium channels are likely to be particularly crucial and that require special attention when implementing interventions.

8 Appendix–Data

- Table 1. The data on the prevalence of HIV/AIDS in Malawi derive from the Demographic and Health Surveys' (DHS) Final Survey for Malawi in 2004. See DHS (2004, Table 12.3). The fraction of sex that is short term is the proportion of people–averaged across men and women–who had sex with a non-marital, non-cohabiting partner during the last year, conditional on being sexually active, and is taken from DHS (2004, Table 11.9). Condom usage for short term sex also derives from DHS (2004, Table 11.9)–and is averaged across men and women. Singles who have short term sex is reported in DHS (2004, Tables 6.71 and 6.72) and corresponds to the weighted average of never married and divorced/separated/widowed men and women. The proportion of the population that is single is contained in DHS (2004, Table 6.1), where single is interpreted as anyone who is not currently married, averaged across men and women. The fraction of males and females that has ever been married is taken from DHS (2004, Table 6.1) for the 20-to-24-year-old age category. The U.S. Census Bureau (2004) estimates the Malawian quarterly death rates without HIV/AIDS to be 0.3% and including HIV/AIDS to be 0.56%. Thus, the percentage of people that die from non-HIV/AIDS related causes in Malawi is taken to be $3/5.6 \times 100 = 54\%$.

- Figure 2. Source, DHS (2004, Table 6.1).

- Figure 3. Source, DHS (2004, Table 12.3).

- Figure 4. Source, Delavande and Kohler (2009, Table 3).

- Parameter values. The actual parameter values used for the benchmark experiment are listed in the table below.

TABLE 6: BENCHMARK PARAMETER VALUES

Category	*Parameter value*	*Criteria*
Tastes	$u = 8, p = 2, l = 3$ (utility)	fit
	$\widetilde{\beta}_1 = 0.987, \widetilde{\beta}_2 = 0.897, \widetilde{\iota}_1 = 0.894, \widetilde{\iota}_2 = 0.795$ (discounting)	"
	$A = 5$ (life with Aids)	"
HIV/AIDS	$\gamma_u^m = 0.94, \gamma_u^f = 0.91, \gamma_p^m = 0.91, \gamma_p^f = 0.91$ (transmission)	literature
	$\alpha = 0.025, \delta_a = 0.125$ (symptoms and death)	"
Shocks	$\delta = 0.003, \xi = 0.03$ (death and divorce)	literature
	$\eta = 0.05, \eta_b = 0.1$ (switch to high discount factor)	fit
Search	$\omega_s = 0.5, \omega_l = 30, \kappa = 0.2$	fit
Miscellaneous	$y = 300$ (income)	literature
	$\mu_1 = 0.01, \mu_2 = 0.001$ (births of types)	imposed

9 Appendix–Theory

9.1 Symptom Probabilities

9.1.1 Pr[**no symptoms in either person at end of period** $n|\phi$]

The odds that neither partner shows the symptoms of HIV/AIDS by the end of period n can occur for three reasons. First, neither party might have the disease at the time of marriage. The likelihood of this is $\phi\bar{\phi}_l$. Second, both parties could have had been infected with the virus when they were married. The chances of neither of them showing symptoms after n periods of marriage is $(1-\phi)\left(1-\bar{\boldsymbol{\phi}}_l\right)(1-\alpha)^{2n}$. Last, only one of the parties might have initially been infected, but neither partner shows symptoms by the end of n. The probability of this compound event is $[(1-\phi)\bar{\phi}_l + \phi(1-\bar{\phi}_l)](1-\alpha)^n[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j} + \gamma_u^n]$. This last event can be decomposed in two three cases. The term $(1-\phi)\bar{\phi}_l(1-\alpha)^n(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}$ gives the odds that the individual in question initially has the disease, transmits it to his partner in some period j, and the symptoms fail to materialize in either person. The expression $(1-\phi)\bar{\phi}_{f,l}(1-\alpha)^n\gamma_u^n$ gives the likelihood that his partner never catches it. The

rest of the formula captures the symmetric case where it was the partner who initially had the virus. Taking stock of all of this gives

$$\Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] = \phi\bar{\boldsymbol{\phi}}_{f,l} + (1-\phi)\left(1-\bar{\boldsymbol{\phi}}_{f,l}\right)(1-\alpha)^{2n}$$
$$+[(1-\phi)\bar{\boldsymbol{\phi}}_{f,l} + \phi(1-\bar{\boldsymbol{\phi}}_{f,l})](1-\alpha)^{n}[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j} + \gamma_u^n]. \tag{18}$$

