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**ABSTRACT**

Eleven percent of the Malawian population is HIV infected. Eighteen percent of sexual encounters are casual. A condom is used one quarter of the time. A choice-theoretic general equilibrium search model is constructed to analyze the Malawian epidemic. In the developed framework, people select between different sexual practices while knowing the inherent risk. The analysis suggests that the efficacy of public policy depends upon the induced behavioral changes and general equilibrium effects that are typically absent in epidemiological studies and small-scale field experiments. For some interventions (some forms of promoting condoms or marriage), the quantitative exercise suggests that these effects may increase HIV prevalence, while for others (such as male circumcision or increased incomes) they strengthen the effectiveness of the intervention. The underlying channels giving rise to these effects are discussed in detail.

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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. Furthermore, the majority of the HIV-positive population is female, compared to less than one third in most developed countries. The current analysis spotlights one part of Africa: The Republic of Malawi. Malawi has a relatively high HIV/AIDS rate and the prevalence for women is higher than for men.<sup>1</sup> It also has very good data related to HIV/AIDS and sexual behavior. So, the facts are clear. Traditionally, two approaches have been taken to studying the transmission of HIV/AIDS in Africa, viz epidemiological studies and field experiments.<sup>2</sup> Epidemiological studies are sophisticated in their treatment of equilibrium but usually lack a feedback loop that captures behavioral responses. Field-experiments are often on a small scale and do not readily allow for the assessment of general equilibrium effects.

The current paper advocates the use of choice-theoretic general equilibrium modelling as an *additional* tool for studying the African HIV/AIDS epidemic. First, the main benefit that economics can bring to the field of epidemiology is *homo economicus*. Here the assumption is that humans pick their sexual behavior on the basis of a rational benefit/cost calculation. This assumption allows one to study the potential behavioral responses of individuals with respect to particular policies (e.g., adjustments in the number of casual relationships prior to marriage in response to changes in transmission risk). Second, at the heart of the HIV/AIDS epidemic is an externality, the transmission of a virus. General equilibrium modelling is well suited for the study of externalities. One can analyze how individual shifts in behavior feed back on each other in equilibrium (e.g., if the rate of infection for some individuals rise, then the rate of infections for their partners may increase in turn, etc.). Thus, the great advantage of choice-theoretic equi-

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<sup>1</sup>Section 2 is devoted to providing background on the Malawian HIV/AIDS epidemic.

<sup>2</sup>A detailed review of the literature is contained in Section 1.1.

librium modelling is the joint assessment of both behavioral change and equilibrium adjustment in response to proposed policy interventions. Nothing is for free, of course. Some abstractions are needed to operationalize choice-theoretic equilibrium modelling that may render it less rich along some dimensions. As such, choice-theoretic equilibrium modelling complements the above two existing approaches to the study of disease transmission.

A choice-theoretic general equilibrium search model is built here to study the Malawian HIV/AIDS epidemic. Despite the fact that computational general equilibrium models have made great progress in recent decades, there is essentially no application to the field of disease transmission (except for few purely theoretical studies that form the basis of departure for this analysis but are too stylized to be directly mapped to data; see the review in the next section). The first applied general equilibrium framework to be used for this purpose is developed here.

The constructed model has three main ingredients. First, individuals make rational choices about risky sexual behavior. In contrast to most of the literature (discussed below), the model explicitly models three important margins of risky behavior. Individuals choose the frequency of sex, whether to get married or engage in casual sex, and whether to use a condom or not. These margins allow people to choose those types of behavior that are thought to be the most important factors in disease transmission: having sex often, with many different people, and not using a condom. Alternatively, people can engage in relatively safe behavior by being abstinent, getting married, or at least by using a condom. When making these choices, individuals take the riskiness of each type of activity into account.

Second, beliefs about the riskiness of various forms of sexual activity are formed rationally. In the analysis a person's past sexual history is private information. Still, the fact that someone desires a casual sexual encounter involving no condom, say, as opposed to seeking a long-term one, may signal something about his 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. An individual's choice about what type of sexual activity to engage in may be influenced by his or her belief about their own health. People assess their odds of having HIV/AIDS rationally using Bayes' rule. They understand how various sexual activities affect their future health, which influences their current decisions about which type of sexual behavior to engage in. 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 seeking short-term unprotected sex and amplify the risk of a relationship with them.

Third, the analysis is general equilibrium in nature. There are "markets" or "meeting places" for the different types of sexual activities. People have differing tastes over various types of relationships and search accordingly on the various markets to fulfill their desires. They can choose how intensively to search for a partner on a particular market (or abstain by not searching). They do this recognizing that some of these meeting places 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. The model embeds the well-documented fact of monetary transfers within relationships. Each market is characterized by a transfer that one of the partners makes to the other. These transfers depend on the number of men relative to the number of women that seek a particular type of relationship, and equilibrate the two sides of the market. For example, in the absence of transfers there may be more men desiring casual unprotected sex than women. To attract women towards this risky activity, men may have to make some form of transfer payment. The market structure eliminates any joint decision problem between partners about whether or not to use a condom. They have the same desires when choosing the same market.

The constructed model is then tuned to fit aspects of the Malawian data. In particular, it is calibrated to match the HIV/AIDS rates for men and women, the fraction of sexual relationships that are short term, the fraction of short-term sexual encounters that use a condom, and the fraction of deaths that arise from HIV/AIDS. The model's ability to match some *non-targeted* lifecycle observations is then examined. These include the profile by age of a symptom-free person's be-

lief about being HIV/AIDS infected, the HIV/AIDS infection rate by age, and the likelihood of a casual sexual encounter by age. The model does very well at matching the data along these dimensions.

Last, some policy experiments are conducted. To name some, policies are examined such as male circumcision, which some believe reduces the risk of HIV/AIDS transmission from females to males, the promotion of condom use in casual sex, and income transfers to females. Earlier purely theoretical work (discussed in the following literature section) focused on reductions in transmission risk and emphasized that it can lead to higher overall prevalence because people could get excessively risky. The quantitative part of this study does not confirm that behavioral adjustments fully negate the positive effects of reduced transmission risk. Nevertheless, it is important to account for behavioral adjustments and equilibrium effects as they do strongly affect the predicted effectiveness. To show this, we explicitly simulate epidemiological studies (which ignore behavioral adjustments) and small scale field experiments (which ignore equilibrium effects) within our model. As an example, focus on the treatment of other sexually transmitted diseases (STDs) which reduces the transmission risk. The effectiveness of this policy is over-predicted (by around 30%) by the epidemiological model and under-predicted (by around 80%) by the field experiment. For other policies the behavioral responses are larger, and indicate the possibility of negating the positive primary effect. For example, the rate of HIV/AIDS displays a hump-shaped relationship in the psychic pleasure that people get from sex using condoms.

This study also showcases channels beyond the simple effect that some agents increase their risky sexual activity. For example, some forms of promoting long-term relationships seem to raise HIV prevalence because some risky people now join the previously safe haven of marriage. While their move towards marriage is usually seen as a reduction in their own risky behavior, it increases the infection risk for their marriage partners who previously had a higher chance of finding a safe match. Such a change in the mixing patterns seriously affects the effectiveness of the policy. For other interventions such one-sided reduction in transmission risk (e.g., male circumcision) or increases in income (especially for women) the behavioral adjustment and equilibrium effects magnify the efficacy of the policy: Especially women react strongly to higher incomes, which makes

them substantially less willing to engage in risky activities. These changes feed back on the men leading to a virtuous feedback loop.

Overall, this research program aims to develop tools to aid researchers and practitioners in their attempts to think through the various channels that are present in different interventions, and highlights areas where further and more in-depth research should be conducted to assess with more confidence the magnitude of these channels.

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

## 1.1 Relationship to the Literature

This appears to be the first quantitative general equilibrium model of disease transmission with purposive decision making. Previous papers are either purely theoretical or do not consider the endogeneity of rational human behavior. In addition, there are purely empirical studies; in particular, a recent literature uses field experiments to study HIV/AIDS prevention policies.

The economic literature on disease transmission is small. Philipson and Posner (1993) provide the first economic analysis of HIV/AIDS. Their book gives a great overview of the different aspects involved in treating HIV/AIDS – from testing, to regulatory intervention, to medical research and the political economy of treatment. Most of their analysis is verbal, however.

Few formal models of risky sexual behavior and HIV / AIDS exist.<sup>3</sup> For tractabil-

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<sup>3</sup>There is of course a large economic literature on mate selection, starting from Becker (1973). Examples include Burdett and Coles (1997), Shimer and Smith (2000) and Boulier and Rosenzweig (1984). The current paper also connects to the simulation-based analysis of sexual behavior in Greenwood and Guner (2010). However, none of these papers study disease transmission.

ity, most of this work has focussed on only one dimension of risky behavior and abstracts from intertemporal considerations of an individual over his lifetime. Most prominently, Kremer (1996) builds a theoretical model where the number of partners is determined endogenously as a choice by rational agents that depends on the prevalence of the disease. The paper focuses on the theoretical implications of such endogeneity and shows that a reduction in the transmission probability can increase the overall prevalence rate because individual agents behave in a more risky fashion. Moreover, it is shown that changes in mixing pattern might be an important channel affecting the overall prevalence rate. To derive these predictions theoretically, the paper abstracts from a number of important issues. People only consider the lifetime probability of contracting HIV/AIDS, but do not care about the exact timing. In particular, people do not update their behavior based on past sexual experience. Condom use is not explicitly modeled, which abstracts from the joint decision problem in using a condom. A distinction between long-term and short-term partners is not made. Finally, the model is a one-gender model that makes the analysis of gender asymmetries in the disease dynamics impossible. 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.

The only other model that explicitly models men and women is Magruder (2011) who develops a Jovanovic (1979) style matching model of marital search to analyze 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. In his setting, however, 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 her or his past sexual history. These margins may be important because healthy (young) individuals might self-select into the safety of marriage while those who believe they are infected have less to gain from safety and might opt for more risky alternatives.

While the economic literature on disease transmission is small, there is a large



literature on disease transmission and specifically HIV/AIDS in epidemiology. The critical difference to the economic literature is that epidemiological models do not model decision-making.<sup>4</sup> In other words, these models do not try to understand why people engage in risky sexual behavior and how this behavior changes in response to changes in the environment. Instead, they take sexual behavior 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. Survey evidence and several empirical studies suggest that people react to a higher presence of HIV/AIDS by adjusting the number of partners, the type of sexual relationships, and the protective measures that they use [Wellings et al. (1994)].

Since economic studies of HIV/AIDS augment epidemiological models with decision-making, a brief review of the epidemiological literature is given here. The workhorse 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 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.<sup>5</sup> 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.

An interesting contribution is Kremer and Morcom (1998) who introduce selective mixing into an SI model: 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 others who are seeking

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<sup>4</sup>Very recently there have been a few attempts to incorporate decision-making into epidemiological models. However, these are pure theoretical contributions that study a generic infectious disease, rather than modeling the specific channels through which HIV/AIDS is transmitted. Examples include Klein et al. (2007), Fenichel et al. (2011), and Toxvaerd (2012).

<sup>5</sup>Recovery and either resistance against further infection, or the possibility of reinfection, are explicitly modeled for other infectious diseases. See Hethcote (2000) for a comprehensive overview of the mathematical modeling of infectious disease.

the same type of relationship allows for selective mixing and avoids modeling any conflict of interest in relationship formation.

Many papers simulate SI models numerically to forecast the disease incidence in the future or to study the effectiveness of prevention policies. For example, Low-Beer and Stoneburner (1997) use such a model to forecast HIV incidence in East Africa; Johnson (2008) studies the role of other sexually transmitted diseases for the epidemic; Clark and Eaton (2008) simulate the effectiveness of male circumcision; and Bracher, Santow, and Watkins (2004) study the importance of condoms. By design, none of these studies takes behavioral changes in response to the reduced transmission risks into account.

