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Twelve percent of the Malawian population is HIV infected. Eighteen percent of sexual encounters are casual. A condom is used a third of the time. To analyze the Malawian epidemic, a choice-theoretic general equilibrium search model is constructed. In the developed framework, people select between different sexual practices while knowing the inherent risk. The calibrated model is used to study several policy interventions, namely, ART, circumcision, better condoms, and the treatment of other STDs. The efficacy of public policy depends upon the induced behavioral changes and equilibrium effects. The framework complements the insights from epidemiological studies and small-scale field experiments.

KEYWORDS: ART, circumcision, condoms, disease transmission, epidemiological studies, HIV/AIDS, knowledge about HIV, Malawi, marriage, policy intervention, search, small field experiments, STDs, sex markets.

1. INTRODUCTION

HIV/AIDS IS A MAJOR CAUSE OF DEATH, currently killing about 1 million people worldwide each year. Within Africa, most transmissions occur through heterosexual sex. Furthermore, the majority of the HIV-positive population in African countries is female, compared to less than one third in most developed countries. A natural question is: What can and should be done to prevent the disease? Public policy remedies depend not only on their medical efficacy, but also on the behavior of the population: A treatment that has been medically shown to reduce HIV transmission at the level of an individual might be less effective if the person starts engaging in more risky sexual practices. This behav-

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ioral response is amplified in equilibrium, since fewer infections in the population at large reduces the negative consequences of risky sexual practices further.

The idea that an endogenous response by the population could reduce policy effectiveness has long been recognized in the theoretical disease transmission literature (Philipson and Posner (1993)). In the most extreme case, behavioral adjustment can be so large that a policy backfires, leading to more HIV infections, as in Kremer (1996). Yet, to date, the degree to which behavioral reactions might quantitatively reduce or even negate the effectiveness of interventions has not been assessed. In fact, existing theoretical models are not even designed for a quantitative assessment. Quantitatively accounting for this behavioral channel seems prudent so as to not overstate the likely effectiveness of policies. This paper takes an important step toward filling this gap.

The main vehicle of analysis is a novel computational choice-theoretic equilibrium model of sexual behavior. It is used to address the HIV/AIDS epidemic in Malawi; some background is provided in Section 2. The model, which is set up in Section 3, features several margins of risky sexual behavior with three goals explicitly in mind: (1) to capture aspects of choice that are particularly relevant to HIV transmission; (2) to match sexual behavior against existing data, an aspect that was not possible in previous theoretical work; and (3) to analyze the importance of shifts in sexual behavior for public policy.¹

In the tradition of *homo economicus*, each individual in the model rationally chooses their sexual behavior to maximize expected discounted lifetime utility. Specifically, people choose what kind of sexual relationship they are looking for by searching in one of three different “markets” or “meeting places”—for long-term sex, casual sex with condoms, or casual unprotected sex. This market structure eliminates any problem with differences in interests between partners: they have the same desires when they meet in the same market. Finding a partner generates utility from sexual behavior. Marriage (or a long-term relationship) has the additional benefit of continued interaction without the need to search again. Searching in a market has both a convex effort cost and the cost of possibly contracting HIV. In line with the data, transmission rates differ according to gender, condom usage, antiretroviral therapy (ART) availability, and male circumcision. People know whether or not they themselves are infected with HIV. But, crucially, when agreeing to sex, individuals do not know whether their partner is infected, treated, or circumcised. When entering a meeting place for a relationship, they rationally recognize that some of these markets will be riskier than others.²

Individuals differ across several dimensions in the model. Men and women feature separately. The degree of patience is allowed to vary across individuals, which induces different people to weigh the risk of sexual behavior differently. As people age, they become on average more patient. Men may be circumcised or not. People are endogenously heterogeneous in whether they are healthy or HIV-infected (with or without symptoms). In the presence of ART, infected people will further differ by whether or not they are being treated.

A stationary equilibrium is solved for. In equilibrium, each market is characterized by its endogenous riskiness and by a transfer that one partner makes to the other. These

¹The use of quantitative choice-theoretic models to study HIV transmission appears to be rare. One instance is Magruder (2011), who built a matching model of marital partner search and HIV. This work, however, abstracts from condom use and equilibrium effects, so there is no feedback from individual choices to the prevalence of the disease in society.

²Endogenous sorting can render sex in marriage safer than in casual relationships. A companion paper addresses policies that promote marriage and dissuade divorce; see Greenwood, Kircher, Santos, and Tertilt (2017).

transfers clear the market and depend on the desires of men relative to women. There is also an adverse selection problem: Individuals with a proclivity toward risky sexual behavior are inclined to enter the market for casual unprotected sex. As a consequence, this market tends to have a high rate of HIV. The private information problem regarding a partner's health status dissuades healthy individuals, without such a proclivity toward risky sexual behavior, from entering this market. This exacerbates the riskiness of the market for casual unprotected sex.

The model is solved numerically and calibrated in Section 4 to match key moments of HIV and sexual behavior in Malawi. The presence of multiple markets, private information, and randomness in meeting a partner and in contracting HIV renders the model too complicated for analytical results. The calibration matches most targeted moments well, even though the model is sparse on gender differences.

Four policy experiments are conducted in Section 5: male circumcision (Section 5.1), ART (Section 5.2), better condoms (Section 5.3), and the treatment of other sexually transmitted diseases or STDs (Section 5.4). Medical research suggests that male circumcision reduces the female-to-male transmission risk of HIV. Small-scale field experiments suggest that circumcised males engage in more risky sexual behavior. The model shows that if one simply applied this reduced transmission risk and behavioral changes at the individual level, without accounting for the general equilibrium spillover effects on their partners, the HIV rate would only be moderately affected by circumcision.³ Yet in cross-country data—which serve as the next-best assessment in lieu of large-scale circumcision experiments—one sees large changes in the HIV rate with respect to the fraction of circumcised males. The calibrated model predicts that male circumcision is indeed quite effective in curbing HIV prevalence. The beneficial effects of reduced HIV transmission powerfully accumulate in equilibrium and dominate the (nevertheless non-trivial) behavior adjustments going in the opposite direction. At the country level, equilibrium forces kick in as not only individuals, but also their partners, and their partners' partners, are affected. The model replicates the (non-targeted) cross-country evidence on circumcision, yielding credibility to the results that it produces.

ART is a complex policy. By treating the sick, infected people live longer and have more time to infect others. Moreover, since infection is no longer perceived as such a bad event, healthy people engage in more risky behavior. Even though the treated are less infectious to others, these effects may dominate and HIV may actually rise. The quantitative results show that whether ART reduces or increases the HIV rate depends on the fraction of the infected population treated. When less than 50% of the infected are treated, the two effects roughly cancel out, and the HIV rate is largely unaffected. Only when more than half of the infected are treated does the reduced infectiousness become powerful enough to decrease the overall HIV rate.

The acceptability of condoms is also a major issue. Studies attribute low acceptability to a number of hard technical problems such as “comfort and breakage” and “unpleasant smell,” as well as soft attitudinal problems due to their stigmatization because of the link to STDs. Suppose one could increase the utility from condom usage, through either technological or attitudinal interventions. The simulations suggest that better condoms

³Throughout the paper, “HIV rate” is used as shorthand for “HIV prevalence rate.” Prevalence is a stock variable. One might also be interested in the flow or incidence, that is, new infections. Whenever incidence (measured as new infections per 1000 healthy people) is meant instead, this is explicitly specified. In the model, there is a tight relationship between incidence and prevalence, since the analysis focuses on steady states. Hence, the numbers for prevalence are often the only ones reported.

have a potential to backfire. People would use them, but they would in parallel increase their sexual activity in single life (both protected and unprotected), which could lead to an increase in the HIV rate. Medical research also suggests that the treatment of STDs leads to a reduction in the transmission risk of HIV. The analysis suggests that this could have a moderate effect on reducing HIV.