9.1.2 Pr[**symptoms just in partner at end of period** $n|\phi$]

What are the chances that just individual's partner shows the symptoms of HIV/AIDS by the end of n periods of marriage? Once again there are three cases to consider. First, perhaps both parties initially had the virus but the symptoms just appear in the partner at the end of n. This will occur with probability $(1-\phi)(1-\bar{\phi}_l)(1-\alpha)^{2n-1}\alpha$. Second, maybe just the partner had the disease initially. It may have transmitted to the individual in some period j, yet he never shows any symptoms. The chances of this are $\phi(1-\bar{\phi}_l)(1-\alpha)^{n-1}\alpha[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}+\gamma_u^n]$. Last, the individual might have been the person who initially had the sickness and it then spread to his partner in some period j. The odds of happening are $(1-\phi)\bar{\phi}_l(1-\alpha)^n\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-1-j}$. It then transpires that

$$\Pr[\text{symptoms just in partner at end of period } n|\phi] = (1-\phi)(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{2n-1}\alpha$$
$$+\phi(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{n-1}\alpha[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}+\gamma_u^n]$$
$$+(1-\phi)\bar{\boldsymbol{\phi}}_l(1-\alpha)^n\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-1-j}. \tag{19}$$

9.1.3 Pr[**symptoms in person at end of period** $n|\phi$]

The likelihood of the individual exhibiting symptoms in period n is now calculated. His partner does not show any symptoms during the first $n-1$ periods, but might in the nth one. As above there are three cases to consider. First, both parties might have had HIV/AIDS at the time of marriage, an event which occurs with probability $(1-\phi)(1-\bar{\phi}_{f,l})(1-\alpha)^{2n-1}\alpha$. Second, maybe only the partner initially had the virus and the person catches it in some

period j. The odds of this are $\phi(1-\bar{\phi}_l)(1-\alpha)^{n-1}\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j-1}$. Last, the individual may have been the one who had it at the beginning of marriage. He may transmit it to his partner, who in turn shows no symptoms before period $n-1$. This occurs with probability $(1-\phi)\bar{\phi}_l\alpha(1-\alpha)^{n-1}[(1-\gamma_u)\sum_{j=0}^{n-2}\gamma_u^j(1-\alpha)^{n-1-j}+\gamma_u^{n-1}]$. Therefore,

$$\begin{aligned}\Pr[\text{symptoms in person at end of period } n|\phi] &= (1-\phi)(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{2n-2}\alpha \\ &+\phi(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{n-1}\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j-1} \\ &+(1-\phi)\bar{\boldsymbol{\phi}}_l\alpha(1-\alpha)^{n-1}[(1-\gamma_u)\sum_{j=0}^{n-2}\gamma_u^j(1-\alpha)^{n-1-j}+\gamma_u^{n-1}]. \qquad (20)\end{aligned}$$

9.2 The Value of a Long-term Relationship, $d=\iota$

The value of a long-term relationship for a type-x person who has a prior of ϕ and low discount factor, $\widetilde{V}_l^{\iota}(\phi,x)$, needs to be characterized. Recall that the discount factor may switch from the low value, ι, to the high one, β, with probability η. If a switch occurs, the discount factor will remain at the high value thereafter. Now, think about the discount factor that will be applied to the utility $n>1$ periods ahead. With probability $(1-\eta)^{n-1}$ the individual will keep the discount factor ι–note that he will use the discount factor ι for the next period with certainty. If this event transpires he will discount utility n periods ahead by $\iota\iota^{n-1}$. Alternatively, he may draw the discount factor β some $k<n$ periods down the road. The new discount factor will start to apply to period-$(k+1)$ utility. This event happens with probability $(1-\eta)^{k-1}\eta$. He will then discount period-n utility by $\iota\iota^{k-1}\beta^{n-k}$. Define the two new discount factors $\underline{\beta}^{\iota}(n)$ and $\overline{\beta}^{\iota}(n)$ by

$$\underline{\beta}^{\iota}(n)\equiv(1-\eta)^{n-1}\iota\iota^{n-1} \quad \text{(no switch)},$$