There is also a recent empirical literature that studies HIV/AIDS prevention policies using mostly field experiments, and sometimes natural experiments or cross-country data. See Behrman and Kohler (2012) for an excellent survey.<sup>6</sup> Some of the field experiments are close to the policy experiments conducted in this paper. For example, several field experiments find that male circumcision lowers the infection risk for males [see, e.g., Auvert et al. (2005) and Gray et al. (2007)]. Based on cross-country data, Oster (2005) argues that the treatment of other STDs reduces the infection risk and thereby lowers the incidence of HIV/AIDS. Auld (2006) analyzes how risky sexual behavior changes with local infection rates using data from San Francisco in the 1980s and finds large elasticities. Again in a field experiment, Duflo, Dupas, and Kremer (2012) evaluate three school-based HIV/AIDS interventions in Kenya. They find that subsidizing education reduces teenage pregnancy but not HIV infections. The interpretation is that the program reduced the number of pregnancies within marriage, but did not affect casual sex. This finding underlines the importance of distinguishing committed from casual relationships, as we do in our model.

Other empirical studies analyze testing, treatment, or information campaigns which are outside the scope of this paper. Lakdawalla, Sood, and Goldman (2006) study the effects of anti-retroviral drugs and find that these led to an increase in sexual behavior in the U.S. Thornton (2008) uses evidence from a randomized field experiment in rural Malawi to conclude that testing is not a very

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<sup>6</sup>See also Padian et al. (2010) for a summary of randomized controlled trials.

cost-effective prevention policy. Dupas (2011) shows in a randomized field experiment in Kenya that teenage girls who are given information about the HIV status of different groups of men respond by shifting sexual behavior to the lower risk groups. De Walque (2007) finds, based on evidence from Uganda, that HIV/AIDS information campaigns have a larger impact on more educated people.<sup>7</sup> Ashraf, Bandiera, and Jack (2013) conduct a field experiment that evaluates different incentive schemes for hairdressers to sell condoms as a prevention policy. While it is found that non-financial incentives are a good motivator to sell condoms, the impact on HIV/AIDS is not explicitly studied. Recently a few studies have analyzed the impact of conditional cash transfers on HIV/AIDS (De Walque et al. 2012). The evidence of the effectiveness of such programs (where people get paid when they continue to test negative for sexually transmitted diseases) to date is weak at best.

The current paper provides a useful complement to the empirical approach. Theory alone cannot infer the elasticity of sexual behavior in response to policy, for example. On the other hand, a purely empirical approach has other shortcomings. Typically a randomized field experiment is conducted on a small subgroup of the population which does not allow for general equilibrium effects due to adjustments in prevalence, prices, and sorting patterns. This might be particularly important in the context of HIV/AIDS which may affect men and women differently, but often data is collected on one gender only, as in many studies of male circumcision.

Finally, while the focus here is on the disease itself, several people have analyzed the importance of HIV/AIDS for development or lack thereof (Young 2005; Santaaulalia-Llopis 2008).

## **2 Families, Sexual Behavior, 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 epi-

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<sup>7</sup>The relationship between education and HIV status is also analyzed in Iorio and Santaaulalia-Llopis (2011).

demic 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 suffers greatly from the HIV/AIDS epidemic.<sup>8</sup> 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 HIV rate has been roughly constant (ranging between 12-14%) since the mid 1990s, yielding some indication that the disease dynamics have settled into a 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. This fact is ignored here. 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. By contrast two thirds of the infected population is male in the Western world—see *World Development Indicators* (2009). In the West, HIV is largely a disease of drug users and homosexuals. In SSA, by comparison, 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-positive, compared to just 0.4% for men in the same age category.

A rational model of HIV only makes sense if people understand what HIV is, are

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<sup>8</sup>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.

aware of how it gets transmitted, and know 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 is a reasonable approximation for the Malawian epidemic.

Sexual behavior conducive to 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—30.1% of women reported using a condom during their last sexual activity, compared to 47.1% 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. Undie, Crichton, and Zulu (2007) single out the following quote from a female teenager in order to capture the overall attitude that they found towards varying partners in their interviews: “[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.’”. Furthermore, divorce is relatively common. Reniers (2003) reports that 45% of marriages end in divorce within 20 years. The quantitative evidence from the DHS suggests that men engage in more risky sexual behavior than women. This difference might be partially due to a social bias as to what is acceptable behavior. Miller, Watkins, and Zulu (2001) analyze gender differences in survey responses in Malawi and find that when husband and wife answers to a variety of questions 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 caution. Several other forms of risky behavior will be abstracted from in the paper. For example, the model does not have concurrent relationships, such as extra-marital affairs or polygyny, both of which are relatively common in Malawi. In 2004, 8.3% of all married men admitted to having had an affair in the last year. Women admit to much fewer affairs. Polygyny is also fairly common in Malawi.

As recently 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. Undie, Crichton, and Zulu (2007) highlight this with the following quote from an interview with a Malawian female about protected sex: "You can't eat [candy] while it's in the wrapper. It doesn't taste [good]." 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 that 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 a 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 an 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 receive sex, and they expect it sooner rather than later. Transfers are not made directly before or after sex (as with prostitution), however; 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 developed here 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 Malawi Diffusion and Ideational Change Project (MDICP). This has led 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. As motivated in Section 2, sex is always unprotected in long-term relationships. Further, 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  $u \geq p > 0$ . The utility flow in a long-term relationship is  $u+l$ , where  $l$  may be negative. A positive  $l$  can be interpreted as a taste for long-term attachment, while a negative  $l$  signifies taste for variety in partners. Individuals also realize utility from the consumption of goods. Let this utility be given by  $\ln(c)$ , where  $c$  is consumption. Each period a person receives income in the amount  $y$ . There is no borrowing or lending in the economy. An individual discounts the future with a stochastic factor that takes two values, viz  $\tilde{\tau}$  and  $\tilde{\beta}$  with  $\tilde{\tau} \leq \tilde{\beta}$ . Individuals start off life with the low rate  $\tilde{\tau}$ . This low factor reflects the impatience of youth, which may lead to a predilection to engage in risky behavior. Then, at every period, a person may switch permanently to the high factor with probability  $\eta$ . Additionally, there is a probability  $\delta$  that an individual dies from natural causes

in a period. Thus, the effective discount factors are given by  $\iota = \tilde{\iota}(1 - \delta)$  and  $\beta = \tilde{\beta}(1 - \delta)$ . The values of  $l, p, u, y, \beta$ , and  $\iota$  may differ across individuals of a given gender. The set of fixed characteristics for a person is denoted by  $x = (l, p, u, y, \beta, \iota)$ , which will be called a person's type.

People can search for partners in different markets.<sup>9</sup> 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  $\pi_l$ . The individual can pick these odds at an increasing cost in terms of lost utility. These search costs are given by  $C_l(\pi_l) = \omega_l[\pi_l/(1 - \pi_l)]^{\kappa_l+1}$ , where  $\kappa_l \geq 0$  and  $\omega_l > 0$ . 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  $\xi$ . If the person is unsuccessful at finding a long-term mate s/he then enters the short-term market, where s/he can still engage in sexual behavior for this period. 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  $\pi_p$  and  $\pi_u$  represent the odds of finding a partner in the protected and unprotected markets for short-term relationships, which will be choice variables. The cost of searching in each market is given by  $C_s(\pi_p)$  and  $C_s(\pi_u)$ , which have the same functional form as  $C_l(\pi_l)$ , but where the parameters  $\kappa_s$  and  $\omega_s$  are allowed to differ from the long-term market. The total cost of searching for a short-term partner will then be  $C_s(\pi_p) + C_s(\pi_u)$ . Assume that an 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  $\pi_p = \pi_u = 0$ .

Given the pervasive evidence on gift giving in the context of sexual relationships (see Section 2), transfers are exchanged for sex. Associated with each market is a transfer payment,  $t$ , that is made between the two partners. For the person making the transfer,  $t$  will be positive, while it will be negative for the individual receiving it. Think about the people receiving the transfers as supplying relationships on the market, and those paying transfers as demanding them. Interpret

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<sup>9</sup>The idea that people can rationally target their search behavior to particular markets is present in many recent theoretical models (e.g. Jacquet and Tan (2007), Eeckhout and Kircher (2010), Gautier, Svarer, and Teulings (2010)).



the transfer as representing the inputs into a relationship: affection, entertainment, gifts, etc. The magnitude of this transfer is determined in equilibrium.<sup>10</sup> 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.

People form beliefs about their own infection status.<sup>11</sup> A person enters a period with a prior belief about the likelihood of not being infected with HIV/AIDS. Denote this prior belief by  $\phi$ , which is private information.<sup>12</sup> The person then may have a relationship involving either protected or unprotected casual sex or enter a long term relationship. The risk of catching HIV/AIDS from an infected person is different for protected vs. unprotected sex. If the individual has sex with an HIV/AIDS infected person then the virus will get transmitted with probability  $1 - \gamma$ , where  $\gamma$  differs across the types of sex and by gender. 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  $\alpha$ . 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, and (iii) in the case of marriage, whether or not he observed symptoms in his partner.

Let the remaining lifetime utility for a person with the symptoms of HIV/AIDS be represented by  $A$ .<sup>13</sup> Assume that a person stricken with HIV/AIDS symptoms engages in no further relationships. The probability that a person displaying symptoms dies is  $\delta_2$ . Since a person with HIV/AIDS symptoms engages in no further activity,  $\delta_2$  does not appear in the value functions. However, it is relevant for computing the average HIV/AIDS rate in society. Note that in the framework

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<sup>10</sup>Alternatively, market clearing could be achieved through different meeting probabilities. This should lead to qualitatively similar results.

<sup>11</sup>The importance of beliefs are emphasized in Delavande and Kohler (2011).

<sup>12</sup>As in Kremer (1996) the current work assumes that characteristics such as age, past sexual activity and sexual preferences cannot be observed and hence no inference about the infection status of a partner can be made.

<sup>13</sup>Note that  $A$  cannot be too large to assure people prefer being healthy over being sick.

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, namely  $l, p, u, y, \beta$ , and  $\iota$ . 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  $\mu$  type- $x$  individuals are born at the beginning of each period.

Before proceeding on to the formal analysis some notation will be defined. An individual will be indexed by his prior that he is healthy,  $\phi$ , his current discount factor,  $d$ , and his exogenous type  $x$ . Let  $\tilde{V}_r^d(\phi, x)$  denote that lifetime utility for a person with prior  $\phi$ , 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. The timing of events is summarized in Figure 6 in Appendix A. Attention will now be directed toward the determination of the functions  $\tilde{V}_r^d(\phi, x)$  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. Recall that there are young and old individuals that differ in their discount factor. Start with a type- $x$  old person (i.e. with discount factor  $\beta$ ), who has failed to match on the short-term sex markets. Thus, he will be abstinent for the current period. Note that the individual's discount factor will remain high forever. The value function for this person is given by

$$\tilde{V}_a^\beta(\phi, x) = \ln(y) + [1 - (1 - \phi)\alpha]\beta V_l^\beta(\phi', x) + (1 - \phi)\alpha\beta A, \quad (1)$$

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

The first term on the righthand side of (1) gives the person's flow utility from current consumption. Two things can happen next period, as the next two terms illustrate. Even though the individual does not 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,  $\phi$ , about his probability of being non-infected. Therefore, he will develop symptoms next period with probability  $(1 - \phi)\alpha$ . In this event the person will realize a utility level of  $A$ , which is discounted at rate  $\beta$ . This reasoning 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. The discounted expected utility from searching on the long-term market next period with prior  $\phi'$  is given by  $V_l^\beta(\phi', x)$ . This logic accounts for the second term in the equation. Also, when the individual does not suffer the symptoms of HIV/AIDS, he updates his prior in a Bayesian fashion to  $\phi'$  summarized by the function  $\Phi_a(\phi)$ , which will be explained fully in Section 3.3. 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.

Next, consider the case of an abstinent young person (with discount factor  $\iota$ ). The discount factor may switch next period to the high value,  $\beta$ , with probability  $\eta$ , or remain at the low one,  $\iota$ , with probability  $1 - \eta$ . It is easy to see that the value function for a type- $x$  person with a low discount factor,  $\iota$ , and a prior  $\phi$ , will now read

$$\begin{aligned} \tilde{V}_a^\iota(\phi, x) = & \ln(y) + [1 - (1 - \phi)\alpha]\iota[\eta V_l^\beta(\phi', x) + (1 - \eta)V_l^\iota(\phi', x)] \\ & + (1 - \phi)\alpha\iota A, \end{aligned} \quad (2)$$

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  $\beta$ . If  $s = p$

then the person will use a condom and enjoy utility  $p$  from sex. If  $s = u$  the individual will enjoy  $u$  from unprotected sex. 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 a person desires a short-term sexual relationship that does not 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 about his own health status,  $\phi$ , 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  $1 - \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)]$ .