Finally, the paper analyzes the importance of information about the disease. An extension is presented in Section 6 in which a fraction of the population is unaware that abstinence and condom use reduce the odds of infection. The extension is used to investigate to what extent better information contributed to the HIV decline over time. When reducing the fraction of uninformed people in line with the data, the model predicts a decline in HIV incidence between 1996 and 2004 comparable to the one observed in the data. The reason is that better informed people engage in less risky behavior. Thus, better information probably played a large role in the positive changes Malawi has seen in its HIV rates.

This work ties in with two other scientific methodologies used to study HIV/AIDS, namely, epidemiological studies and small-scale field experiments. The policy experiment results obtained from the choice-theoretic general equilibrium model are compared with those arising from synthetic versions of epidemiological studies and small-scale field experiments. The epidemiological literature on disease transmission in general, and HIV in particular, is large, but does not model decision-making and consequently takes sexual behavior as exogenous (see [Hethcote \(2000\)](#) for a comprehensive overview of the mathematical modeling of infectious disease). Additionally, the infected and uninfected mix randomly.⁴ Several studies suggest that people react to a higher presence of HIV/AIDS by adjusting aspects of their sexual behavior ([Wellings, Field, Johnson, Wadsworth, and Bredshaw \(1994\)](#)). As will be discussed, behavioral changes can reduce the effectiveness of public policies if people switch to riskier sexual practices. Thus, by neglecting to account for behavioral changes, epidemiological studies may overstate the efficacy of some public policies.

There is a large recent literature that studies HIV/AIDS prevention policies using field experiments (see [Padian, McLoy, Balkus, and Wasserheit \(2010\)](#) for a survey). The vast majority of field experiments treat only a small segment of the population, but the insights arising from these studies are very valuable and are used in the present study to calibrate some parameters (such as in the circumcision experiment). While small-scale field experiments establish the effect of a treatment on an individual, they neglect general equilibrium effects. Public policy treatments affect not only individuals, but also their partners, their partners' partners, and so on. These effects may accumulate in the population as a whole. Hence, small-scale field experiments may understate the efficacy of some public policies, such as circumcision and the treatment of STDs.

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. Overall, this research program aims to develop tools to aid researchers and practitioners, and highlights areas where further and more in-depth research should be conducted. Some background information on sexual behavior and HIV/AIDS in Malawi is now provided.

⁴[Kremer and Morcom \(1998\)](#) allowed for different groups in the population to have different mixing rates. This idea also features prominently here, but is applied to the type of sexual behavior that a person *purposely* seeks.

2. FAMILIES, SEXUAL BEHAVIOR, AND HIV/AIDS IN MALAWI

The Republic of Malawi is a country in southeast Africa with a population of 14 million. Malawi suffers greatly from the HIV/AIDS epidemic. In 2004, 12% of the adult population was infected.⁵ This was well above the average within Sub-Saharan Africa (SSA), which has an adult prevalence rate of about 7.2% (Canning (2006)). However, it was below the HIV rate of the most affected countries, such as Botswana, with its adult prevalence rate of 37%. As in many other countries, HIV in Malawi displays a hump-shaped profile over time. The HIV prevalence rate peaked in 1999 and has been continuously falling since. The incidence rate peaked earlier in 1994, with 21.8 infections per 1000 healthy population.

The principal mode of HIV transmission in Malawi is through heterosexual sex. Mother-to-child transmissions account for about 10% of all new HIV infections. This fact is ignored here, as most people born with HIV die before they reach sexual maturity and therefore do not add to the propagation of HIV. As with the rest of Sub-Saharan Africa, more than half of the HIV-infected population in Malawi are women. By contrast, two-thirds of the infected population in the Western world are men. The HIV rate among adult women was 13% in 2004, compared to 10% among men, suggesting important gender differences.

A rational behavior model of HIV only makes sense if people understand what HIV is, are aware of how it gets transmitted, and know how to avoid it. This seems largely to have been the case in Malawi since the 2000s. Almost 100% of 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—knew where to get condoms. Further, Delavande and Kohler (2009) documented that people in Malawi are relatively good at assessing their own probability of being infected with HIV. Prior to 2000, people were less informed about the HIV transmission mechanism, and due to this ignorance might not have switched to safer sexual practices. The analysis here examines whether the diffusion of information about HIV could have contributed to the observed decline in the Malawian HIV rate as people engaged in less risky sexual behavior.

Treating HIV with antiretroviral drug therapy (ART) was not introduced until the mid-2000s in Malawi. ART increased gradually from 3% of the infected in 2005 to 50% in 2014. The importance of ART will be examined in detail in this paper. Further, about 20 percent of the male Malawian population were circumcised in 2004, a rate which rose only slightly (to 22%) in 2010. Circumcision seems largely related to religion and ethnicity rather than health concerns, which explains its relative stability over time.⁶ There has been a slight uptake more recently, likely related to official HIV prevention interventions, so that by 2015, 28% of men were circumcised.

Sexual behavior conducive to the spread of the disease is relatively common in Malawi. Condoms were used by less than half of the population in their last sexual act. It is also considered normal for unmarried people to change partners often (Undie, Crichton, and Zulu (2007)). Divorce is relatively common, with Reniers (2003) reporting that 45% of marriages end in divorce within 20 years. Several other forms of risky behavior are abstracted from in the paper to keep the model tractable. For example, concurrent multiple relationships, such as extramarital affairs or polygyny, are not modeled.

⁵Unless 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.

⁶For example, in Malawi 93% of Muslims are circumcised but only 8.6% of Catholics. Similarly, 82% of the Yao ethnicity are circumcised but only 2% of the Tumbuka. See 2004 DHS Final Report.

Just because people engage in risky sexual behavior does not necessarily imply that they are uninformed or irrational: The decision could be due to a trade-off between increased safety versus decreased pleasure. For example, sex with a condom is often compared to eating candy “while it’s in the wrapper” or a banana with its peel (Undie, Crichton, and Zulu (2007)). Condom use within marriage is also essentially non-existent in Malawi (Chimbiri (2007)). Using a condom in marriage may be interpreted as a signal of infidelity (Bracher, Santow, and Watkins (2004)). Note that while using a condom lowers transmission risk substantially, it does not decrease the risk to zero. Bracher, Santow, and Watkins (2004) cited 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.

Poulin (2007) documented that money and gift transfers in sexual partnerships are part of courting practices in Malawi. In addition to an expression of love and commitment, she argued that these transfers are a way of acquiring sex for men and meeting their financial needs for women. A gift might be in the form of sugar or soap, but also in cash. Transfers are not made directly before or after sex (as with prostitution), however; rather, gift giving is an integral part of a relationship. The model allows for such transfers between men and women.

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, namely, short-term (“casual”) 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 between partners. It provides less enjoyment, though. Individuals interested in a short-term relationship must decide what type of sex they desire. Put simply, they must weigh the extra momentary utility associated with unprotected sex against the increased odds of contracting the HIV/AIDS virus. 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 $u > p > 0$. The utility flow in a long-term relationship is $u + l$, where l may be negative. A positive l can be interpreted as a preference for long-term attachment, while a negative l can be construed as taste for variety in partners. Individuals also realize utility from the consumption of goods. Let this utility be given by $\ln(w)$, where w is consumption (income minus transfer). Each period, a person receives income in the amount y . There is no borrowing or saving in the economy.⁷ An individual discounts the future with a stochastic discount factor that takes two values, namely, $\tilde{\tau}$ and $\tilde{\beta}$ with $\tilde{\tau} < \tilde{\beta}$. There is a distribution across individuals for the $\tilde{\tau}$ s. Connected with each $\tilde{\tau}$ is a $\tilde{\beta}$. A person starts life with the low rate $\tilde{\tau}$ (dubbed “young”). This low factor reflects the impatience of youth, which results in a predilection for engaging in risky behavior.⁸ Then, in every period, a person may switch permanently to the high factor (dubbed “old”) with probability η . Additionally, there is a probability δ that an individual dies from natural causes in a period.