and

$$\begin{aligned}\overline{\beta}^{\iota}(n) &\equiv \sum_{k=1}^{n-1}\eta(1-\eta)^{k-1}\iota\iota^{k-1}\beta^{n-k} \quad \text{(switch at some time } k) \\ &= \eta\iota\beta^{n-1}\frac{1-[\iota(1-\eta)/\beta]^{n-1}}{1-\iota(1-\eta)/\beta},\end{aligned}$$

with

$$\overline{\beta}^{y}(1) = 0.$$

Given this, the value of a long-term relationship for the low-discount factor case is given by

$$\begin{aligned}
\widetilde{V}_l^{\iota}(\phi, x) \;=\; & \frac{(y - t_l)^{1-\sigma}}{1-\sigma} + u \\
& + \sum_{n=1}^{\infty} [\underline{\beta}^{\iota}(n) + \overline{\beta}^{\iota}(n)](1-\xi)^n \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\
& \times [\frac{(y - t_l)^{1-\sigma}}{1-\sigma} + u + l] \\
& + \sum_{n=1}^{\infty} \underline{\beta}^{\iota}(n)(1-\xi)^{n-1}\xi \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\
& \times [\eta V_l^{\beta}(\phi^h(n,\phi), x) + (1-\eta) V_l^{i}(\Phi^h(n,\phi), x)] \\
& + \sum_{n=1}^{\infty} \overline{\beta}^{\iota}(n)(1-\xi)^{n-1}\xi \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\
& \times V_l^{\beta}(\Phi^h(n,\phi), x)x) \\
& + \sum_{n=1}^{\infty} \underline{\beta}^{\iota}(n)(1-\xi)^{n-1} \Pr[\text{symptoms just in partner at end of period } n|\phi] \\
& \times [\eta V_l^{\beta}(\Phi^a(n,\phi), x) + (1-\eta) V_l^{\iota}(\Phi^a(n,\phi), x)] \\
& + \sum_{n=1}^{\infty} \overline{\beta}^{\iota}(n)(1-\xi)^{n-1} \Pr[\text{symptoms just in partner at end of period } n|\phi] \\
& \times V_{m,l}(\Phi^a(n,\phi), x) \\
& + \sum_{n=1}^{\infty} [\underline{\beta}^{\iota}(n) + \overline{\beta}^{\iota}(n)](1-\xi)^{n-1} \Pr[\text{HIV/AIDS symptoms in person at end of period } n|\phi] \\
& \times A.
\end{aligned} \tag{21}$$

Except for the possibility of a switch in the discount factor, the formula resembles (10). In fact, (10) collapses to (21) when $\eta = 1$ implying $\underline{\beta}(n) = 0$–here $\overline{\beta}^{\iota}(n)$ should be set to β^n. As before, the first line reports the current utility from the relationship. Suppose that a match sustains until period $n+1$. The second and third lines give the discounted expected utility accruing over the next n periods. The next lines handle breakup events for period

$n+1$. Lines 4 and 5 cover the situation where the discount factor applying to period $n+1$ remains low and an exogenous breakup occurs. Note that the discount factor may switch upwards with probability η in period $n+1$. Lines 6 and 7 assume an exogenous breakup occurs and that the discount factor has switched sometime before $n+1$.

9.3 Stationary Distributions

Before starting, define the function J by $J(z)=1$, if $z=0$, and $J(z)=0$, if $z\neq 0$.

9.3.1 Singles distributions, $d=\iota$

Consider the case where singles have the low discount factor, ι. Note that a person can only get into the low discount factor state from the low discount factor state. The equilibrium distribution for type-x singles, with a low discount factor, and prior ϕ is specified by

$$\begin{aligned}\mathcal{S}^{\iota}(\phi',x) &= \mu(1-\eta) \\ &\quad +(1-\delta)\sum_{\phi}[1-\Pi_l^{\iota}(\phi,x)](1-\eta)\mathcal{S}^{\iota}(\phi,x) \\ &\quad \times\{\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_p)(1-\gamma_p)]\}J(\phi'-\Phi_p(\phi))\Pi_p^{\iota}(\phi,x) \\ &\quad +\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_u)(1-\gamma_u)]\}J(\phi'-\Phi_u(\phi))\Pi_u^{\iota}(\phi,x) \\ &\quad +[1-\alpha(1-\phi)]J(\phi'-\Phi_a(\phi))\Pi_a^{\iota}(\phi,x)\} \\ &\quad +\mathcal{L}^{\iota}(\phi',x).\end{aligned} \tag{22}$$