Summarizing, the value function for a type- $x$  individual with prior  $\phi$  who is currently having a short-term relationship in market  $s$  is given by

$$\begin{aligned} \tilde{V}_s^\beta(\phi, x) &= \ln(y - t_s) + pI(s) + u[1 - I(s)] \\ &\quad + \{1 - \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)]\} \beta V_t^\beta(\phi', x) \\ &\quad + \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)] \beta A, \end{aligned} \quad (3)$$

with

$$\phi' = \Phi_s(\phi), \text{ for } s = p, u. \quad (4)$$

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 in Section 3.3. Re-

call that the person's exogenous type,  $x$ , determines his tastes for protected sex, unprotected sex, short-term relationships, and his level of income. The dependence of  $p$ ,  $u$ ,  $y$ , and  $\beta$  on  $x$  in the value functions (1) and (3) is suppressed for convenience. Similarly, the gender of the individual is also 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.

Next, consider the case of a young person (with discount factor  $\iota$ ). The discount factor will switch next period to the high value,  $\beta$ , with probability  $\eta$ , or remain at the low one,  $\iota$ , with probability  $1 - \eta$ . Therefore, the analogue to (3) is

$$\begin{aligned} \tilde{V}_s^\iota(\phi, x) = & \ln(y - t_s) + pI(s) + u[1 - I(s)] \\ & + \{1 - \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)]\} \iota [\eta V_l^\beta(\phi', x) + (1 - \eta) V_l^\iota(\phi', x)] \\ & + \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_s)(1 - \gamma_s)] \iota A, \end{aligned} \quad (5)$$

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  $\pi_p$  and  $\pi_u$ . This is done in accordance with the following problem.

$$\begin{aligned} V_s^d(\phi, x) = & \max_{\substack{0 \leq \pi_u^d, \pi_p^d \leq 1, \\ \pi_u^d + \pi_p^d \leq 1}} \{ \pi_p^d \tilde{V}_p^d(\phi, x) + \pi_u^d \tilde{V}_u^d(\phi, x) + (1 - \pi_p^d - \pi_u^d) \tilde{V}_a^d(\phi, x) \\ & - C(\pi_p^d) - C(\pi_u^d) \}, \text{ for } d = \iota, \beta. \end{aligned} \quad (6)$$

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

### 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, and the partnership endures until some form of exogenous breakup occurs. Suppose that the person entered this relationship  $n$  periods ago with prior  $\phi$ . At the

end of period  $n$  this relationship can breakup for three reasons (besides natural death).<sup>14</sup> First, it could transpire that both people are symptom free and that the relationship terminates due to an exogenous breakup which happens with probability  $\xi$ . 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.

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

$$\begin{aligned} \tilde{V}_l^\beta(\phi, x) &= \ln(y - t_l) + u + l & (7) \\ &+ \sum_{n=1}^{\infty} \beta^n (1 - \xi)^n \Pr[\text{no symptoms in either person, end period } n | \phi] [\ln(y - t_l) + u + l] \\ &+ \sum_{n=1}^{\infty} \beta^n (1 - \xi)^{n-1} \xi \Pr[\text{no symptoms in either person, end period } n | \phi] V_l^\beta(\Phi_l^h(\phi, n), x) \\ &+ \sum_{n=1}^{\infty} \beta^n (1 - \xi)^{n-1} \Pr[\text{symptoms just in partner, end period } n | \phi] V_l^\beta(\Phi_l^A(\phi, n), x) \\ &+ \sum_{n=1}^{\infty} \beta^n (1 - \xi)^{n-1} \Pr[\text{symptoms in person, end period } n | \phi] \times A. \end{aligned}$$

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 term gives 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 C.2 of the Appendix, as are the formulae for the other probabilities shown. The rest of the expression enumerates the expected utilities

<sup>14</sup>For simplicity assume that both partners die together; i.e., with probability  $\delta$  the pair dies, and with probability  $(1 - \delta)$  both survive.

associated with breakup events in period  $n + 1$ . In particular, the third line 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, where he starts by searching in the long term market. At that time he updates his prior according to the function  $\Phi_l^h(\phi, n)$ . 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 term, line 4. In this situation the individual will update using the function  $\Phi_l^A(\phi, n)$ . The formulae for  $\Phi_l^h(\phi, n)$  and  $\Phi_l^A(\phi, n)$  are specified in Appendix C.1. Finally, HIV/AIDS may manifest itself in the individual at the end of some period  $n$ . This is captured by the last line. Also recall that the odds of dying from a natural death are incorporated into  $\beta$ .

The value of a long-term relationship for a person with a low discount factor  $\iota$  is determined analogously. One now must take into account that the discount factor may switch at some future date from  $\iota$  to  $\beta$ . The expression for  $\tilde{V}_l^\iota(\phi, x)$  is now slightly more complicated. It is developed in Appendix C.3 – see (21). The value of searching in the long-term market for a type- $(\phi, x)$  person with discount factor  $d$  is given by

$$V_l^d(\phi, x) = \max_{\pi_l^d} \left[ \pi_l^d \tilde{V}_l^d(\phi, x) + (1 - \pi_l^d) V_s^d(\phi, x) - C(\pi_l) \right], \text{ for } d = \iota, \beta \quad (8)$$

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

### 3.3 Belief Updating

The easiest case for belief updating is a person who was abstinent in a given period. The function for updating the prior,  $\Phi_a(\phi)$ , is given by Bayes' rule:

$$\begin{aligned} \Phi_a(\phi) &= \frac{\Pr(\text{not being infected this period} \mid \phi)}{\Pr(\text{not observing any symptoms this period} \mid \phi)} \\ &= \frac{\phi}{\phi + (1 - \phi)(1 - \alpha)} = \frac{\phi}{1 - (1 - \phi)\alpha}. \end{aligned} \quad (9)$$

The prior probability of not being infected is  $\phi$ . The probability of not observing any symptoms has two components: the odds of not having HIV/AIDS,  $\phi$ , 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.

After engaging in a short-term relationship, Bayes' rule says that the prior should be updated according to the formula

$$\begin{aligned}\Phi_s(\phi) &= \frac{\text{Pr}(\text{not being infected this period} \mid \phi)}{\text{Pr}(\text{not observing any symptoms this period} \mid \phi)} & (10) \\ &= \frac{\phi\bar{\phi}_s + \phi(1 - \bar{\phi}_s)\gamma_s}{\phi\bar{\phi}_s + (1 - \phi)(1 - \alpha) + \phi(1 - \bar{\phi}_s)[(1 - \gamma_s)(1 - \alpha) + \gamma_s]}\end{aligned}$$

for  $s = p, u$ . There are two reasons why the individual might not have HIV/AIDS. Perhaps neither him nor his partner had it. The odds of this are  $\phi\bar{\phi}_s$ . Or, maybe his partner does have it, but it failed 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 develops symptoms at the end of the current period.

Finally, updating in long-term relationships is relevant only when the relationship ends. Relationships can end for three reasons: an exogenous breakup, the person develops symptoms, or the partner develops symptoms. Obviously if a person develops symptoms, he knows that he is sick and no further updating is necessary. Denote Bayesian updating for the other two cases by  $\Phi_t^h(\phi, n)$  and  $\Phi_t^A(\phi, n)$  respectively. The exact formulae are given in Appendix C.1.



### 3.4 Stationary Equilibrium

A stationary equilibrium for the developed framework will now be formulated.<sup>15</sup> First, the equilibrium type distributions for singles will be specified. Let  $\mathcal{S}^d(\phi; x)$  represent the (non-normalized) stationary distribution of singles at the beginning of a period. It denotes the measure of singles of type- $x$  that have prior  $\phi$  and discount factor  $d$ . Similarly, let  $\mathcal{L}^d(\phi'; x)$  denote the distribution over the prior  $\phi'$  for type- $x$  individuals with discount factor  $d$  who exit long-term relationships at the end of a period. Given some distributions  $\mathcal{S}$  and  $\mathcal{L}$  of singles and married people, the sexual behavior of individuals according to their decision rule  $\Pi$  [ $\Pi_{g,r}^d = \Pi_{g,r}^d(\phi, x)$  for each type] gives rise to a new distribution of singles and married people, which can be described by a mapping  $\mathbf{T}$  that is characterized fully in Section C.4 of the Appendix—see equations (22) to (28). In steady-state the distributions of singles and married people remain constant, and are determined by a fixed point of this operator:

$$(\mathcal{S}^\beta, \mathcal{L}^\beta, \mathcal{S}^\iota, \mathcal{L}^\iota) = \mathbf{T}(\mathcal{S}^\beta, \mathcal{L}^\beta, \mathcal{S}^\iota, \mathcal{L}^\iota; \Pi). \quad (11)$$

Next, the prevalence rates in each market have to arise from the aggregation of individual choices. 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. The number of market participants for sexual activity  $r$  ( $= l, p, u$ ), who are of gender  $g$  and type- $x$  with belief  $\phi$  and discount factor  $d$ , is given by

$$\mathcal{M}_{g,r}^d(\phi, x) \equiv \begin{cases} \Pi_{g,r}^d(\phi, x) \mathcal{S}_g^d(\phi; x), & \text{if } r = l, \\ [1 - \Pi_{g,l}^d(\phi, x)] \Pi_{g,r}^d(\phi, x) \mathcal{S}_g^d(\phi; x), & \text{if } r = p, u. \end{cases} \quad (12)$$

The number of market participants equals the number of singles times their probability of participating in a particular market. For the short-term market this also entails the probability of not previously finding a long-term partner within the current period. The odds that a person of gender  $g$  within market  $r$  is not infected equals the fraction of healthy market participants among the total number

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<sup>15</sup>The restriction to steady states seems justified since prevalence rates in Malawi have remained roughly constant in recent years.

of market participants:

$$\bar{\phi}_{g,r} = \frac{\sum_d \sum_x \sum_{\phi} \phi \mathcal{M}_{g,r}^d(\phi, x)}{\sum_d \sum_x \sum_{\phi} \mathcal{M}_{g,r}^d(\phi, x)}, \text{ for all } g \text{ and } r. \quad (13)$$

Note that  $\bar{\phi}_{f,r}$  denotes the non-prevalence among women, which is relevant for *men* when determining their odds of getting infected. Similarly,  $\bar{\phi}_{m,r}$  refers to the odds among men, but is relevant for the *women* when making their decisions.

Market clearing means that the number of female participants equals the number of male participants in any market:

$$\sum_d \sum_x \sum_{\phi} \mathcal{M}_{f,r}^d(\phi, x) = \sum_d \sum_x \sum_{\phi} \mathcal{M}_{m,r}^d(\phi, x), \text{ for all } r. \quad (14)$$

Additionally, a transfer paid by one gender on a market is a transfer earned by the other so that

$$t_{f,r} + t_{m,r} = 0, \text{ for all } r. \quad (15)$$

This leads to the following formal definition of equilibrium.

**Definition 1** *A stationary equilibrium is described by a set of decision rules for search effort,  $\Pi_{g,r}^d(\phi, x)$ , a set of transfer payments,  $t_{g,r}$ , a set of stationary type distributions,  $\mathcal{S}_g^d(\phi; x)$  and  $\mathcal{L}_g^d(\phi', x)$ , 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,  $\Pi_{g,r}^d(\phi, x)$ , satisfy the appropriately gender subscripted versions of the generic problems (6) and (8), taking as given transfer payments and HIV/AIDS prevalence rates;*
2. *The stationary type distributions,  $\mathcal{S}_g^d(\phi; x)$  and  $\mathcal{L}_g^d(\phi, x)$ , solve the appropriately gender subscripted version of (11) using (12);*
3. *The prevalence rates for HIV/AIDS on each market,  $\bar{\phi}_{g,r}$ , are given by (13) using (12);*
4. *The transfer payments,  $t_{r,g}$ , are such that the markets for all types of relationships clear according to (14). Additionally, the flow of transfers across the genders must balance as specified by (15).*

## 4 Calibration

To address the HIV/AIDS epidemic in Malawi, the model is analyzed numerically. A benchmark simulation is constructed that displays features that are broadly consistent with the Malawian case. In particular, the simulated model has an HIV/AIDS infection rate that corresponds with the Malawian data, a proportion of casual sexual encounters that is approximately the same, and a reasonable fraction of these encounters use a condom.