⁷Magalhães and Santaaulàlia-Llopis (2018) provided evidence for the lack of savings in Malawi.

⁸The impatience of youth and the resultant increase in risk-taking behavior has been documented in many contexts. For example, Rolison, Hanoch, Wood, and Liu (2013) found that risk-taking attitudes drop smoothly with age, especially in the health domain.

Thus, the effective discount factors are given by $\iota \equiv \tilde{\iota}(1 - \delta)$ and $\beta \equiv \tilde{\beta}(1 - \delta)$. People differ in the (ι, β) pair from which their discount factors in youth and old age are drawn. Finally, a male individual may be circumcised (denoted by $c = 1$) or not ($c = 0$). A circumcised male is less likely to contract the HIV virus from his sexual partner. The values of c , ι , and β differ across individuals of a given gender. The set of fixed characteristics for a person is denoted by $x = (c, \iota, \beta)$, called a person's type.

People can search for partners in different markets.⁹ Depending on how much they value a particular type of relationship, people choose their search effort by picking the odds of finding a partner, which comes at a cost. At the beginning of each period, an unattached individual may search for a long-term partner. The odds of finding a partner in the long-term market are denoted by π_l . The individual picks these odds at an increasing cost in terms of lost utility. These search costs are given by $C_l(\pi_l) = \omega_l[\pi_l/(1/2 - \pi_l)]^{\kappa_l+1}$, where $\kappa_l \geq 0$ and $\omega_l > 0$. Observe that $C_l(0) = 0$ and $C_l(1/2) = \infty$. A long-term relationship may break up (at the end of) each period with exit probability ϵ . If the person is unsuccessful at finding a long-term mate, then they enter the short-term market, where they 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 casual sex, they can choose to search for protected and unprotected sex simultaneously. Let π_p and π_u represent the odds of finding a partner in the protected and unprotected markets for short-term relationships, which are choice variables. If she prefers one type of sex over the other, she can choose the effort (and hence the odds) accordingly. 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 κ_s and ω_s are allowed to differ from the long-term market. The total cost of searching for a short-term partner will then be $C_s(\pi_p) + C_s(\pi_u)$. An individual cannot simultaneously draw a partner in both markets. Since $C_s(1/2) = \infty$, the odds are such that $\pi_p + \pi_u < 1$, and a single will be abstinent with probability $\pi_a \equiv 1 - \pi_p - \pi_u$. Also, observe that individuals can choose abstinence by picking $\pi_p = \pi_u = 0$.

The probabilistic nature of finding a partner in the three markets is meant to capture the randomness of meeting someone in everyday life. Finding a partner is costly. The more you invest in it, the more likely you are to succeed. While π_l , π_p , and π_u represent the probabilities of an individual finding a partner in the three markets, they also represent the fractions of people searching in each market that will find a partner, given the large nature of the economy. Finally, this randomness in the meeting structure helps to smooth out the equilibrium under study because people who are currently of the same type will evolve differently depending on the relationship they enter into.

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, z , that is made between the two partners. For the person making the transfer, z 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. The magnitude of this transfer is determined in equilibrium. It will depend upon the demand and supply for a given type of relationship by each gender. This hinges on the utility that each gender realizes from a partnership in the various markets and the riskiness of participating in them.

⁹The idea that people can rationally target their search behavior to particular markets is present in many recent theoretical models, for example, [Eeckhout and Kircher \(2010\)](#).

People know their own health status ϕ .¹⁰ A healthy individual has $\phi = 1$. An individual with HIV infection and no antiretroviral therapy (ART) treatment has $\phi = 0$. An infected individual who receives treatment has health status $\phi = t$. So, $\phi \in \{0, 1, t\}$. All individuals are born healthy. If a non-circumcised individual has sex with a partner with health status $\hat{\phi}$, then the virus will be transmitted with probability $1 - \gamma(\hat{\phi})$, which is trivially zero if the other individual is healthy.¹¹ Further, $1 - \gamma(t) < 1 - \gamma(0)$, so that a treated individual is less likely to infect others. Similarly, an individual of type ϕ transmits the infection to a healthy partner with probability $1 - \hat{\gamma}(\phi)$. The transmission probabilities can differ by gender. Circumcised men are also less likely to contract the virus compared to non-circumcised individuals. Denote this reduction in transmission probabilities that circumcision provides by $\chi(c)$, with $\chi(1) = \chi < 1$ and $\chi(0) = 1$. For women, $\chi(c) \equiv 1$; that is, circumcision is not a factor. These transmission probabilities are also lower for protected sex than for unprotected sex. People only know their own health status. While they cannot discern the health status of other individuals, they hold correct expectations, $R_r(\hat{\phi})$, about the fraction of potential partners of each health status in each market, $r = l, p, u$. (As will be discussed later, for women $R_l(\hat{\phi})$ will also depend on whether or not their partner is circumcised.)

If treatment is available, a currently infected untreated individual ($\phi = 0$) will enter next period under ART with probability q . Once in treatment, the individual remains in treatment forever. So the probability of obtaining treatment in the next period, conditional on current health status, ϕ , can be summarized by the function $Q(\phi)$, where $Q : \{0, 1, t\} \rightarrow \{q, 0, 1\}$; that is, a currently infected untreated person obtains treatment in the next period with probability $Q(0) = q$; a healthy person will not receive treatment, corresponding to $Q(1) = 0$; and a treated person will still receive ART, implying $Q(t) = 1$.

The health status indicator refers to individuals in the early stages of the disease, where it is assumed that their health status is not visible to other people and neither income nor sexual activity is restricted. Individuals transit to the final stages of AIDS (where they display symptoms) with probability α_ϕ , where $\alpha_1 = 0 < \alpha_t < \alpha_0$, since healthy individuals do not enter the final stage of the disease and treatment prolongs a healthy existence. Assume that a person stricken with final-stage HIV/AIDS symptoms engages in no further relationships. Let the remaining lifetime utility for a person with final-stage HIV/AIDS symptoms be represented by A . The probability that a person displaying symptoms dies is δ_2 . Since a person with HIV/AIDS symptoms engages in no further sexual activity, δ_2 does not appear in the value functions. It is still relevant for computing the average HIV/AIDS rate in society.

Note that in the framework, there is attrition in the population each period due both to natural death and to HIV/AIDS. This loss is replenished by an inflow each period of newly-born females and males. Assume that $\mu(x)$ type- x individuals are born at the beginning of each period. Recall that x denotes the set of permanent characteristics for an individual, namely, c , ι , and β . 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.

¹⁰Greenwood, Kircher, Santos, and Tertilt (2013) explored the evolution of beliefs about a person's own likelihood of being HIV-positive in a world where individuals do not immediately observe their own health status. People update their beliefs about being healthy in a Bayesian fashion, conditional on their past sexual history (and also on what they observe about their partners in long-term relationships). Daron Acemoglu is thanked for suggesting the current simplification.

¹¹The symbol ' $\hat{\cdot}$ ' denotes the characteristics of an individual's partner.

Before proceeding to the formal analysis, some notations will be defined. An individual will be indexed by his health status, $\phi \in \{0, 1, t\}$, his current discount factor, d , drawn from his fixed sample space $\{\iota, \beta\}$, and his exogenous type, $x = (c, \iota, \beta)$. Let $\tilde{V}_r^d(\phi, x)$ denote the expected lifetime utility for a person with health status ϕ , realized discount factor d , and 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 but has not yet found a partner in a type- r relationship (for $r = l, s$, where s denotes short-term). The timing of events is summarized in Figure 5 in Appendix A (Greenwood, Kircher, Santos, and Tertilt (2019)). 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 is on studying a stationary equilibrium.