At the beginning of any period the pool of singles comes from five sources. First, new ones are born. A fraction of these people will have a low discount factor. These people have not had sex yet, so they know with certainty they do not have the HIV/AIDS virus. This inflow is given by the first term on right-hand side. Now, some older singles will fail to find a partner on the market for long-term relationships. The fraction of populace in this situation is specified by the second line. Three things can happen to them. Some of these people will find a mate on the short-term protected sex market. This is second source of singles and is given on the third line. Others will match on the short-term unprotected sex market. This accounts for the third source of inflow and is shown by the fourth line. Still other will fail

to match, which gives the fourth influx, as the fifth line indicates. The fifth inflow is from long-term relationships that have broken up. The last line gives the contribution from this source. This term is explained next.

Now, suppose that a type-x individual with prior ϕ and a low discount factor enters into a long-term relationship. There are two reasons why this person may exit into single life at a later date: the match dissolves exogenously, or his partner develops HIV/AIDS symptoms. These two events are not mutually exclusive. But, note that if a male enters into single life because his partner develops HIV/AIDS symptoms, then it doesn't matter whether or not there was a breakup as well, because the probability of the latter two events sum to one. Now, if the match terminates in period n solely due to a breakup then the male will exit into single life with the prior $\Phi_l^h(\phi, n)$, while if his partner develops HIV/AIDS symptoms then he will exit with prior $\Phi_l^a(\phi, n)$. The odds that a type-x person with a low discount factor will exit into single life at some future date with a low discount factor, ι, and a prior ϕ', conditional on starting with a prior ϕ, will consequently read

$$\begin{aligned}\mathcal{L}^\iota(\phi', x|\phi) &= \sum_{n=1}^{\infty}(1-\eta)^n\xi(1-\xi)^{n-1}(1-\delta)^n J(\phi' - \Phi_l^h(\phi, n)) \\ &\quad \times \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\ &\quad + \sum_{n=1}^{\infty}(1-\eta)^n(1-\xi)^{n-1}(1-\delta)^n J(\phi' - \Phi_l^a(\phi, n)) \\ &\quad \times \Pr[\text{symptoms just in partner at end of period } n|\phi],\end{aligned} \tag{23}$$

where the probabilities for symptoms are specified by (18) to (20) above. From this it is easy to calculate that the unconditional exit distribution, $L^\iota(\phi', x)$, is given by

$$\mathcal{L}^\iota(\phi', x) = \sum_{\phi} \mathcal{L}^\iota(\phi', x|\phi)\Pi_l^\iota(\phi, x)\mathcal{S}^\iota(\phi, x). \tag{24}$$

9.3.2 Singles distributions, $d = \beta$

Next, the situation where singles have the high discount factor, β, will be presented. An person can move into the high discount factor state from either the high or low one. The

equilibrium distribution function for type-x singles, with a high discount factor, and prior ϕ is given by

$$\begin{aligned}
\mathcal{S}^{\beta}(\phi',x) &= \eta\mu \\
&\quad +(1-\delta)\sum_{\phi}[1-\Pi_l^{\beta}(\phi,x)]\mathcal{S}^{\beta}(\phi,x) \\
&\quad \times\{\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_p)(1-\gamma_p)]\}J(\phi'-\Phi_p(\phi))\Pi_p^{\beta}(\phi,x) \\
&\quad +\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_u)(1-\gamma_u)]\}J(\phi'-\Phi_u(\phi))\Pi_u^{\beta}(\phi,x) \\
&\quad +[1-\alpha(1-\phi)]J(\phi'-\Phi_a(\phi))\Pi_a^{\beta}(\phi,x)\} \\
&\quad +\mathcal{L}^{\beta}(\phi',x) \\
&\quad +(1-\delta)\sum_{\phi}[1-\Pi_l^{\iota}(\phi,x)]\eta\mathcal{S}^{\iota}(\phi,x) \\
&\quad \times\{\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_p)(1-\gamma_p)]\}J(\phi'-\Phi_p(\phi))\Pi_p^{\iota}(\phi,x) \\
&\quad +\{1-\alpha[(1-\phi)+\phi(1-\bar{\boldsymbol{\phi}}_u)(1-\gamma_u)]\}J(\phi'-\Phi_u(\phi))\Pi_u^{\iota}(\phi,x) \\
&\quad +[1-\alpha(1-\phi)]J(\phi'-\Phi_a(\phi))\Pi_a^{\iota}(\phi,x)\}\,.
\end{aligned} \tag{25}$$