Interpret a model period as lasting one quarter. Even though the model is set up to allow for heterogeneity along many dimensions, such as the utility from sex, income and the discount factor, only two dimensions of heterogeneity are exploited in the application. First, assume that people differ in their discount factor. Second, suppose that men and women have different transmission rates, so that the same level of sexual activity leads to a discrepancy in the HIV rate across genders. This limited degree of heterogeneity economizes on the number of parameters to be specified.

The calibration is conducted in three steps. First, to the extent possible, parameters with direct data analogs are taken from the literature. In particular, all parameters relating directly to the biology of the disease are chosen in this way. Second, the remaining parameters are chosen to match 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. Using the micro data from this survey, a number of statistics are computed regarding HIV prevalence rates, sexual behavior, marital status, etc. Finally, the model's predictions are compared to the data along several non-targeted dimensions. The model performs surprisingly well, which can be interpreted as an additional validity check.

### 4.1 Parameters Based on Direct Evidence

The most important parameter values for the simulation are those concerning HIV/AIDS. Fortunately, for the most part, these can be taken from the medi-

cal literature. The transmission risk for one-time male unprotected sex is taken to be 2.3 per 1,000. This number falls in the range of estimates reported by a variety of studies.<sup>16</sup> Since couples on average have sex 9 times a month, as reported in Gray et al. (2001), this translates into a quarterly non-transmission risk of  $\gamma_u^m = 0.94$ . The transmission risk when condoms are used is obviously lower, but protection is far from perfect—Bracher, Santow, and Watkins (2004). Select  $\gamma_p^m = 0.98$ , corresponding to a 67% efficacy rate, which is in line with Weller (1993) who conducted a meta-analysis of condom efficacy. Further, for physiological and anatomical reasons, and in accord with the medical evidence, females are assumed to have a higher risk of contracting HIV than males. Nicolosi et al. (1994) reports a risk that is 2.3 times as high for women. However, the range of estimates is wide. On the one extreme, Gray et al. (2001) find no statistically significant difference between transmission rates by gender. On the other extreme, Padian, Shiboski, and Jewell (1991) calculate a factor as high as 20. Erring on the conservative side, pick  $\gamma_p^f = 0.965$ , which corresponds to women being 75% more likely to get infected. Using the same gender gap in transmission also for unprotected sex, set  $\gamma_u^f = 0.895$ .

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 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%.<sup>17</sup> A rate twice this number is used: First, polygyny is fairly common in Malawi, from which the analysis abstracts. Second, extra-marital affairs are relatively common as well. Therefore, interpret, for example,

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<sup>16</sup>For example, the annual report of the UNAIDS Joint United Programme on HIV/AIDS (2007) gives a range of 2 to 6 per 1,000, depending on whether other STDs are present or not. Baeten et al. (2005) also report a transmission risk of 6 per 1,000. Gray et al. (2001) report a somewhat lower number of 1.1 per 1,000 for Uganda; however, free condoms were distributed as part of the study. Wawer et al. (2005) finds transmission rates as high as 82 per 1,000 during the first few months after infection.

<sup>17</sup>A similar number is also reported by Reniers (2003).

a 10-year marriage with one affair as two long-term relationships with a third casual one in between. Section 5.2.3 explores what happens to equilibrium outcomes when the risk of divorce is lower.

The quarterly (non-HIV related) death hazard is taken to be  $\delta = 0.006$ . A study conducted by the U.S. Census Bureau (2004) reports a life expectancy without HIV of 56.3 years for Malawi. Since the model starts at age 15, the quarterly death hazard is selected to match a life expectancy of 41.3 years. Malawi is a very poor country. Set  $y = 320$  which corresponds roughly to quarterly GDP per working age population (note that only about half the population is of working age in Malawi). Table 1 summarizes the preceding paragraphs by listing all parameters that are set a priori.

Table 1: PARAMETERS CHOSEN OUTSIDE THE MODEL

<i>Parameter</i>	<i>Value</i>	<i>Interpretation</i>
$\gamma_u^m$	0.94	6% quarterly transmission risk, unprotected sex, men
$\gamma_p^m$	0.98	2% quarterly transmission risk, protected sex, men
$\gamma_u^f$	0.895	10.5% quarterly transmission risk, unprotected sex, women
$\gamma_p^f$	0.965	3.5% quarterly transmission risk, protected sex, women
$\alpha$	0.025	10 years from infection to symptoms
$\delta$	0.006	6% quarterly death risk
$\delta_2$	0.125	2 years from symptoms to death
$\xi$	0.03	3% quarterly divorce hazard
$y$	320	quarterly income

## 4.2 Parameters Chosen to Match Data Moments

The remaining parameters have no clear data analogues. 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 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 ( $l < 0$ ). These parameters are picked to match several facts

related to sex, marriage and HIV/AIDS in Malawi.<sup>18</sup>

As specified above, the only exogenous heterogeneity (in addition to the gender difference in transmission risk) is the degree of patience people have. Assume that the young are more impatient than the old and that there is a small subset of the population that is extremely impatient.<sup>19</sup> Recall that  $\tilde{\beta}$  and  $\tilde{\iota}$  denote the discount factors for the young and the old. Subscript these variables by either 1 or 2 to connote regular and impatient agents respectively. Note that these are “pure” discount factors, i.e., net of mortality risk.<sup>20</sup> With these additional assumptions, there are 14 free parameters:  $p, u, \ell, \omega^u, \omega^\ell, \kappa, A, \mu_1, \mu_2, \eta, \tilde{\beta}_1, \tilde{\beta}_2, \tilde{\iota}_1$  and  $\tilde{\iota}_2$ . Note that the population size is a pure scaling parameter and only the relative sizes of the two types matter:  $\mu_2/\mu_1$ . To render the structure more parsimonious, let  $\tilde{\beta}_2 = \tilde{\iota}_1$  and  $\tilde{\iota}_2/\tilde{\beta}_2 = \tilde{\iota}_1/\tilde{\beta}_1$ . Thus, the discount factor for an old impatient person is equal to that of a young patient one. Additionally, the relative difference between the impatient and patient is preserved across age. Thus, there are 11 parameters to choose, see Table 2 for a summary. To discipline the choice of the parameters, 11 data moments are targeted. Table 3 gives an overview of the data moments and shows how well the benchmark model matches them.<sup>21</sup>

The main targets are the overall prevalence rate for HIV/AIDS in society, and the prevalence rates for each gender. Some additional targets are selected to discipline the exercise, such as the fraction of sex that is casual as opposed to long-term, the fraction of the population that is single, and the pattern of marriage by age. This ensures that there is not too much reliance on risky short-term interac-

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<sup>18</sup>At this stage in the development cycle, the model is too complicated to formally 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, theoretically speaking, models with externalities can display multiple equilibria, the simulations recover only a single interior equilibrium; although, some additional boundary equilibria may exist. Clearly, only interior equilibria have a chance of matching the data. This and some other algorithmic issues require monitoring when running the computer program and occasionally some intervention is required. This makes an automated estimation process difficult. Therefore, a more informal approach is taken to construct the benchmark simulation.

<sup>19</sup>The rationale for these choices is as follows. First, a small group of risky people is needed to generate any HIV in equilibrium. Second, assuming young people to be particularly impatient leads to more risky behavior among them. The importance of small risky groups (e.g. truck drivers and prostitutes) and differential risky behavior by age is discussed in the literature (e.g. Stonebumer et al. (1996), Kremer (1996), Nzyuko et al. (1997) and Oster (2012)).

<sup>20</sup>That is,  $\beta = \tilde{\beta}(1 - \delta)$  and  $\iota = \tilde{\iota}(1 - \delta)$ .

<sup>21</sup>All data sources for the tables and figures are discussed in Appendix B.

Table 2: CALIBRATED PARAMETERS

<i>Interpretation</i>	<i>Parameter value</i>
Flow utility unprotected sex	$u = 7.5$
Flow utility protected sex	$p = 2.3$
Flow utility long-term sex	$l = -5$
Discount factors	$\tilde{\beta}_1 = 0.995, \tilde{\tau}_1 = 0.8955$
Value of life with Aids	$A = 5$
Prob. of switch to high discount factor	$\eta = 0.06$
relative group size	$\mu_1/\mu_2 = 10$
Search cost parameters	$\omega_s = 0.5, \omega_l = 30, \kappa = 0.2$

tions. Many people are married, and get married quickly. Moreover, females get married faster. Additionally, the fraction of condom users in casual encounters and the number of such encounters are considered. This is important because the model emphasizes the choice of whether to engage in risky activity. Finally, the fraction of people that die of natural causes, as opposed to HIV/AIDS, is included for overall consistency.

The upshot of the analysis is that the benchmark simulation matches these key features of the Malawian HIV/AIDS epidemic pretty well. To begin with, as can be seen from Table 3, the HIV/AIDS prevalence rate predicted by the model is 11.8%, matching the data (11.5%) well. Moreover, the experiment captures the gender difference in HIV/AIDS infection rates, with females experiencing a rate that is 3 percentage points higher than that for males (13% versus 10%).

In addition to exactly matching moments on HIV/AIDS prevalence, the benchmark model also matches moments of other aspects of sexual activity quite well. In the model casual or short-term sex is a small fraction of all sexual encounters: 24%, relatively 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 33% use a condom. This is less than 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 et al. (2003)], these two numbers may be closer in reality than first meets the eye. The next row in Table 3 reports the fraction of singles who had casual sex in the last year. These statistics are different from the fraction of

Table 3: TARGETED MOMENTS

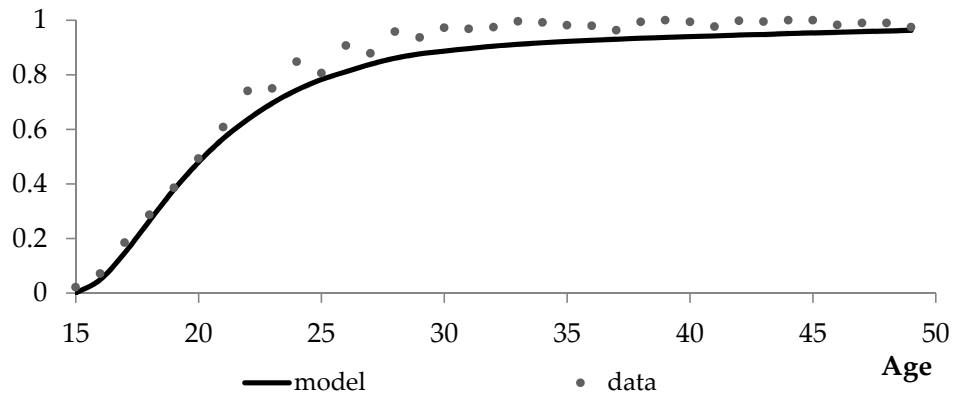
<i>Observation</i>	<i>Data</i>	<i>Model</i>
HIV/AIDS rate, %	11.8	11.5
–Males	10	10
–Females	13	13
Fraction of all sex that is casual, %	18	24
Condom use for casual sex, %	39	33
% (of) Singles that had casual sex in past year	37	54
% Singles	33	38
% Married by age 22		
–Males	58	60
–Females	90	67
% Married by age 50		
–Males	100	97
–Females	100	96
% of deaths related to HIV	29	23

all sexual activity that is casual because (all) married people have sex while 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 HIV/AIDS is comparable across model and data (23 versus 29%).