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 who differ in their discount factor. Start with a type- x old person (i.e., with discount factor β) with health status ϕ , who has failed to match on the short-term sex markets. Thus, the person will be abstinent in the current period. Note that the individual's discount factor will remain high forever. The value function for this person is given by

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

for $\phi = 0, 1, t$. The first term on the right-hand side covers the flow utility from current consumption. The continuation value depends on whether the person enters the final stages of the disease, which happens at rate α_ϕ and yields a discounted continuation value of βA . With complementary probability, $1 - \alpha_\phi$, the individual's continuation value is the discounted expected value of either continuing in their current state or of perhaps obtaining treatment. Healthy individuals have a zero probability of entering the final stages of the disease or of obtaining treatment, so their value function reduces simply to $\tilde{V}_a^\beta(1, x) = \ln(y) + \beta V_l^\beta(1, x)$, where the discount factor already incorporates other sources of death separate from HIV/AIDS.

Next, consider the case of an abstinent young person (with discount factor ι). The discount factor may switch in the next period to the high value, β , with probability η , or remain at the low one, ι , with probability $1 - \eta$. Therefore, the value functions for young individuals retain the same structure as (1) with two modifications: first, the discount factor β needs to be replaced by ι , and second, the value function $V_l^\beta(\phi, x)$ on the right-hand side has to be replaced by the expected value $\eta V_l^\beta(\phi, x) + (1 - \eta) V_l^\iota(\phi, x)$ for all ϕ . This difference between the value functions for young and old individuals holds throughout the analysis. Therefore, the focus is placed on defining the value functions for old individuals, with the adjustments for young individuals summarized in Appendix C.1.

3.1.2. Sexual Relationships

Now consider short-term relationships. Here, individuals have to take into account both the transfers in each market as well as the distributions over the health status of the partners they might encounter. Additionally, they also experience the utility from sexual activ-

ity. For now, assume a person is already matched. (The search behavior for an unmatched individual will be described at the end of this section.)

Again, focus on an old individual with a high discount factor, β . 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 flow joy from a short-term sexual relationship can be written as $pI(s) + u[1 - I(s)]$. Apart from this addition and the incorporation of the transfer z_s in the short-term market, the value function of infected individuals with $\phi \in \{0, t\}$ looks similar to the one for abstinence (1):

$$\begin{aligned} \tilde{V}_s^\beta(\phi, x) = & \ln(y - z_s) + pI(s) + u[1 - I(s)] + \alpha_\phi \beta A \\ & + (1 - \alpha_\phi) \beta \{Q(\phi)V_t^\beta(t, x) + [1 - Q(\phi)]V_l^\beta(0, x)\}, \end{aligned} \tag{2}$$

for $s = p, u$. It comprises the utility from current consumption and sexual activity, and the continuation value of either entering the last stages of the disease or continuing as either an infected or treated individual.

The type of sexual activity in the current period that a healthy person ($\phi = 1$) engages in affects the odds that they will enter next period infected with HIV. First, the transmission risk of catching HIV/AIDS from an infected person differs across markets. Specifically, the transmission risk in the protected market is lower than in the unprotected one. Second, the average level of healthiness in the pool of participants in the two markets will differ. The fact that a person desires a short-term sexual relationship that does not include condom use signals something about their past tendencies to engage in risky behavior. For instance, in the market for protected sex, the fraction of healthy partners $R_p(1)$ will presumably be higher than the corresponding fraction $R_u(1)$ in the market for unprotected sex. If the individual enters the next period newly infected with HIV, it will still take at least one period for the symptoms of AIDS to appear. The value function for a healthy individual who engages in sex of type $s = u, p$ is then

$$\begin{aligned} \tilde{V}_s^\beta(1, x) = & \ln(y - z_s) + pI(s) + u[1 - I(s)] \\ & + \sum_{\hat{\phi}} R_s(\hat{\phi}) [1 - \gamma_s(\hat{\phi})] \chi(c) \beta [qV_t^\beta(t, x) + (1 - q)V_l^\beta(0, x)] \\ & + \left\{ 1 - \sum_{\hat{\phi}} R_s(\hat{\phi}) [1 - \gamma_s(\hat{\phi})] \chi(c) \right\} \beta V_l^\beta(1, x). \end{aligned} \tag{3}$$

It entails the flow utility of consumption and sex and the continuation values. The individual gets infected with probability $\sum_{\hat{\phi}} R_s(\hat{\phi}) [1 - \gamma_s(\hat{\phi})] \chi(c)$. This infection probability takes into account the distribution in market s over health status, as given by $R_s(\hat{\phi})$. For each health status, it factors in the transmission risk, $[1 - \gamma_s(\hat{\phi})] \chi(c)$, which depends on whether the individual is circumcised. If the individual contracts the virus, they have the probability q of getting ART in the next period and obtaining a treated person's continuation utility, $\beta V_t^\beta(t, x)$; while continuation as an untreated infected individual, which has value $\beta V_l^\beta(0, x)$, arises with probability $1 - q$. With probability $\{1 - \sum_{\hat{\phi}} R_s(\hat{\phi}) [1 - \gamma_s(\hat{\phi})] \chi(c)\}$, the individual remains healthy, in which case they obtain the continuation utility $\beta V_l^\beta(1, x)$.

Finally, to be matched in the short-term market, a person must first decide how much effort to expend searching in each market; that is, they must choose π_p and π_u . This is

done in accordance with the following problem:

$$\begin{aligned}
 V_s^d(\phi, x) = \max_{\substack{0 \leq \pi_u^d, \pi_p^d, \\ \pi_u^d + \pi_p^d < 1}} \{ & \pi_p^d \tilde{V}_p^d(\phi, x) + \pi_u^d \tilde{V}_u^d(\phi, x) + (1 - \pi_p^d - \pi_u^d) \tilde{V}_a^d(\phi, x) \\ & - C(\pi_p^d) - C(\pi_u^d) \}, \quad \text{for } d = \iota, \beta, \tag{4}
 \end{aligned}$$

where, with a slight abuse of notation, the subscript s now simply denotes “short term.” The value function $V_s^d(\phi, x)$ gives the ex ante value for a type- x individual, with discount factor d and health status ϕ , of entering the market for short-term sex. The solution for the 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 with a high discount factor, β , 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. A long-term relationship may end due to an exogenous divorce, the partner dying of natural causes, or the partner developing the symptoms of HIV/AIDS.

To understand the value of a continuing relationship in which an individual of health status ϕ and circumcision type c is married to a partner of status $\hat{\phi}$ and circumcision type \hat{c} , it is important to derive the probability $Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c})$ that the pair enters next period together with status ϕ' and $\hat{\phi}'$. A couple who is currently healthy will stay healthy, so that $Y(1, 1 | 1, 1, c, \hat{c}) = 1$ for all (c, \hat{c}) . To illustrate a more complicated case, the following equation presents the probability of a transition to a state in the next period where the individual is infected and the partner is treated, from a state in this period where the individual is healthy and the partner is infected or treated ($\hat{\phi} = 0, t$):

$$Y(0, t | 1, \hat{\phi}, c, \hat{c}) = [1 - \gamma_u(\hat{\phi})] \chi(c) (1 - q) Q(\hat{\phi}). \tag{5}$$

The first term captures the chance that the individual becomes infected, $[1 - \gamma_u(\hat{\phi})] \chi(c)$. The second and third terms capture the probabilities that the individual is not treated, $1 - q$, but the partner is, $Q(\hat{\phi})$. All other transition probabilities are described in Appendix C.2.