The first six lines are the direct analogue to equation (22), only with a high discount factor. The exit distribution from married life, $L^{\beta}(\phi',x)$, is defined below. It includes married individuals whose own discount factor moved up from ι to β at some time during their marriage. The last four lines reflect the inflow of low discount factor singles who transit to a high discount factor from the low one.

To calculate the exit distribution, $L^{\beta}(\phi',x)$, imagine a type-x person who entered married life with a high discount factor and a prior of ϕ. The probability that he will exit married life with the prior ϕ' is given by

$$\begin{aligned}
\mathcal{L}^{\beta}(\phi',x|\phi) &= \sum_{n=1}^{\infty}\xi(1-\xi)^{n-1}(1-\delta)^n J(\phi'-\Phi_l^h(n,\phi)) \\
&\quad \times \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\
&\quad +\sum_{n=1}^{\infty}(1-\xi)^{n-1}(1-\delta)^n J(\phi'-\Phi_l^a(n,\phi)) \\
&\quad \times \Pr[\text{symptoms just in partner at end of period } n|\phi].
\end{aligned} \tag{26}$$

Likewise, consider those start married life with a low discount factor and a prior of ϕ but who switch to high discount factor. The odds that they will exit marriage with the prior ϕ'

are

$$\begin{aligned}\mathcal{L}^{\iota\beta}(\phi',x|\phi) &= \sum_{n=1}^{\infty}\left[1-(1-\eta)^n\right]\xi(1-\xi)^{n-1}(1-\delta)^n J(\phi'-\Phi_l^h(n,\phi)) \\ &\quad\times \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi] \\ &\quad+\sum_{n=1}^{\infty}\left[1-(1-\eta)^n\right](1-\xi)^{n-1}(1-\delta)^n J(\phi'-\Phi_l^a(n,\phi)) \\ &\quad\times \Pr[\text{symptoms just in partner at end of period } n|\phi]. \qquad (27)\end{aligned}$$

Therefore, the unconditional exit distribution for people entering into single life from married life with a high discount factor is

$$\mathcal{L}^{\beta}(\phi',x)=\sum_{\phi}\mathcal{L}^{\beta}(\phi',x|\phi)\Pi_l^{\beta}(\phi,x)\mathcal{S}^{\beta}(\phi,x)+\sum_{\phi}\mathcal{L}^{\iota\beta}(\phi',x|\phi)\Pi_l^{\iota}(\phi,x)\mathcal{S}^{\iota}(\phi,x). \qquad (28)$$

Last, equations (22) to (28) fully describe the operator T in (12).

10 Appendix–Algorithm

10.1 Computing the Value Functions and Distribution Functions

The algorithm enters each iteration with a guess for the set of values functions, $\widetilde{V}_{g,r}^d$ and $V_{g,r}^d$, and stationary distributions, L_g^d and S_g^d, for each type of individual x. It is easy to create a guess for the nonprevalence rates, $\bar{\phi}_{g,r}$, from the distribution functions by using (13) to (14). On each iteration the value functions are updated using equations (1), (3), (4), (6), (7), (10), (11), and (21). At the same time the distribution functions are revised on each iteration using (22) to (28). A grid is constructed for the individual's prior, ϕ, over his nonprevalence rate. The above functions are computed at each grid point. Even though ϕ may lie on a grid point there is no guarantee that ϕ' will, given the form of the updating functions (2), (5), (9) and (8). This is resolved using an interpolation scheme (Matlab's cubic Hermite scheme). For example to compute $\widetilde{V}_a^{\beta}(\phi',x)$ on the righthand side of (1) for an off-the-grid point ϕ'a weighted average of $\widetilde{V}_a^{\beta}(\phi_i',x)$ and $\widetilde{V}_a^{\beta}(\phi_{i+1}',x)$ at the two nearest adjacent grid points, $\phi_i'\leq\phi'\leq\phi_{i+1}'$, is computed.[16] A similar issue arises when computing