What probably is not misreported is people’s marital status. Table 3 lists the fraction of singles in the entire population. The model predicts this fraction to be 38%, close to the 33% observed in the data. Moreover, it captures some of the gender differences in the timing of marriage. Women marry much earlier than men—in the data, 90% of women are married by age 22, whereas only 58% of men are married by this age. The corresponding numbers in the model are 67% and 60%. The model generates the earlier marriage of women (relative to men) via their higher infection risk. This makes the safety of marriage more attractive for women vis à vis men. In reality HIV risk is only one reason why women get married earlier than men. Additional considerations such as the risk of pregnancy make casual encounters more risky for women, which might explain the larger wedge between the genders in the data compared to the model. As is only reasonable, men eventually “catch up,” and by age 50 almost everyone is married,



Figure 1: Fraction ever Married – Model vs. Data



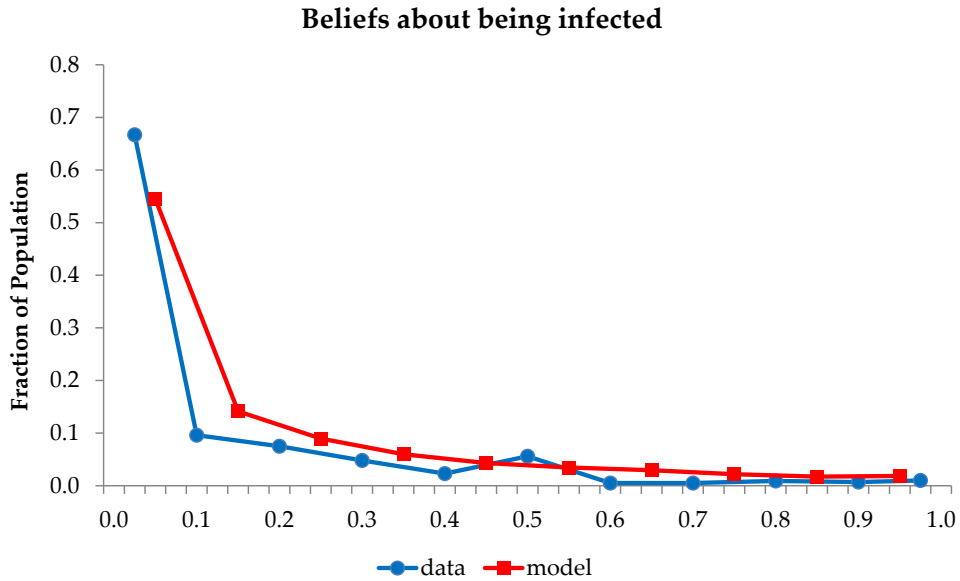
both in the model and the data. See Figure 1 for a comparison of the fraction of the population that has ever married in the model vs. the data.

### 4.3 Non-Targeted Observations

The benchmark model generates some other predictions that are not targeted when picking values for parameters. Recall that agents in the model have rational beliefs about their HIV/AIDS status. Figure 2 plots the belief distribution for symptom-free people in the model and compares it to that 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 (or healthy)—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. After this drop, beliefs are flat throughout the rest of the distribution—both in the model and the data. Moreover, in the data the prevalence rates implied by the reported beliefs roughly match the actual prevalence rates, which is supportive of the modeling assumption that agents are rational.

Next, several life-cycle implications of the model are compared to the data. The model tracks these data patterns surprisingly well despite the limited degree of

Figure 3: Distribution of Beliefs: Model vs. Data

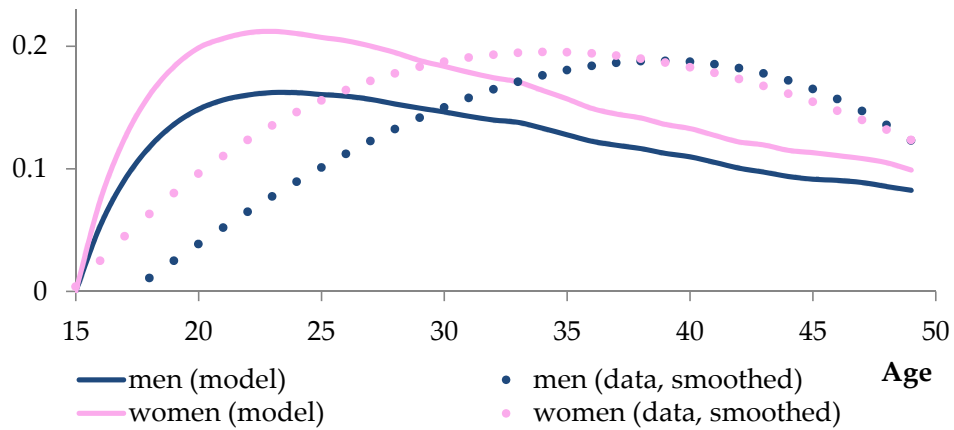


heterogeneity and despite the limited state variables that describe the agent’s life-cycle. This finding adds additional confidence for the policy experiments.

Figure 3 plots HIV/AIDS prevalence by age.<sup>22</sup> Both the model and data agree on a hump-shaped infection pattern, despite the fact that agents in the model become sexually active earlier than is observed in the data, which shifts the model’s life-cycle predictions on HIV/AIDS infections to the left. The hump-shaped pattern is explained by two opposing forces. 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 older people is infected with HIV/AIDS. However, people who are infected early in life will die before they make it to old age. Put differently, people who have made 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 effect explains the eventual drop in HIV/AIDS prevalence seen at older ages. Figure 3 also illustrates the differentiated patterns of infection between the sexes. The figure shows that women get infected earlier than men, both in the model and the data.

<sup>22</sup>The data is fitted with a third-order polynomial. See Figures 7 and 8 in Appendix A for a comparison of the raw data and the fitted line. The somewhat choppy raw data is due to the small sample sizes.

Figure 3: HIV Rate – Men vs. Women, Model vs. Data



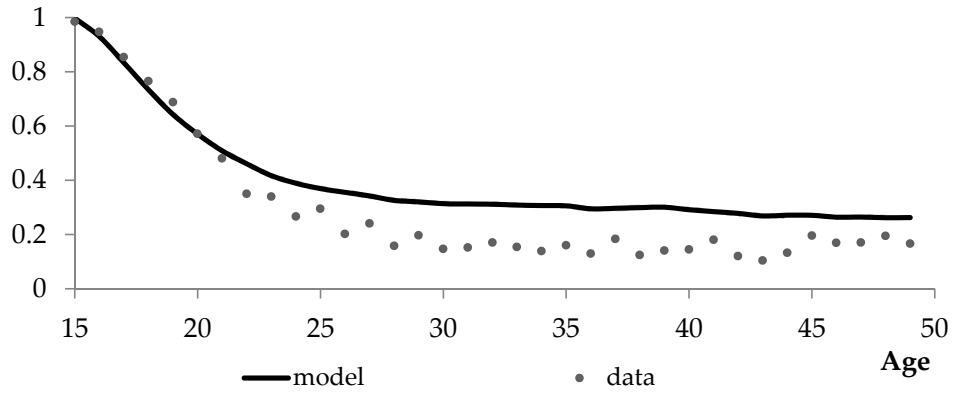
The model also does a very nice job in matching the decline in risky activity over the life cycle. Older people are less likely to be single, see Figure 4a. As people age, they are thus less likely to engage in casual sex; this is reported in Figure 4b. Note that a related but different statistic is the fraction of *singles* that have sex in a given period, since some of them choose to be abstinent. Figure 4c compares the data to the model counterpart. The data exhibits a declining fraction of singles that engage in sex over the life cycle and the model also generates a similar pattern. The reason is that people become more patient as they age and, again, the survivors are more likely to be the individuals who prefer safer lifestyles.

An additional prediction of the model relates to death causes, since agents may die due to HIV/AIDS or due to other natural causes. Figure 5 compares the model prediction over the life cycle with its data counterpart. Both the model and the data exhibit a hump-shaped pattern of HIV/AIDS caused deaths; this is consistent with the hump-shaped pattern of infection rates.

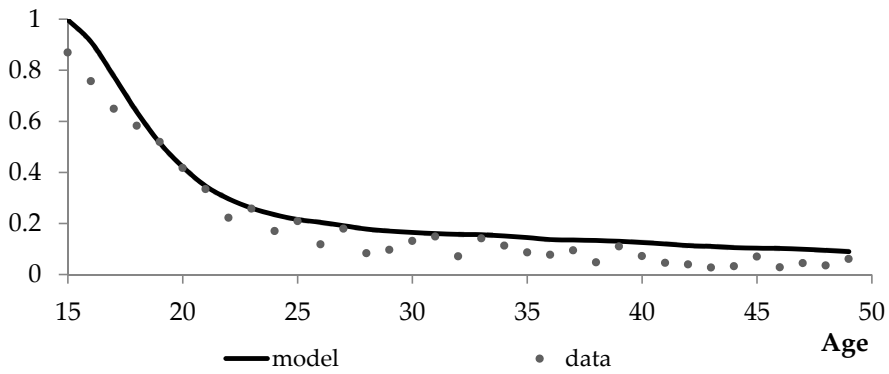
Finally, protected sex in the benchmark simulation is substantially cheaper than unprotected sex. The premium for unprotected casual sex is 67%. Note that such a premium has in fact been documented in the literature. Gertler, Shah, and Bertozzi (2005) use data from commercial sex workers in Mexico to document a 23% premium for unprotected sex. The premium increases to 46% when the sex worker is considered to be very attractive.

Figure 4: Sexual Behavior by Age – Model vs. Data

(a) Fraction of Singles



(b) Casual Sex



(c) Non-abstinent Singles

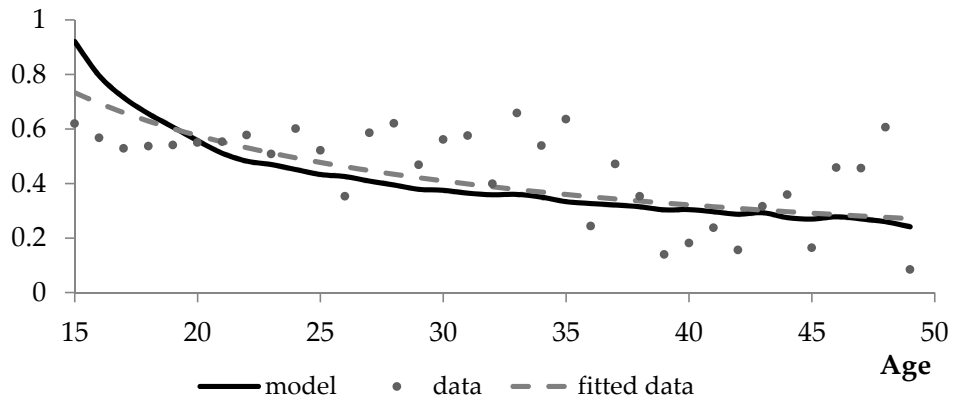
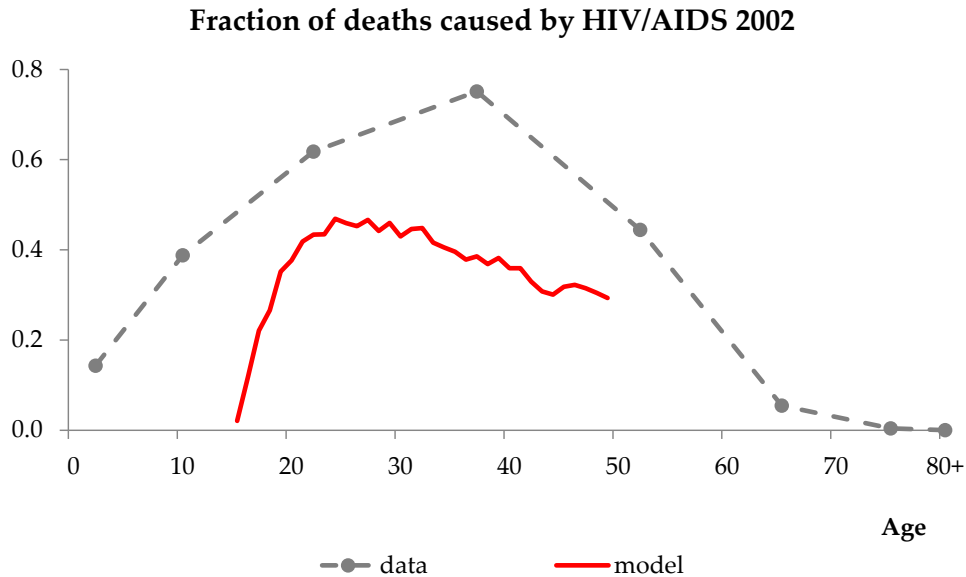


Figure 5: Deaths by HIV/AIDS by Age – Model vs. Data



## 5 Policy Experiments

The model is now ready to explore the effectiveness of various policies intended to curb the spread of HIV/AIDS. Equate effectiveness with the reduction of the prevalence rate, as this is the stated goal of many governments and non-profit organizations. It should be mentioned, however, that a decrease in HIV/AIDS does not necessarily imply an increase in welfare. On the one hand, if people have less sex, an activity they enjoy, their welfare might decrease. On the other hand, the model features an externality as people do not internalize the effect of their own risky behavior on the health of future partners. Moreover, men and women might be differentially affected due to a change in prices.