Given these transition probabilities, consider an individual of health status ϕ who starts the period matched to a partner of health status $\hat{\phi}$ and circumcision type \hat{c} . Focus on a high-discount-factor individual for illustration. His continuation utility is

$$\begin{aligned}
 \tilde{V}_l^\beta(\phi, \hat{\phi}, \hat{c}, x) = & \ln(y - z_t) + u + l + \alpha_\phi \beta A \\ & + (1 - \alpha_\phi)(1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}}) \\ & \times \beta \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c}) \tilde{V}_l^\beta(\phi', \hat{\phi}', \hat{c}, x) \\ & + (1 - \alpha_\phi) [1 - (1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}})] \\ & \times \beta \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c}) V_l^\beta(\phi', x). \tag{6}
 \end{aligned}$$

The first three terms on the first line capture the flow utility from consumption and sex in long-term relationships. Additionally, the individual develops final-stage symptoms with

probability α_ϕ , which is captured by the last term in the first line. With complementary probability, $1 - \alpha_\phi$, the individual remains either married (second and third lines) or becomes single (fourth and fifth lines). The marriage will persist if there is no exogenous breakup (chance $1 - \epsilon$), the partner does not die of natural causes (chance $1 - \delta$), and the partner does not develop symptoms (chance $1 - \alpha_{\hat{\phi}}$). In this case, the individual obtains the continuation value connected with marriage, $\beta \tilde{V}_i^\beta(\phi', \hat{\phi}', \hat{c}, x)$, taking into account the transition probability of health status. With complementary probability, the marriage breaks up and the individual carries his new status as a single into the long-term market with associated continuation value, $\beta V_i^\beta(\phi', x)$.

Since individuals do not know their partner's health status, the value of being matched in the long-term market for an individual with discount factor $d = \iota, \beta$ and health status $\phi = 0, 1, t$ is given by the weighted average of possible partners in the long-term market:

$$\tilde{V}_i^d(\phi, x) = \sum_{\hat{\phi}, \hat{c}} R_i(\hat{\phi}, \hat{c}) \tilde{V}_i^d(\phi, \hat{\phi}, \hat{c}, x). \tag{7}$$

Note that, in long-term relationships, unlike short-term relations, the relevant fraction $R_i(\hat{\phi}, \hat{c})$ also depends on the circumcision type of the potential partner. This is so because the circumcision type of the spouse matters for the evolution of the marriage, as can be seen from the probabilities given in Appendix C.2.¹²

The value of searching in the long-term market for a status/type- (ϕ, x) person with discount factor d is given by

$$V_i^d(\phi, x) = \max_{\pi_i^d} [\pi_i^d \tilde{V}_i^d(\phi, x) + (1 - \pi_i^d) V_s^d(\phi, x) - C(\pi_i)], \tag{8}$$

for $d = \iota, \beta$ and $\phi = 0, 1, t$. The solution for the search effort, π_i^d , is represented by the function $\pi_i^d = \Pi_i^d(\phi, x)$.

3.3. Stationary Equilibrium

The analysis focuses on a steady-state competitive equilibrium where the number of people of a particular gender, discount factor, permanent type, and treatment status is constant through time. From the above dynamic programming problems, time-invariant decision rules arise for search in each of the three markets. By using these decision rules, the steady-state type distribution can be formulated.¹³ In the equilibrium modeled, beliefs about the prevalence rates in each market are rational and thereby consistent with the time-invariant decision rules and steady-state type distribution. Decision rules are optimal given beliefs and prices. Finally, prices are market clearing, so that the number of men who form a sexual relationship in a given market equals the number of women who form a relationship in that market. Refer to Appendix C.3 for an exact definition of the equilibrium and for more information on its formulation.

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

¹²This is only relevant for women as just men are circumcised; that is, in the model, a man cannot have a circumcised mate.

¹³Appendix C.4 outlines how to derive the steady-state type distributions.

with the Malawian case. Interpret a model period as lasting one quarter. The calibration is conducted in three steps. First, parameters with direct data analogs are assigned values from the literature. In particular, all parameters relating directly to the biology of the disease are chosen this way. Second, the remaining parameters are selected to match some stylized facts about the HIV/AIDS epidemic in Malawi. The stylized facts are based mostly on micro data from the 2004 Demographic and Health Survey (DHS) that was conducted in Malawi. Third, the model's predictions are compared (in Sections 5 and 6 and Appendix E) to the Malawian data along several non-targeted dimensions.

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 medical literature. The transmission risk for one-time male unprotected sex is taken to be 4.5 per 1000 encounters. This number falls in the range of estimates reported by a variety of studies.¹⁴ 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 $\gamma_u^m = 0.879$.¹⁵ The transmission risk when condoms are used is obviously lower, but protection is far from perfect (Bracher, Santow, and Watkins (2004)). Select $\gamma_p^m = 0.96$, corresponding to a 67% efficacy rate, which is in line with Weller (1993), who conducted a meta-analysis of condom efficacy. Assume that circumcised men are 60% less likely to contract the HIV/AIDS virus and set $\chi = 0.4$ accordingly. This accords with the improvements reported by Auvert, Taljaard, Lagarde, Sobngwi-Tambekou, Sitta, and Puren (2005), Bailey et al. (2007), and Gray et al. (2007). Set the fraction of circumcised men to 20% (DHS Final Report (2004)). Further, for physiological and anatomical reasons, and in accord with the medical evidence, women are assumed to have a higher risk of contracting HIV than men. Nicolosi, Correa Leite, Musicco, Arici, Gavazzeni, and Lazzarin (1994) reported a risk that is 2.3 times higher for women.¹⁶ However, the range of estimates is wide. At one extreme, Gray et al. (2001) found no statistically significant difference between transmission rates by gender. At the other extreme, Padian, Shiboski, and Jewell (1991) calculated a factor as high as 20. Assuming that women are 75% more likely to get infected implies a one-time risk of 7.87 per 1000 encounters for unprotected sex. This delivers a quarterly non-transmission rate of $\gamma_u^f = 0.787$. Using the same gender gap for protected sex yields $\gamma_p^f = 0.929$.

The average time from infection to the outbreak of symptoms is equal to 10 years (DHS Final Report (2004)). Therefore, let $\alpha = 0.025$, that is, 40 quarters. The average time from the outbreak of symptoms to death is 2 years (DHS Final Report (2004)). Thus, set $\delta_2 = 0.125$, that is, 8 quarters.

Some other parameter values can also be pinned down using a priori information. Set the quarterly divorce hazard equal to $\epsilon = 0.03$. Bracher, Santow, and Watkins (2004) reported 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%. A rate twice this number is used: First, polygyny is fairly common in Malawi, from which

¹⁴For example, Baeten, Richardson, Lavreys, Rakwar, Mandaliya, and Bwayo (2005) reported a transmission risk of 6 per 1000. A lower number of 1.1 per 1000 for Uganda was documented by Gray et al. (2001); however, free condoms were distributed as part of the study.

¹⁵This quarterly rate also is similar in magnitude to the per-partnership transmission rates for Sub-Saharan Africa reported in Oster (2005) and the correction appendix.

¹⁶Oster (2005) also reported a factor of 2 in her measures of per-partnership transmission rates.

TABLE I
PARAMETERS CHOSEN OUTSIDE THE MODEL

Parameter	Value	Interpretation
γ_u^m	0.879	12.1% quarterly transmission risk, unprotected sex, uncircumcised men
γ_p^m	0.96	4% quarterly transmission risk, protected sex, uncircumcised men
χ	0.4	Circumcised men are 60% less likely to contract HIV
γ_u^f	0.787	21.3% quarterly transmission risk, unprotected sex, women
γ_p^f	0.929	7.1% quarterly transmission risk, protected sex, women
α	0.025	10 years from infection to symptoms
δ	0.006	6% quarterly death risk
δ_2	0.125	2 years from symptoms to death
ϵ	0.03	3% quarterly divorce hazard
y	320	Quarterly income

the analysis abstracts. Second, extramarital affairs are relatively common as well. Therefore, interpret, for example, a 10-year marriage with one affair as two separate long-term relationships with a third casual one in between.¹⁷

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 $y = \$320$ (U.S.), which corresponds roughly to quarterly GDP per working age population in 2001 (only about half the population is of working age in Malawi). Table I summarizes the preceding paragraphs by listing all parameters that are set a priori.

4.2. Parameters Chosen to Match Data Moments

The remaining parameters have no clear data analogs. For example, the utilities from the different types of sexual relationships are free parameters. The values for these parameters are picked to match several stylized facts related to sex, marriage, and HIV/AIDS in Malawi.