[16] The Hermit scheme preserves monotonicity of $\widetilde{V}_a^{\beta}$ in ϕ, and therefore the extrapolation can be interpreted as a weighted-average of the nearest grid points. The weights themselves

the distribution functions. Take the density shown in (22), for example. The updating rules (2) and (5) in general will not map a grid point ϕ into a ϕ' that lies on the grid. Therefore, ϕ is mapped onto the two closest adjacent points, ϕ'_i and ϕ'_{i+1}, such that $\phi'_i \leq \Phi_r(\phi) \leq \phi'_{i+1}$, for $r = a, u, p$, using a linear weighting scheme.

10.2 Pseudo Code for Monte Carlo - number of partners up to age t

The task is to simulate the average number of partners for n individuals, say males, over m periods. Create matrices to store the sample paths across individuals for variables such as ϕ, n, p, a, h, w where n is the number of periods in a long-term relationship ($n = 0$ for a single), p is the number of partners to date, a is a variable indicating whether the person is alive ($a = 0$ for a dead person and 1 for a living one), h is a variable indicating whether the person is healthy ($h = 0$ for a person with AIDS symptoms and 1 for a person without symptoms) and w is the number of marriages ("weddings") the person was in to date. For each individual do the following:

1. At the beginning of life draw two random numbers from a uniform distribution on $[0, 1]$. If the first is below η, then start the person with high discount factor and only use decision rules with superscript β throughout his life. Otherwise, in the first period $t = 1$ of a person's life use decision rule with superscript ι. The probability of switching by the end of period t is $n(t) = [1 - (1 - \eta)^t]$. Consider the realization of the second random variable (call it r) and find the integer $\bar{t} \in \{1, 2, ...\}$ such that $n(\bar{t} - 1) \leq r < n(\bar{t})$. For every period $t \leq \bar{t}$ of the simulated persons life use policy rules with the index ι, for every period $t > \bar{t}$ use policy rule with the index β. (Note that $\bar{t}$ might be larger than T, the maximum number of periods we want to simulate, in which only subscript y applies).

2. Start off all people as newly born singles with $\phi = 1, n = 0$, $p = 0$, $w = 0$, $h = 1$,

are computed based on the entire set of grid points, not just based on the nearest ones, to preserve certain smoothness properties.

and $a = 1$. For each person i draw a matrix of $m \times 3$ uniformly distributed random variables. The seed for the random number generator should be a function of i.

3. Individual i will enter a given period t with some values for d, ϕ, n, p, h and a, denoted by $d_{i,t}$, $\phi_{i,t}$, $n_{i,t}$, $p_{i,t}$, $h_{i,t}, w_{i,t}$ and $a_{i,t}$. If $a_{i,t} = 0$ the individual is dead and nothing has to be done. Terminate going down the time path for this individual. Otherwise, if $d_{i,t-1} = \iota$ check whether $t > \bar{t}$. If so, set $d_{i,t} = \beta$. Alternatively, when $d_{i,t-1} = \beta$ then $d_{i,t} = \beta$. If he is alive $a_{i,t} = 1$ but sick $h_{i,t} = 0$, go to step 4. Otherwise, if he is married, $n_{i,t} \geq 1$, go to step 5; if he is single, $n_{i,t} = 0$, go to step 6.

4. If he is alive $a_{i,t} = 1$ but sick $h_{i,t} = 0$, draw the first random variable from column t. If it is below δ_2 the person dies, i.e. $a_{i,t+1} = 0$. If it is above δ_2 then he lives $a_{i,t+1} = 1$ and $h_{i,t+1} = 0$. (δ_2 is the probability of death for a person with AIDS symptoms.)