There are two broad classes of policies surrounding HIV: treatment and prevention. Overall, prevention is thought to be more cost-effective than treatment—see Canning (2006). Thus, the focus of this paper is on prevention. Specifically, we analyze several medical policies (such as male circumcision and treatment of other STDs) as well as policies aimed at behavioral change (e.g. promoting condoms or marriage). Several other prevention policies are beyond the scope of this paper. In particular, no attempts are made to account for information campaigns

and testing. The former requires the introduction of some systematic biases in beliefs, which can then be reduced through the dissemination of information about HIV/AIDS. The second brings in some deeper issues regarding human nature.<sup>23</sup>

In addition to studying the effectiveness of the various policies in the full model, two alternative versions of the model are simulated: (i) small scale field experiments and (ii) epidemiological experiments. To be concrete, the field experiment assumes that only a small fraction of the population is treated and changes their behavior, but that this fraction interacts in equilibrium with everyone else at pre-existing equilibrium prices and infection rates. The epidemiological experiment assumes that people make no behavioral adjustments and therefore use the policy functions from the benchmark calibration but the infection probabilities and assessments of transmission risks are governed by the new transmission probabilities. Comparing the effectiveness of policies across the three versions of the model demonstrates the importance of both elements—behavioral adjustment and equilibrium interaction. It also gives insights into the extent to which actual field experiments and epidemiological studies might (or might not) generate reliable policy advice.

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 in its infancy. The goal of obtaining hard numbers that can be used for policy analysis is still some way down the road. However, the model does provide a useful tool for elaborate thought experiments to discover areas that might need further investigation. The simulations do illustrate potential pitfalls in efforts to limit the disease. Specifically, as will be illustrated with an example on condom policy, moderate policy interventions have the potential to backfire due to the shifts in sexual behavior that

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<sup>23</sup>Currently, it is assumed that in casual relationships people care only about their own risk of becoming infected (and they are committed to faithfulness in marriage). This assumption might be justifiable when the belief about being infected is moderate and agents can argue to themselves and others that they did not know they were infected. Under testing, infected people are told with (near) certainty that they are infected. The consequences for their partners are clearly brought to their attention. Extrapolating the current behavioral assumption to such an extreme setting would imply reckless behavior after being tested positive. Concern for others might yield very different implications. Thus, incorporating testing would bring issues of altruism to the fore. It is unclear what the best way of dealing with this is.

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.

## 5.1 Medical Policies

An analysis will now be undertaken of several medical policies that have been recommended recently.

### 5.1.1 Policies that Reduce Transmission for Both Sexes

Several policies involve the reduction in transmission risk. For example, results of a new vaccine trial were made public very recently showing a 30% efficacy.<sup>24</sup> Similarly, antiretroviral drugs and certain gels are thought to reduce the transmission risk. Another policy that has recently been advocated is the treatment of other sexually transmitted diseases (STDs). The idea is that the presence of other STDs makes a person more susceptible to contracting HIV. 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. Oster (2005) compares data from African countries and from the US and Western Europe and reaches the conclusion that treating other STDs would be an effective policy. Using a “diff-in-diff” approach, she argues that the most likely cause for different HIV rates are STDs—rather than behavioral differences. This conclusion is somewhat problematic. If current sexual behavior in African countries (where the HIV rate and related risks are high) is similar to European countries (where the rate and associated risks are much smaller), then, this may 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 that crowd

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<sup>24</sup>Promising results of a new vaccine that would reduce transmission by 30% were recently reported in the media, e.g. in a *Wall Street Journal* article on September 25, 2009: “Vaccine Shows Promise in Preventing HIV Infections.” However, additional data analysis found that the results are statistically insignificant.

Table 4: TREATING OTHER STDs

	<i>Benchmark</i>	<i>Lower Risk</i>	<i>Epidem.</i>	<i>Small Field</i>
$\gamma_u^m$	0.940	0.948	0.948	0.948
$\gamma_u^f$	0.895	0.902	0.902	0.902
HIV / AIDS rate, %	11.5	9.9	9.4	11.1
–Males	10.2	8.7	8.3	9.8
–Females	12.8	11.1	10.6	12.4
Fraction of sex that is casual, %	23.9	24.2	—	24.3
% (of) Casual sex with condom	33.0	29.7	—	30.9
% Singles who have casual sex	54.0	57.6	—	56.0
% Men who are single	42.8	42.1	—	42.6
% Women who are single	38.7	37.9	—	38.6

out the gains from the reduced risk. Simulating people’s behavior in response to changed circumstances is one way of taking such behavioral responses into account and trying to assess the overall impact of such a policy.

Table 4 shows the simulation results for this policy in our quantitative model. As the transmission risk for both men and women declines by about 13%, the HIV incidence decreases by 1.5 percentage points from 11.5 to 9.9%. Note that this decrease in HIV prevalence masks the find that, when faced with better odds when having sex, agents engage in riskier behavior. The fraction of sex that is casual increases even though there are less singles around (since unprotected sex is safer and sex within marriage is unprotected, people have an extra incentive to marry). The reason is that the fraction of non-abstinent singles increases from 54% to 57.6%. Moreover, out of the singles having sex, condom usage falls from 33% to 29.7%. The upshot of this experiment is that agents can dramatically change their behavior in response to the policy and that these behavioral changes can have non-trivial effects, which can be seen as follows.

Compare the results with the epidemiological version of the experiment. In the epidemiological experiment, the decline in HIV prevalence is much larger to 9.4%, a 0.5% difference compared to the benchmark. The reason for this difference is exactly the lack of behavioral changes described above.

The field experiment goes in the opposite direction: it predicts a much smaller de-



crease in HIV incidence compared to the benchmark (11.1% in the former versus 9.9% in the latter). The reason is that, in the field experiment, the reduced number of infections does not lead to an overall decrease in the *population* prevalence rate. Therefore, it does not feed back into lower infection rates for the treated population, something that is naturally part of the full model. It is interesting to note that eight of the nine studies of STD treatment for HIV prevention surveyed by Padian et al. (2010) delivered flat results. Even though the authors discuss some potential explanations for these weak results, the simulations presented here highlight a novel reason, namely the missing general equilibrium effects in randomized field experiments.

Taking stock, treating other STDs seems to decrease the overall HIV prevalence rate even though people engage in riskier behavior in response to the lower likelihood of getting infected. This shift in behavior makes the use of a choice-theoretic model like the one proposed here essential. The differences arising from these behavioral responses seem to be quantitatively important. Moreover, conducting randomized field experiments on a small scale might not be enough to assess the effects of such a policy. General equilibrium effects are present and they might be powerful.

### 5.1.2 Male Circumcision

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.<sup>25</sup> The most widely cited evidence comes probably from Gray et al. (2007) who, based on a randomized field experiment in Uganda, 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

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<sup>25</sup>See <http://www.unaids.org/en/PolicyAndPractice/Prevention/MaleCircumcision/> accessed on October 7, 2009.

Table 5: MALE CIRCUMCISION

	<i>Benchmark</i>	<i>Lower Risk</i>	<i>Epidem.</i>	<i>Small Field</i>
$\gamma_u^m$	0.940	0.948	0.948	0.948
HIV/AIDS rate, %	11.5	10.0	10.7	11.2
–Males	10.2	8.7	9.3	9.8
–Females	12.8	11.5	12.2	12.8
Fraction of sex that is casual, %	23.9	23.4	—	24.2
% (of) Casual sex with condom	33.0	31.1	—	31.1
% Singles who have casual sex	54.0	55.3	—	55.8
% Men who are single	42.8	42.2	—	42.6
% Women who are single	38.7	37.6	—	38.7
Transfers				
–Protected	107	115	—	—
–Unprotected	189	237	—	—
–Long term	94	115	—	—

effects. If all men were circumcised, perhaps the entire village would feel safer and engage in riskier behavior, which would crowd out some of the gains.

Table 5 reports the results in the model when men (and only men) are faced with lower odds of infection. The first thing to note is that, even though sex is safer only for men, the prevalence rates for *both* sexes decrease. The reason for this is that, with the lower likelihood of getting infected, men are on average healthier and thus the spread of the disease slows down.

Two other things are worth noting in Table 5. First, both the small field experiment and the epidemiological study suggest that the policy is less effective than what the full equilibrium says; and that is even more true for women.<sup>26</sup> Second, agents' behavior regarding single life versus marriage, condom usage, casual sex, etc. do not seem to change much. Actually, both of these observations can be explained once one looks at the changes in transfers, also reported in the table. Given that the likelihood of infection does not change for women, they demand higher transfers from men; that is particular true, of course, where sex is unprotected: short-term unprotected and long-term relationships. Given these higher

<sup>26</sup>In the small field experiment, given that there is no change in the odds of infection for women and the absence of general equilibrium effects by design, the HIV prevalence rate for females will not change by construction.

transfers, men do not change their behavior as much as they would in say, the small field experiments where general equilibrium effects of this type do not play any role.

## 5.2 Policies aimed at Behavioral and Societal Change

Other policies have been aimed directly at social and behavioral 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 behavioral 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 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.<sup>27</sup> In the experiments conducted here the effects of “promoting marriage” are investigated for various interpretations of what this policy might mean. Promoting condoms is modeled as an increase in the utility from using them.

### 5.2.1 Condoms

Suppose one could design more pleasurable condoms (or perhaps raise the psychic pleasure of sex with a condom through a publicity campaign). Would this be desirable? The results are reported in Table 6. It turns out that the HIV rate displays a hump-shaped pattern when increasing pleasure from condoms,  $p$ . The reason that increasing the utility from protected sex does not always lead to a decrease in the prevalence rate is that single life becomes more attractive. So, even though condom usage increases, there are more singles in total and they engage in both protected and unprotected sex. From the table, see that the fraction of single men and women increases substantially as condoms become more attractive. Moreover, the fraction of singles that engage in short-term sex skyrockets.

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<sup>27</sup>This point is also made in Dupas (2011).

Table 6: BETTER CONDOMS

$p$	<i>Benchmark</i>	<i>Better</i>	<i>Better Still</i>
HIV/AIDS rate, %	11.5	13.3	12.7
Fraction of sex that is casual, %	23.9	39.9	53.9
% (of) Casual sex with condom	33.0	66.2	73.6
% Singles who have casual sex	54.0	92.2	95.6
% Men who are single	42.8	52.5	65.1
% Women who are single	38.7	48.2	61.6

Even though more people use condoms, there is a lot more activity in the casual sex market. These two forces push the prevalence rate in opposite directions and this tension can be seen in Table 6. As condoms first get better, the HIV/AIDS prevalence rate initially goes up. As more and more people start to use condoms, however, the lower likelihood of getting infected becomes more important and the prevalence rate starts to go back down. This experiment highlights the potential of some policies to backfire and actually *increase* the overall prevalence rate. Note also that these effects can be quantitatively quite important: the prevalence rate goes up by almost two percentage points before it starts to come down.

### 5.2.2 Increased Utility from Marriage

Table 7 reports the results of an experiment in which marriage is more attractive. That is, the utility of being married ( $l + u$ ) increases. The first thing to note from the table is that, as expected, the fraction of single people plummets. Consequently, the fraction of sex that is casual also exhibits a marked decrease; there is thus less risky activity taking place in the economy. This adds up to a lower overall HIV prevalence rate.

### 5.2.3 Marriage: Entry vs. Exit

Making marriage more attractive 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,

Table 7: PROMOTING MARRIAGE

	<i>Benchmark</i>	<i>Better</i>	<i>Better Still</i>
$l + u$	2.5	3.5	4.5
HIV/AIDS rate, %	11.5	10.0	7.8
Fraction of sex that is casual, %	23.9	18.0	12.2
% (of) Casual sex with condom	33.0	33.3	33.2
% Singles who have casual sex	54.0	56.3	53.1
% Men who are single	42.8	34.3	26.8
% Women who are single	38.7	27.7	20.7

social events organized by community or religious groups may facilitate searching for a spouse. Likewise, the provision of marriage counselling services may reduce divorce. Similarly, pro-family tax codes could promote marriage and dissuade divorce. The impact effect of both strategies should be an increase in marriage which may have the potential to lower the HIV/AIDS rate. These policies are operationalized by decreasing the odds of divorce,  $\xi$ , and by lowering the search cost of finding a marriage partner,  $\omega_{LT}$ . The corresponding experiments are depicted in Table 8.