As specified above, the only exogenous heterogeneity (in addition to the difference in transmission risks for circumcised and uncircumcised males and the difference in transmission risks across genders) is the degree of patience people have. Recall that $\tilde{\tau}$ and $\tilde{\beta}$ denote the discount factors for the young and old. These are “pure” discount factors, that is, net of mortality risk.¹⁸ Suppose that β varies across individuals according to a uniform distribution with support $[\beta_{\min}, \beta_{\max}]$. Moreover, assume the ratio of discount factors when young and old is always given by the same value $\iota_{\text{change}} = \tilde{\tau}/\tilde{\beta} < 1$. Thus, there are 11 free parameters: $p, u, \ell, \omega_s, \omega_l, \kappa, A, \eta, \tilde{\beta}_{\min}, \tilde{\beta}_{\max}$, and ι_{change} . See Table II for a summary. To discipline the choice of the parameters, 11 data moments are targeted. Table III presents the data moments and shows how well the benchmark model matches them.¹⁹

¹⁷Greenwood et al. (2017) explored what happens to equilibrium outcomes when the risk of divorce is lower.

¹⁸That is, $\beta \equiv \tilde{\beta}(1 - \delta)$ and $\iota \equiv \tilde{\tau}(1 - \delta)$.

¹⁹All data sources for the tables and figures are discussed in Appendix B.

TABLE II
CALIBRATED PARAMETERS

Interpretation	Parameter Value
Flow utility unprotected sex	$u = 7.8$
Flow utility protected sex	$p = 1.4$
Flow utility long-term sex	$l = -4.8$
Discount factor, min and max support	$\tilde{\beta}_{\min} = 0.969, \tilde{\beta}_{\max} = 0.9999$
Ratio discount factors, young vs. old	$\iota_{\text{change}} = 0.874$
Value of life with AIDS	$A = 5.8$
Prob. of switch to high discount factor	$\eta = 0.116$
Search cost parameters	$\omega_s = 0.44, \omega_l = 17.5, \kappa = 0.115$

The upshot of the analysis is that the benchmark simulation matches these key features of the Malawian HIV/AIDS epidemic pretty well.²⁰ To begin with, as can be seen from Table III, the HIV/AIDS prevalence rate predicted by the model is 10.3%, close to the 11.8% in the data. Moreover, the benchmark simulation captures the gender difference in HIV/AIDS infection rates, with females experiencing a rate that is 3.5 percentage points higher than that for males (12.1% versus 8.6%). The model also captures well the HIV incidence rate (which was not specifically targeted). In the model, 11.7 per 1000 healthy people are newly infected every year, compared to 11.1 in the data.

In addition to matching moments on HIV/AIDS prevalence, the benchmark model also mimics some other moments relevant for sexual activity quite well. In the model, casual or short-term sex represents a small fraction of all sexual encounters: 16%, 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

TABLE III
TARGETED MOMENTS

Observation	Data	Model
HIV/AIDS prevalence rate, %	11.8	10.3
Males	10	8.6
Females	13	12.1
Sex that is casual, % (of all)	18	16
Condom use for casual sex, %	39	33
Singles that had casual sex in past year, %	47	54
Singles, %	33	48
Married by age 22, %		
Males	58	57
Females	90	63
Married by age 50, %		
Males	100	98
Females	100	98
Deaths related to HIV, %	29	25

²⁰Appendix F provides sensitivity analysis for these estimated parameters. Both benchmark moments and the response to some policy experiments (discussed in Section 5) are analyzed.

protected sex they have (Allen et al. (2003)), these two numbers may be closer in reality than first meets the eye. The next row in Table III reports the fraction of singles who had casual sex in the last year. These statistics are different from the fraction of all sexual activity that is casual because (all) married people have sex while some singles are abstinent. Singles in the model have a little more casual sex than their real-life counterparts (54 versus 47%). Finally, the fraction of the population that dies from HIV/AIDS is comparable across model and data (25 versus 29%).

The fraction of singles in the entire population is 48% in the model, somewhat higher than the 33% observed in the data, which is partially due to the modeling choice to break up marriages more frequently than in the data to account for extramarital affairs. The model also 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 63% and 57%. In the model, women marry earlier than men due to the higher female infection risk, which makes the safety of marriage more attractive for women compared to men. In reality, HIV risk is only one reason why women marry earlier than men; other reasons may include the biological clock, polygyny, and the fear of pregnancy. By age 50, almost everyone is married, in both the data and the model.

In Section 5, the model's predictions are compared with cross-country data on the relationship between HIV and male circumcision. Additionally, in Section 6, time-series data for Malawi are confronted with a version of the model with a richer information structure regarding knowledge about HIV. Last, the framework also captures surprisingly well some non-targeted life-cycle moments—see Appendix E. This all provides additional validity checks on the calibration.

5. POLICY EXPERIMENTS

The model is now ready to explore the effectiveness of various policies intended to curb the spread of HIV/AIDS. This section investigates four specific policies: male circumcision, ART, better condoms, and the treatment of other STDs. The first two policies have been at the forefront of the policy discussion.

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. The synthetic small-scale field experiment is a partial equilibrium version of the general equilibrium one. It 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 the *preexisting* equilibrium prices and infection rates. The synthetic epidemiological experiment assumes that people make no behavioral adjustments. It therefore uses the policy functions from the benchmark calibration in conjunction with the new exogenous transmission probabilities.

Comparing the effectiveness of policies across the three versions of the model can be thought of in two ways. First, they can be viewed as thought experiments. A comparison of the small-scale field experiment with the benchmark model experiment shows the importance of general equilibrium effects, while a comparison of the epidemiological experiment with the benchmark model experiment illustrates the significance of behavioral responses. Second, they give insights into the extent to which actual small-scale field experiments and epidemiological studies might (or might not) generate reliable policy advice.

5.1. Male Circumcision and HIV

A policy intervention that has received significant recent attention is male circumcision. UNAIDS lists circumcision as one of five prevention pillars in their 2016 report (UNAIDS (2016)). Similarly, the World Health Organization has been promoting voluntary male circumcision as a prevention tool (World Health Organization (2016)).

To what extent does cross-country variation in circumcision explain the observed differences in HIV prevalence and incidence rates across countries? While circumcision has been advocated recently as a medical policy, it has not yet seen large-scale implementation. Thus, existing cross-country differences in circumcision rates are unrelated to HIV and are due instead to cultural reasons. Data from 32 Sub-Saharan African countries are used to explore the empirical relationship between HIV and circumcision.²¹ Table IV reports some regression analysis, controlling for various potentially confounding factors such as GDP, ART, religion, and the price of condoms.

Now contemplate performing the analogous exercise in the model. In other words, vary the fraction of men who are circumcised in the model and compute the implied equilibrium HIV prevalence and incidence rates. Start with prevalence. There is an almost linear relationship with a slope of -0.05 . In other words, for each 10 percentage point increase in circumcision, the HIV prevalence rate declines by about half a percent. This is quite similar in magnitude to the coefficient from the regressions. Controlling for observables, regression (4) in Table IV shows a coefficient of -0.064 . Since the prevalence rate is a stock variable, another way is to compare the incidence of HIV. In the model, the relationship between HIV incidence and circumcision rates yields a coefficient of -6.389 . Looking at regressions (5) to (8) in Table IV, the coefficients estimated in the data are close to the model counterpart. Thus, the model quite closely replicates the cross-country

TABLE IV
HIV AND CIRCUMCISION ACROSS COUNTRIES—REGRESSIONS^a

	Dep. Variable: HIV Prevalence Rate				Dep. Variable: HIV Incidence Rate			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Circumcision	-0.1122 (0.001)	-0.0765 (0.028)	-0.0796 (0.027)	-0.064 (0.126)	-9.840 (0.015)	-4.972 (0.238)	-6.191 (0.150)	-7.339 (0.125)
Log GDP p.c.	0.0314 (0.001)	0.0293 (0.002)	0.0288 (0.002)	0.0296 (0.008)	3.87 (0.004)	3.73 (0.003)	3.43 (0.006)	2.459 (0.040)
ART	0.0816 (0.126)	0.104 (0.049)	0.105 (0.051)	0.098 (0.134)	5.63 (0.396)	8.71 (0.175)	9.05 (0.155)	6.266 (0.398)
Syphilis	0.0025 (0.380)	0.0029 (0.316)	0.003 (0.286)	0.0045 (0.334)	0.359 (0.322)	0.42 (0.234)	0.526 (0.146)	0.711 (0.187)
Muslim		-0.002 (0.491)	-0.00056 (0.461)	-0.0012 (0.181)		-0.026 (0.529)	-0.128 (0.161)	-0.207 (0.081)
Christian			-0.00039 (0.618)	-0.00065 (0.411)			-0.121 (0.207)	-0.171 (0.069)
Condom price				-0.268 (0.056)				-17.5 (0.249)
R^2	0.72	0.73	0.74	0.79	0.61	0.65	0.67	0.71
N	32	31	31	23	30	29	29	22

^a p -values are in parentheses.