5. Married. If $a_{i,t} = h_{i,t} = 1$ and $n_{i,t} \geq 1$ then the individual is alive and married. Draw the second random variable from the tth row of the matrix of random variables. Define the probabilities of some of the events discussed in Section 9.1.

$$N_1 \equiv \Pr[\text{no symptoms in } \textit{either} \text{ person at end of period } n|\phi],$$

$$E_1 \equiv (1-\phi)(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{2n-1}\alpha, \text{ cf (19)},$$

$$E_2 \equiv \phi(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{n-1}\alpha[(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j}+\gamma_u^n], \text{ cf (19)},$$

$$E_3 \equiv (1-\phi)\bar{\boldsymbol{\phi}}_l(1-\alpha)^n\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-1-j}, \text{ cf (19)},$$

$$A_1 \equiv (1-\phi)(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{2n-2}\alpha, \text{ cf (20)},$$

$$A_2 \equiv \phi(1-\bar{\boldsymbol{\phi}}_l)(1-\alpha)^{n-1}\alpha(1-\gamma_u)\sum_{j=0}^{n-1}\gamma_u^j(1-\alpha)^{n-j-1}, \text{ cf (20)},$$

$$A_3 \equiv (1-\phi)\bar{\boldsymbol{\phi}}_l\alpha(1-\alpha)^{n-1}[(1-\gamma_u)\sum_{j=0}^{n-2}\gamma_u^j(1-\alpha)^{n-1-j}+\gamma_u^{n-1}], \text{ cf (20)}.$$

(a) Marriage persists. If the random variable is less than $(1-\varepsilon)(1-\delta)N_1(n_{i,t})/\Lambda(n_{i,t})$, where $\Lambda(n_{i,t}) \equiv [(1-\varepsilon)N_1(n_{i,t}) + \varepsilon N_1(n_{i,t}) + E_1(n_{i,t}) + E_2(n_{i,t}) + E_3(n_{i,t}) + A_1(n_{i,t}) + A_2(n_{i,t}) + A_3(n_{i,t})]$, then the marriage persists. Here, set $\phi_{i,t+1} = \phi_{i,t}$, $n_{i,t+1} = n_{i,t} + 1, p_{i,t+1} = p_{i,t}$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$.

(b) Exogenous breakup. If the random variable lies between $(1-\varepsilon)(1-\delta)N_1(n_{i,t})/\Lambda(n_{i,t})$ and $(1-\delta)[(1-\varepsilon)N_1(n_{i,t}) + \varepsilon N_1(n_{i,t})]/\Lambda(n_{i,t})$ then the marriage marriage breaks up exogenously and the male enters single life. Here, set $\phi_{i,t+1} = \Phi_l^h(n_{i,t}, \phi_{i,t})$, $n_{i,t+1} = 0, p_{i,t+1} = p_{i,t}$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$.

(c) Partner shows symptoms of AIDS/HIV. Alternatively, when the random variable lies between $(1-\delta)[(1-\varepsilon)N_1(n_{i,t}) + \varepsilon N_1(n_{i,t})]/\Lambda(n_{i,t})$ and $(1-\delta)[(1-\varepsilon)N_1(n_{i,t}) + \varepsilon N_1(n_{i,t}) + E_1(n_{i,t}) + E_2(n_{i,t}) + E_3(n_{i,t})]/\Lambda(n_{i,t})$ then the marriage marriage breaks up because the male's partner has the symptoms of AIDS/HIV. Again, the male enters single life. Here, $\phi_{i,t+1} = \Phi_l^a(n_{i,t}, \phi_{i,t})$, $n_{i,t+1} = 0, p_{i,t+1} = p_{i,t}$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$.

(d) AIDS symptoms or death. With probability $Q \equiv 1 - (1-\delta)[(1-\varepsilon)N_1(n_{i,t}) + \varepsilon N_1(n_{i,t}) + E_1(n_{i,t}) + E_2(n_{i,t}) + E_3(n_{i,t})]/\Lambda(n_{i,t})$ the person either gets AIDS symptoms or dies or both. Draw the third random variable in the tth column. If it is below δ/Q the person dies, i.e. $a_{i,t+1} = 0$. If it is above the person survives with AIDS symptoms, i.e. $a_{i,t+1} = 1$ and $h_{i,t+1} = 0$.

(e) Accounting for newly wedded agents. If $n_{i,t} = 1$, then set $p_{i,t+1} = p_{i,t} + 1$, $w_{i,t+1} = w_{i,t} + 1$. This step has to be done no matter which event a to d occurred.

6. Single. If $a_{i,t} = h_{i,t} = 1$ and $n_{i,t} = 0$ then the individual is alive and single. Draw the first random variable from the tth row of the matrix of random variables. If this random variable lies below π_l then the person is newly wedded and enters period-t married life (set $n_{i,t} = 1$). Then move to the marriage state in period t, described in step 5. Otherwise, go through the three cases denoted in the notes for a single person.