As divorce risk decreases, long-term relationships become longer and people become less promiscuous. The fraction of casual sex thus goes down. This happens of course along with a marked decrease in the number of singles. All this contributes to a lower prevalence rate in equilibrium. However, the decrease of the prevalence rate is limited because singles have a lot more sex now, which counteracts the benefits from more marriage.

Interestingly, when the search costs in the long-term market are lower (last two columns), the HIV prevalence rate actually increases. The reason is a worsening of the pool of people searching in the long-term market. As the search costs in this market decrease, riskier types find it more profitable to look for a long-term partner. This worsens the marriage pool and drives the prevalence rate up.

Table 8: MARRIAGE - SEARCH COSTS AND DIVORCE RISK

	Benchmark	Divorce risk ↓		Search cost ↓	
Divorce risk, $\xi$	0.03	0.02	0.015	0.03	0.03
Marital search cost, $\omega_{LT}$	30	30	30	28	20
HIV/AIDS rate, %	11.5	10.6	10.0	11.6	12.0
Fraction of sex that is casual, %	23.9	20.7	19.2	23.5	22.2
% Singles who have casual sex	54.0	62.4	67.6	55.2	60.8
% Men who are single	42.8	34.3	30.3	41.7	37.2
% Women who are single	38.7	31.7	28.2	37.9	34.8

### 5.2.4 Other Experiments

The framework lends itself to analyzing a host of policies. Space constraints do not permit a detailed accounting of all the experiments entertained. Therefore, in this section we briefly summarize a few additional experiments.

#### *Higher Incomes*

Developing countries receive substantial financial aid from other countries. Could such financial aid play any direct role in HIV/AIDS prevention? Increasing income,  $y$ , lowers the HIV/AIDS rate substantially in the model. The reason is that richer people value life more and hence behave in a more risk-averse fashion. There are fewer singles and in addition singles have less sex. If income is increased for women only, then the effects are even larger. Women now have more to lose and hence demand a higher price for sex. When male income is held fixed, men reduce their risky behavior in face of a higher price for sex. This shows up very clearly in the experiments, where the price of all forms of sex goes up substantially when income for both genders is increased, but only very little when only women receive the subsidy. Thus, interestingly, the cheaper policy (which involves payment to half of the population only) is the more effective one.

#### *Discouraging Casual Sex*

One might imagine a policy that makes meeting people of the opposite sex more difficult, for example by taxing popular meeting venues or the inputs they use such as alcohol. This is operationalized here by increasing  $\omega_s$ . This policy leads to a decrease in the prevalence rates for both genders. This lower prevalence rate

comes from two reasons. First, a lower fraction of singles have casual sex, since it is more costly to find a mate in these short-term markets. Second there are less singles in equilibrium. The reason for the second effect is that, since agents know it is more costly to search in the short-term markets, they search more heavily in the long-term market.

## 6 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 of these choices crucially affect the spread of HIV/AIDS in society.

The theoretical model developed is simulated to see whether or not it can capture some of the salient features of the Malawian HIV/AIDS epidemic. It can. For example, the framework can match the fraction of sex that is casual, the number of encounters that use a condom, and the HIV/AIDS prevalence rates for men and women. Furthermore, it can mimic the decline in casual sex by age and the hump-shaped pattern of HIV/AIDS prevalence over the lifecycle. 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, some policies (such as promoting condoms) may backfire and actually increase HIV. The aim of the analysis is to provide a toolbox that allows the study of various interventions, identifies where behavioral change might be important, and thereby identifies areas where further and deeper exploration might be most warranted.

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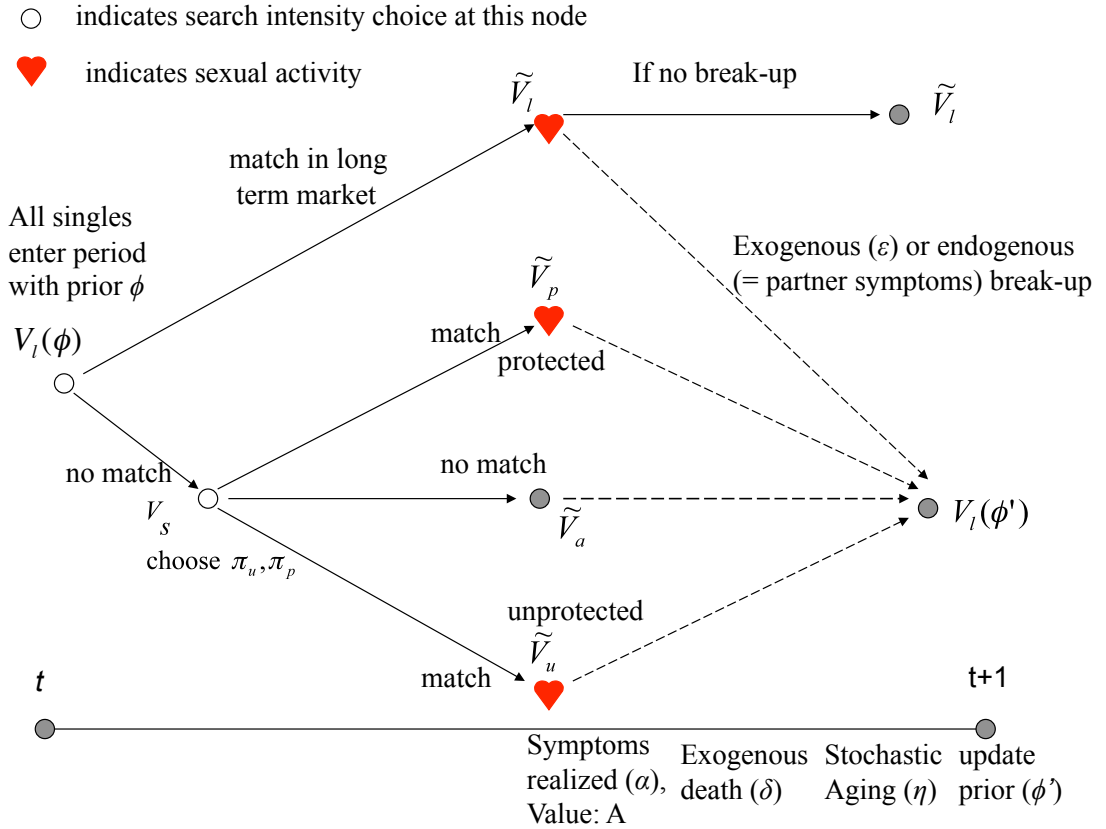
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# A Appendix—Additional Figures

Figure 6: Timing of Events



# B Appendix—Data

Most of the empirical moments are based on information from the individual interviews of the Malawi Demographic and Health Survey (MDHS) in 2004, carried out by the Malawi National Statistical Office. In this survey 11,698 women aged 15 to 49 and 3,261 men aged 15 to 54 were interviewed. Means are calculated using sample weights. For several figures means are calculated by age.

Since men are underrepresented in the survey, separate means are calculated by sex, and then averaged. Whenever sources other than the MDHS are used, it will be indicated. More details on each figure follow. For the interested reader the details also include the variable names corresponding to each question.

- Figure 1: Fraction ever Married - Model vs. Data  
The fraction of ever-married people is derived by dividing the number of people who are currently married (including cohabitation) or have been formerly married by all people. The corresponding question is “Have you ever been married or lived with a man/woman?” (MDHS 2004: v/mv502).
- Figure 2: Distribution of Beliefs - Model vs. Data  
Data is based on Delavande and Kohler (2009), Table 3.
- Figure 3: HIV Rate - Men vs. Women, Model vs. Data  
In order to calculate the HIV rates by age (MDHS 2004: v012/mv012) and gender, individual information from the MDHS 2004 is matched with the HIV test results (MDHS 2004: hiv03) for those people who agreed on doing the test along with the interview (since not everyone agreed, the sample size is smaller here: 2404 men and 2864 women). The resulting HIV rates are smoothed using a third order polynomial. The raw data are shown in Figures 7 and 8.
- Figure 4a: Singles by Age - Model vs. Data  
Those women and men who reported that they have never been married or are widowed, divorced (living or not living together) are defined as singles (MDHS 2004: v/mv501).
- Figure 4b: Casual Sex by Age - Model vs. Data  
To identify the fraction of sex that occurs in casual relationships, all men and women are considered who had sex in the last year (MDHS 2004: v/mv529). Those people are asked with whom they had sex (MDHS 2004: v/mv767a). They are also asked whether they had sex with a second (MDHS 2004: v/mv761b, v/mv767b) and third (MDHS 2004: v/mv761c, v/mv767c) partner. If one of the sex partners was not the spouse or cohabiting partner, then the sex in the last year is categorized as casual sex. Men in addition



are asked whether they have ever paid for sex (MDHS 2004: mv792). Those men who have paid for sex in the last year are also defined as being active in the short-term market.

- Figure 4c: Non-abstinent Singles by Age - Model vs. Data  
People were asked when was the last time they had sex. If they did not have sex in the last quarter they are classified as abstinent (MDHS 2004: v/mv501 and v/mv529). In this figure we only use data from singles (MDHS 2004: v/mv501).
- Figure 5: Deaths by HIV/AIDS by Age - Model vs. Data  
The data on deaths caused by HIV/AIDS are taken from Bowie (2006), pages 31-42. He reports the fraction of HIV/AIDS related deaths by age groups, based on the WHO Global Burden of Disease Malawi from 2002.
- Table 1: Parameters Chosen Outside the Model  
All sources are described in the text.
- Table 3: Targeted Moments  
The data on the prevalence of HIV/AIDS in Malawi derive from the Demographic and Health Surveys' (MDHS) Final Survey for Malawi in 2004. See MDHS (2004, Table 12.3). The fraction of sex that is casual 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 MDHS (2004, Table 11.9). Condom usage for short term sex also derives from MDHS (2004, Table 11.9)—and is averaged across men and women. The fraction of singles who have casual sex is reported in MDHS (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 MDHS (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 by a certain age is the same as in Figure 1. The World Health Organisation (2008) reports that 29% of all deaths in Malawi in 2004 were due to HIV/AIDS.

Figure 7: Male HIV Rate – Model vs. Data

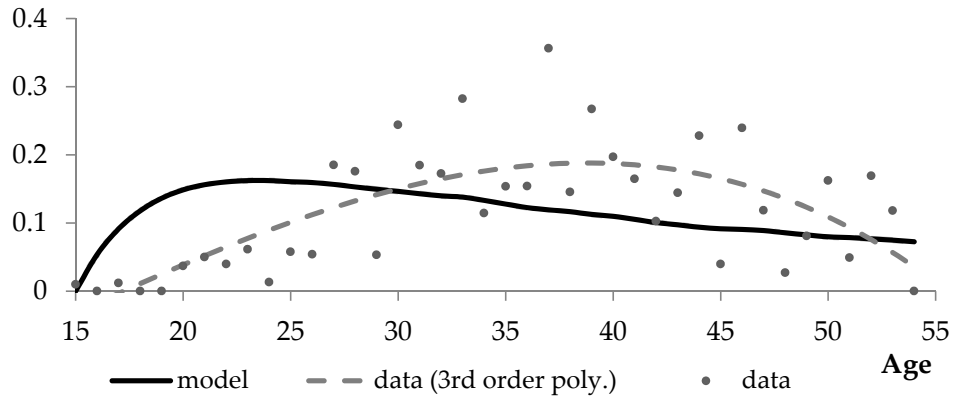
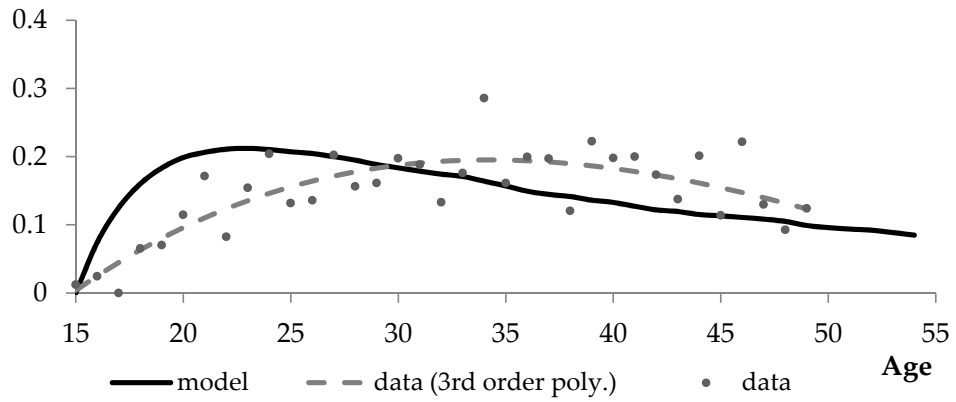


Figure 8: Female HIV Rate – Model vs. Data



## C Appendix—Theory

### C.1 Updating in a Long-term Relationship

Whether a breakup occurs for exogenous reasons or because the partner developed symptoms contains information about one's own HIV status. Below we develop two separate updating formulae, one for each case. Note that when the person himself develops symptoms, no updating is necessary since in this case the person knows he is HIV positive.