²¹ See Appendix B for details on these data.

TABLE V
CIRCUMCISION

	Benchmark (20% Circ.)			100% Circ.	
	All	Males		G.E.	Epidem.
		Not Circ.	Circ.		
HIV prevalence, %	10.3			5.6	4.3
Males	8.6	8.75	8.0	3.8	4.1
Females	12.1			7.6	4.6
Sex that is casual (males), %	16	14	22	29	–
Condom use for casual sex (males), %	33	35	27	22	–
Single men, %	50	49	53	59	–
Casual sex in past year, single men, %	21	19	28	31	–
Price—protected	–6.5	–	–	53	–
Price—unprotected	278	–	–	309	–
Price—long term	125	–	–	161	–

relationship between HIV and circumcision. Since cross-country data were not used in the calibration, this can be seen as an external validation of the model.

So is male circumcision a promising policy that should be promoted? Probably yes.²² Table V shows that scaling circumcision up from the current 20% to 100% would cut the HIV rate by almost half—from 10.3 to 5.6%. This works despite significant behavioral adjustments by men. The model suggests that men would engage in more risky behavior along all dimensions: less marriage, decreased condom use, and more sex.²³ The equilibrium model also shows a sizable positive effect for women: The prevalence rate among women falls from 12.1 to 7.6%. This is worth emphasizing, as the effect on women is theoretically unclear. Women do not directly benefit from circumcision as the male-to-female transmission rate does not change. Moreover, the increased risky behavior of their partners would, all else equal, lead to more HIV among women. Yet, the model shows that women benefit through the equilibrium effects. Thus, the lower female-to-male transmission rate also leads to a decline in the female prevalence rate.

The conclusion that circumcision is an effective policy is in line with findings in field experiments. Padian et al. (2010) compared the effects of 36 randomized controlled trials (RCTs) in the context of HIV. Only six RCTs delivered definitive results on HIV—three of which were circumcision RCTs. But what about the size of the effect? In the benchmark model, the behavior of circumcised men corresponds to the treated group in a small field experiment, while non-circumcised men constitute the control group. So, in Table V, compare the statistics for non-circumcised with circumcised men in the benchmark model. Clearly, in line with RCTs, the treated men in the model have a lower HIV rate. However, the effect is small (8 versus 8.75%). This is due to the missing equilibrium effects and behavioral responses among the circumcised that crowd out some of the gains from the intervention. The simulation suggests that circumcised men have 50% more casual sex, use condoms about 25% less, and are slightly more likely to be unmarried. The result that circumcised men engage in more risky behavior is in line with the empirical findings

²²This ignores any potential negative physiological and psychological side effects. In a Malawian field experiment, Chinkhumba, Godlonton, and Thornton (2014) found important barriers to male circumcision based on cultural norms and fear of pain.

²³This is in line with cross-sectional studies showing that circumcised men engage in more risky sexual behavior along several dimensions, such as Bailey, Neema, and Othieno (1999).

from RCTs. Bailey et al. (2007) found that unprotected sex fell much less for the newly circumcised relative to the control group.²⁴ Similarly, Auvert et al. (2005) found a statistically significant increase in the number of partners for the circumcised men. In a unique long-run follow-up after a circumcision RCT with teenagers, Kim, Kim, Pop-Eleches, and Jung (2018) found evidence of risk compensation four years after the intervention. Those treated (i.e., being offered free circumcision) have a higher incidence of other STDs and are less likely to believe that they should use a condom during a casual sexual encounter.

A simple extrapolation from such a field experiment might suggest that circumcision is useful, but with expected small effects. Yet, the experiments show, when circumcising the entire male population, powerful equilibrium effects kick in. The male HIV rate falls by more than half (from 8.6 to 3.8%). This is despite even larger behavioral responses in equilibrium. Because female-to-male transmission rates are lower, fewer men get infected, given sexual behavior. Since fewer men are infected, fewer women get infected, given sexual behavior. And so on.

The equilibrium effect would of course also be present in epidemiological studies. The epidemiological model version is reported in the last column of the table. Not surprisingly, the epidemiological experiment exaggerates the beneficial impact of the policy, as it completely ignores the additional risk-taking behavior. This potential overstatement of effects is a concern of the epidemiological literature. Williams et al. (2006) reported large positive effects of circumcision in Sub-Saharan Africa, but added a cautionary note remarking that increases in risk-taking may reduce some of the benefits of this policy. The study concluded by asserting that population-level studies of male circumcision are needed, a call echoed by De Walque (2012). Since population-level studies are expensive and difficult to implement, the equilibrium model offers a potentially promising alternative.

5.2. Was ART Successful in Reducing HIV?

A second policy that has gained widespread attention is the introduction of ART. While initially invented as a treatment for sick people, it is now also believed to have a preventive component. ART lowers the viral load and makes the person taking the drugs feel healthier, live longer, and be less likely to pass the virus on. Since the existence of ART makes life with HIV more tolerable, this may lead healthy people to engage in riskier behavior. Moreover, since HIV-infected people on ART live longer, they have more time to pass the virus on. Thus, the net effect of ART on HIV prevalence is not obvious. Previous research, based on different methodologies, finds a wide range of effects. The predicted long-run effects range from the complete eradication of HIV to an increase in the prevalence rate. Much of the medical literature seems convinced of the effectiveness of the policy (e.g., Cohen et al. (2011)). Yet, for example, Lakdawalla, Sood, and Goldman (2006) showed empirically that ART has led to an increase in HIV in the United States. Wilson (2012) provided a survey of the empirical studies and warned that widespread enthusiasm for ART as prevention may be misguided, since expected outcomes are currently mostly based on clinical trials alone that are not informative for what would happen if the entire population was treated.

In Malawi, ART was introduced in 2005. Figure 1(a) shows that from 2004 to 2014, the fraction of infected people on ART increased from essentially zero to 50%. At the same

²⁴Note that most circumcision RCTs were combined with counseling sessions for both the treatment and control group. Since this increased awareness likely led to more cautious sexual behavior, the relevant thing to look at is the behavioral change in the treatment relative to the control group, rather than the absolute change. Section 6 discusses the impact of increased awareness about the HIV transmission mechanism.