(a) Abstinence is first choice. If $\pi_{a,t} = 1$ then the individual chooses abstinence.

Draw a random variable from the second column of the matrix. If the number is less than δ the person dies and $a_{i,t+1} = 0$. If the number is between δ and $\delta + (1-\delta)\alpha(1-\phi_{i,t})$ the person gets AIDS symptoms but continues living so that $a_{i,t+1} = 1$ and $h_{i,t+1} = 0$. Otherwise the individual lives on to the next period so that $\phi_{i,t+1} = \Phi_a(\phi_{i,t})$, $n_{i,t+1} = 0, p_{i,t+1} = p_{i,t}$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$.

(b) Abstinence is second choice. If $0 < \pi_{a,t}, \pi_{s,t} < 1$ then the individual's first choice is short-term market s. Abstinence is his second choice. Draw a random variable from the second column of the matrix. If the first number is less than $\pi_{s,t}$ then the person enters the short-term market. Draw a random variable from the third column of the matrix. If the number is less than δ the person dies and $a_{i,t+1} = 0$. If the number is between δ and $\delta + (1-\delta)\alpha[(1-\phi_{i,t}) + \phi_{i,t}(1-\overline{\boldsymbol{\phi}}_s)(1-\gamma_s)]$ the person gets AIDS symptoms but continues living so that $a_{i,t+1} = 1$ and $h_{i,t+1} = 0$. Otherwise the individual lives on to the next period so that $\phi_{i,t+1} = \Phi_s(\phi_{i,t})$, $n_{i,t+1} = 0, p_{i,t+1} = p_{i,t} + 1$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$.

(c) Abstinence is last choice. If $0 < \pi_{u,t}, \pi_{p,t} < 1$ then the individual's first choice and second choices are the short term markets. Abstinence is his third choice. Draw a random variable from the second column of the matrix. If the second number is less than $\pi_{u,t}$ then the person enters the unprotected short-term market. Draw a random variable from the third column of the matrix. If the third number is less δ the person dies and $a_{i,t+1} = 0$. If the number is between δ and $\delta+(1-\delta)\alpha[(1-\phi_{i,t})+\phi_{i,t}(1-\overline{\boldsymbol{\phi}}_u)(1-\gamma_u)]$ the person gets AIDS symptoms but continues living so that $a_{i,t+1} = 1$ and $h_{i,t+1} = 0$. Otherwise the individual lives on to the next period so that $\phi_{i,t+1} = \Phi_u(\phi_{i,t})$, $n_{i,t+1} = 0, p_{i,t+1} = p_{i,t} + 1$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$. If the second number lies in the interval $(\pi_{u,t}, \pi_{p,t} + \pi_{u,t})$ then the person enters the protected short-term market. Draw a random variable from the third column of the matrix. If the number is less than δ the person dies and $a_{i,t+1} = 0$. If the number is between δ and $\delta + (1-\delta)\alpha[(1-\phi_{i,t}) + \phi_{i,t}(1-\overline{\boldsymbol{\phi}}_p)(1-\gamma_p)]$ the person gets AIDS symptoms but continues living so that $a_{i,t+1} = 1$ and $h_{i,t+1} = 0$.

Otherwise the individual lives on to the next period so that $\phi_{i,t+1} = \Phi_p(\phi_{i,t})$, $n_{i,t+1} = 0, p_{i,t+1} = p_{i,t} + 1$, $w_{i,t+1} = w_{i,t}$, $a_{i,t+1} = h_{i,t+1} = 1$. If the second number is greater than $\pi_{u,t} + \pi_{p,t}$ then the analysis proceeds as in the abstinence case.

7. The average number of partners during their life for people at age t (counted at the beginning of the period before having sex), who are still alive, is given by $\Sigma_i h_{i,t} p_{i,t} / \Sigma_i h_t$. For the number of agents that are infected, first adjust the beliefs for those who are married. For any observation (i,t) with $n_{i,t} \geq 2$ set $\phi_{i,t} = \phi^*(n_{i,t} - 1, \phi_{i,t})$. The reason for the adjustment is that n is already updated in the period where an agent get's newly wed. Then the prevalence in society is $\Sigma_i h_{i,t} \phi_{i,t} / \Sigma_i h_t$.

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