First, consider 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 should update his prior according to the rule

$$\begin{aligned}\Phi_l^h(\phi, n) &= \frac{\text{Pr}(\text{not being infected and partner showing no symptoms at end of period } n|\phi)}{\text{Pr}(\text{no symptoms in either person at end of period } n|\phi)} \\ &= \frac{\phi\bar{\phi}_l + \phi(1 - \bar{\phi}_l)\gamma_u^n(1 - \alpha)^n}{\Delta^h},\end{aligned}\tag{16}$$

with

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

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  $\gamma_u^n$ ; (iii) the partner never shows symptoms, which occurs 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 do not 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  $\gamma_u^n$  in the brackets) or because 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 HIV/AIDS symptoms at the end of period  $n$  is similar. Denote this case with superscript  $A$ . 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

$$\begin{aligned} \Phi_l^A(n, \phi) &= \frac{\text{Pr}(\text{not being infected and partner showing symptoms at end of period } n | \phi)}{\text{Pr}(\text{no symptoms in oneself and symptoms in partner at end of period } n | \phi)} \\ &= \frac{\phi(1 - \bar{\phi}_l)\gamma_u^n(1 - \alpha)^{n-1}\alpha}{\Delta^A}. \end{aligned} \quad (17)$$

with

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

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 (16), 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 the 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.

## C.2 Symptom Probabilities

### C.2.1 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  $(1 - \phi)(1 - \bar{\phi}_l)(1 - \alpha)^{2n}$ . 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 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  $(1 - \phi)\bar{\phi}_l(1 - \alpha)^n\gamma_u^n$  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

$$\begin{aligned} \Pr[\text{no symptoms in either person at end of period } n|\phi] &= \phi\bar{\phi}_l + (1 - \phi)(1 - \bar{\phi}_l)(1 - \alpha)^{2n} \\ &+ [(1 - \phi)\bar{\phi}_l + \phi(1 - \bar{\phi}_l)](1 - \alpha)^n [(1 - \gamma_u) \sum_{j=0}^{n-1} \gamma_u^j (1 - \alpha)^{n-j} + \gamma_u^n]. \end{aligned} \quad (18)$$

### C.2.2 Pr[symptoms just in partner at end of period $n|\phi$ ]

What are the chances that just the 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 this 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

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

### C.2.3 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  $n$ th 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  $(1 - \phi)(1 - \bar{\phi}_l)(1 - \alpha)^{2n-2}\alpha$ . 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  $n$ . 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,

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

### C.3 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  $\phi$  and low discount factor,  $\tilde{V}_l^\iota(\phi, x)$ , needs to be characterized. Recall that the discount factor may switch from the low value,  $\iota$ , to the high one,  $\beta$ , with probability  $\eta$ . 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  $\iota$ —note that he will use the discount factor  $\iota$  for the next period with certainty. If this event transpires he will discount utility  $n$  periods ahead by  $\iota^{n-1}$ . Alternatively, he may draw the discount factor  $\beta$  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  $\iota^{k-1}\beta^{n-k}$ . Define the two new discount factors  $\underline{\beta}^\iota(n)$  and  $\bar{\beta}^\iota(n)$  by

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

and

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

with

$$\bar{\beta}^l(1) = 0.$$

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

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



Except for the possibility of a switch in the discount factor, the formula resembles (7). In fact, (21) collapses to (7) when  $\eta = 1$  implying  $\underline{\beta}(n) = 0$ —here  $\bar{\beta}^t(n)$  should be set to  $\beta^n$ . 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  $\eta$  in period  $n + 1$ . Lines 6 and 7 assume an exogenous breakup occurs and that the discount factor has switched sometime before  $n + 1$ . The rest of the lines should now be self evident.

## C.4 Stationary Distributions

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

### C.4.1 Singles distributions, $d = \iota$

Consider the case where singles have the low discount factor,  $\iota$ . 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  $\phi$  is specified by

$$\begin{aligned}
\mathcal{S}^t(\phi', x) &= \mu(1 - \eta)J(\phi' - 1) & (22) \\
&+ (1 - \delta)(1 - \eta) \sum_{\phi} [1 - \Pi_l^t(\phi, x)] \mathcal{S}^t(\phi, x) \\
&\times \{ \{1 - \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_p)(1 - \gamma_p)]\} J(\phi' - \Phi_p(\phi)) \Pi_p^t(\phi, x) \\
&+ \{1 - \alpha[(1 - \phi) + \phi(1 - \bar{\phi}_u)(1 - \gamma_u)]\} J(\phi' - \Phi_u(\phi)) \Pi_u^t(\phi, x) \\
&+ [1 - \alpha(1 - \phi)] J(\phi' - \Phi_a(\phi)) \Pi_a^t(\phi, x) \} \\
&+ \mathcal{L}^t(\phi', x).
\end{aligned}$$

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 draw a low discount factor in

the first period. 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 the second source of singles and it is given by 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 others 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  $\phi$  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,  $\iota$ , and a prior  $\phi'$ , conditional on starting with a prior  $\phi$ , will consequently read

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

where the probabilities for symptoms are specified by (18) to (20) above. From

this it is easy to calculate that the unconditional exit distribution,  $\mathcal{L}^\iota(\phi', x)$ , is given by

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

#### C.4.2 Singles distributions, $d = \beta$

Next, the situation where singles have the high discount factor,  $\beta$ , will be presented. A 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  $\phi$  is given by

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

The first six lines are the direct analogue to equation (22), only with a high discount factor. The exit distribution from married life,  $\mathcal{L}^\beta(\phi', x)$ , is defined below. It includes married individuals whose own discount factor moved up from  $\iota$  to  $\beta$  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.

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

will exit married life with the prior  $\phi'$  is given by

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

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

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

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

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

Last, equations (22) to (28) fully describe the operator  $\mathbf{T}$  in (11).

## D Appendix—Algorithm

### D.1 Computing the Value Functions and Distribution Functions

The algorithm enters each iteration with a guess for the set of values functions,  $\tilde{V}_{g,r}^d$  and  $V_{g,r}^d$ , and stationary distributions,  $\mathcal{L}_g^d$  and  $\mathcal{S}_g^d$ , 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) for each market. On each iteration the value functions are updated using equations (1), (2), (3), (5), (6), (7), (8), 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,  $\phi$ , over his nonprevalence rate. The above functions are computed at each grid point. Even though by construction  $\phi$  is a grid point there is no guarantee that  $\phi'$  will be, given the form of the updating functions (9), (10), (16) and (17). This is resolved using an interpolation scheme (MATLAB's cubic Hermite scheme). For example to compute  $\tilde{V}_a^\beta(\phi', x)$  on the righthand side of (1) for an off-the-grid point  $\phi'$  a weighted average of  $\tilde{V}_a^\beta(\phi'_i, x)$  and  $\tilde{V}_a^\beta(\phi'_{i+1}, x)$  at the two nearest adjacent grid points,  $\phi'_i \leq \phi' \leq \phi'_{i+1}$ , is computed.<sup>28</sup> A similar issue arises when computing the distribution functions. Take the density shown in (22), for example. The updating rules (9) and (10) in general will not map a grid point  $\phi$  into a  $\phi'$  that lies on the grid. Therefore,  $\phi$  is mapped onto the two closest adjacent points,  $\phi'_i$  and  $\phi'_{i+1}$ , such that  $\phi'_i \leq \Phi_r(\phi) \leq \phi'_{i+1}$ , for  $r = a, u, p$ , using a linear weighting scheme.

## D.2 Pseudo Code for Monte Carlo - 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  $\phi, n, p, a, h, w$  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 dis-

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<sup>28</sup>The Hermit scheme preserves monotonicity of  $\tilde{V}_a^\beta$  in  $\phi$ , and therefore the extrapolation can be interpreted as a weighted-average of the nearest grid points. The weights themselves are computed based on the entire set of grid points, not just based on the nearest ones, to preserve certain smoothness properties.

tribution on  $[0, 1]$ . If the first is below  $\eta$ , then start the person with high discount factor and only use decision rules with superscript  $\beta$  throughout his life. Otherwise, in the first period  $t = 1$  of a person's life use decision rules with superscript  $\iota$ . 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  $\bar{t} \in \{1, 2, \dots\}$  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  $\iota$ , for every period  $t > \bar{t}$  use policy rule with the index  $\beta$ . (Note that  $\bar{t}$  might be larger than  $T$ , the maximum number of periods we want to simulate, in which case only subscript  $y$  applies).

2. Start off all people as newly born singles with  $\phi = 1, n = 0, p = 0, w = 0, h = 1$ , 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  $d, \phi, n, p, h$  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  $\delta_2$  the person dies, i.e.  $a_{i,t+1} = 0$ . If it is above  $\delta_2$  then he lives  $a_{i,t+1} = 1$  and  $h_{i,t+1} = 0$ . ( $\delta_2$  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  $t$ th row of the matrix of random variables. Define the probabilities of some of the events discussed in Section C.2.

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

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

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

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

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

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

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

- (a) Marriage persists. If the random variable is less than  $(1 - \varepsilon)(1 - \delta)N_1(n_{i,t})/\Lambda(n_{i,t})$ , where  $\Lambda(n_{i,t}) \equiv [N_1(n_{i,t}) + E_1(n_{i,t}) + E_2(n_{i,t}) + E_3(n_{i,t}) + A_1(n_{i,t}) + A_2(n_{i,t}) + A_3(n_{i,t})]$ , 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  $(1 - \delta)N_1(n_{i,t})/\Lambda(n_{i,t})$  then the 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  $(1 - \delta)N_1(n_{i,t})/\Lambda(n_{i,t})$  and  $(1 - \delta)[N_1(n_{i,t}) + E_1(n_{i,t}) + E_2(n_{i,t}) + E_3(n_{i,t})]/\Lambda(n_{i,t})$  then the marriage 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  $Q \equiv 1 - (1 - \delta)[N_1(n_{i,t}) + E_1(n_{i,t}) + E_2(n_{i,t}) + E_3(n_{i,t})]/\Lambda(n_{i,t})$  the person either gets AIDS symptoms or dies or both. Draw the third random variable in the  $t$ th column. If it is below  $\delta/Q$  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  $t$ th row of the matrix of random variables. If this random variable lies below  $\pi_l$  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 outlined below 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  $\delta$  the person dies and  $a_{i,t+1} = 0$ . If the number is between  $\delta$  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  $\delta$  the person dies and  $a_{i,t+1} = 0$ . If the number is between  $\delta$  and  $\delta + (1 - \delta)\alpha[(1 - \phi_{i,t}) + \phi_{i,t}(1 - \bar{\phi}_s)(1 - \gamma_s)]$  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  $\delta$  the person dies and  $a_{i,t+1} = 0$ . If the number is between  $\delta$  and  $\delta + (1 - \delta)\alpha[(1 -$



$\phi_{i,t}) + \phi_{i,t}(1 - \bar{\phi}_u)(1 - \gamma_u)]$  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  $\delta$  the person dies and  $a_{i,t+1} = 0$ . If the number is between  $\delta$  and  $\delta + (1 - \delta)\alpha[(1 - \phi_{i,t}) + \phi_{i,t}(1 - \bar{\phi}_p)(1 - \gamma_p)]$  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  $\sum_{i,t} h_{i,t} p_{i,t} / \sum_{i,t} h_{i,t}$ . 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 gets newly wed. Then the prevalence in society for those who do not display severe AIDS symptoms yet is  $\sum_{i,t} h_{i,t} \phi_{i,t} / \sum_{i,t} h_{i,t}$ .