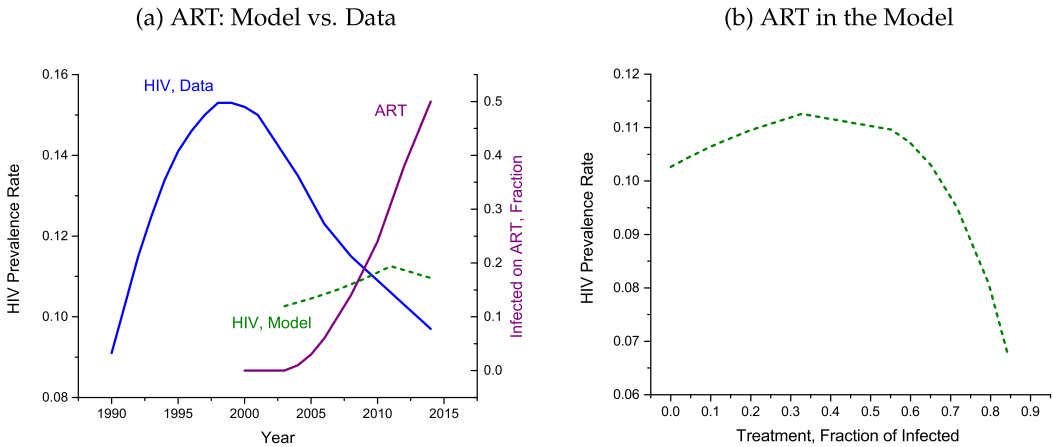


FIGURE 1.—ART in Malawi.

time, HIV was declining.²⁵ The Malawian government seems to have concluded that the decline was due to the successful ART scale-up.²⁶ However, simple inspection of the two time series in Figure 1(a) makes it clear that ART cannot be the whole story. The HIV decline started in 2000, five years prior to the introduction of ART. Anticipation effects would go in the other direction—as anticipating ART should make people behave in a riskier fashion without experiencing the benefits of the lower transmission risk. It is of course possible that the introduction of ART did contribute to the HIV decline in the later years. The model can be used to assess (and quantify) this hypothesis.

ART is modeled as a decline in the (out-going) transmission rate. Assume a reduction in infectivity by two-thirds, which is within the range of empirical estimates.²⁷ Specifically, the quarterly transmission risk for unprotected sex declines from 0.21 to 0.07 for females having sex with someone on ART, and from 0.12 to 0.04 for males.²⁸ The reduced mortality (and accordingly increased quality of life) is modeled as a longer life without symptoms. This means that infected people on ART live longer, but also enjoy a better life after infection (relative to those not on drugs). Specifically, reduce α , the probability of symptoms breaking out, from 0.025 to 0.0125. Since symptoms are quickly followed by death, this means that mortality is essentially reduced by 50%, which is in line with the evidence.²⁹

²⁵The time-series data for the HIV prevalence rate come from UNAIDS and are estimated largely based on information collected at antenatal clinics. The UNAIDS 2004 HIV rate for Malawi is 13.5%, which is substantially higher than the prevalence rate in the DHS, reported in Table III. Since the DHS is a nationally representative survey, it is the more reliable source. However, UNAIDS provides the only consistent time-series data.

²⁶“Malawi’s rapid and successful Antiretroviral Therapy scale-up from 2004 to 2014 has critically influenced the trajectory of the HIV epidemic . . .,” see p. 2 in the Malawi AIDS Response Progress Report 2015, Government of Malawi.

²⁷While the estimates cover a wide range, the number used is close to Porco, Martin, Page-Shafer, Cheng, Charlebois, and Grant (2004), who found a decline of 60%.

²⁸This corresponds to an increase in γ from 0.787 to 0.929 for females and from 0.879 to 0.96 for males. The numbers for protected sex and for circumcised men are adjusted accordingly when having sex with a treated individual.

²⁹One study specifically pertaining to Malawi is Lowrance et al. (2007). Friedman (2018) also fed in a 50% reduction in mortality for treated individuals in her simulation study.

TABLE VI
ART

	Benchmark	$q = 0.01$	$q = 0.075$		
		G. E.	G. E.	Small Field	Epidem.
Fraction on ART, %	none	33	80	80	80
HIV prevalence, %	10.3	11.3	8.1	16.7	2.5
Casual sex, % (of all)	15.7	20.5	42	22	–
Casual sex with condom, %	33	27	24	30	–
Singles who have casual sex, %	54	63	84	62	–
Single men, %	50	53	67	54	–
Single women, %	46	49	65	51	–
Price—protected	–6.5	7.8	25	–	–
Price—unprotected	279	285	259	–	–
Price—long term	125	126	100	–	–

In the model, infected people are treated randomly. The probability of being selected for ART is q . Since treatment is an absorbing state, $q\%$ newly treated each period cumulates to a substantially higher percentage of the infected on ART. Envisage the following exercise: increase q such that the equilibrium fraction of infected on ART goes up in line with the data. The model then gives, at various levels of treatment, the long-term HIV rate. Since the model compares steady states (i.e., ignores transitional dynamics), this exercise will give an upper bound on the fraction of the HIV decline due to ART.

The dashed line in Figure 1(a) displays the simulation results. The surprising answer is that hardly any of the HIV decline can be attributed to the introduction of ART. In other words, the negative effects (increased risk-taking, longer time to infect others) of the policy dominate the positive effects (from the lower transmission rates making sex safer). Now, should one conclude from this that ART is not an effective policy to curb HIV? Probably not. The experiments show that the relative importance of the positive versus negative effects changes with the ART rate. Figure 1(b) shows that higher levels of treatment are actually quite effective. Once more than 50% of the infected are treated, the preventive effect dominates. Thus, going forward, a further expansion of ART appears promising.³⁰

To better understand the nonmonotonicity, also consult Table VI. When a third of the infected are treated ($q = 0.01$), people realize they may have a reasonable chance of getting treatment if they contract HIV, and accordingly take fewer precautions. Condom use goes down by six percentage points, casual sex rises from 54% to 63%, and more people become single. When 80% of the infected are treated ($q = 0.075$), the behavioral adjustments are even more dramatic. The increase in risk-taking is in line with the empirical findings. Most evidence to date comes from developed countries.³¹ However, there is some limited evidence from African countries as well. Identifying the behavioral response to ART is notoriously difficult. Clearly, a randomized controlled trial to assess the effect on the uninfected is difficult to conceive.³² De Walque, Kazianga, and Over (2012) found a

³⁰The point that ART may be beneficial only once a large enough fraction of the infected is treated is also emphasized in Friedman (2018), who simulated a one-gender model with only one margin of risky behavior.

³¹For example, see Crepez, Hart, and Marks (2004) for a meta-analysis of 25 studies in the United States, Europe, and Australia.

³²In the synthetic field experiment, it is easy to “treat” some uninfected by changing their q , that is, the probability that they would get treated with ART should they get sick. Implementing this in reality would

large increase in self-reported risky sexual activity in response to ART in Mozambique, in terms of both more casual sex and less condom use. Similarly, Cohen et al. (2009) found increased risk-taking behavior in men who have stronger beliefs in the effectiveness of ART compared to those with more skeptical beliefs. Friedman (2018) used a difference-in-difference strategy based on proximity to an ART provider in Kenya and discovered an increase in self-reported risky behavior of about 40%, in terms of both the incidence of casual sex and condom use. When using a biomarker (pregnancy), the effect is even larger, at about 80%.

Behavioral adjustments alone would lead to a large increase in the HIV rate, as the synthetic field experiment (which predicts an increase in the HIV rate to 16.7%) shows. However, these effects are mitigated in equilibrium by the fact that the treated interact with everyone else. In the general equilibrium experiment where 80% of the population is treated, sex in general is safer, which leads to a lower aggregate prevalence rate. In the field experiment, people realize that once they get sick they have an 80% chance of getting treated eventually (7.5% per period) but they still have sex with the general population; in essence, none of their potential partners is treated. So their behavioral adjustments lead to a very high HIV rate of 16.7%, with no offsetting effect created by a lower chance of catching the virus. By contrast, by completely ignoring the behavioral adjustments in the epidemiological experiment, the opposite mistake occurs. The epidemiological version predicts a massive HIV decline to a prevalence rate of only 2.5%. In other words, the behavioral and equilibrium effects go in opposite directions, and which of them dominates depends on the fraction of people treated. A too-cautious attempt may backfire.

5.3. Better Condoms

Suppose one could design more pleasurable condoms (or perhaps raise the psychic pleasure of sex with a condom by reducing stigmatization through publicity campaigns).³³ Would this be desirable? The effect of more pleasurable condoms is displayed in Figure 2,

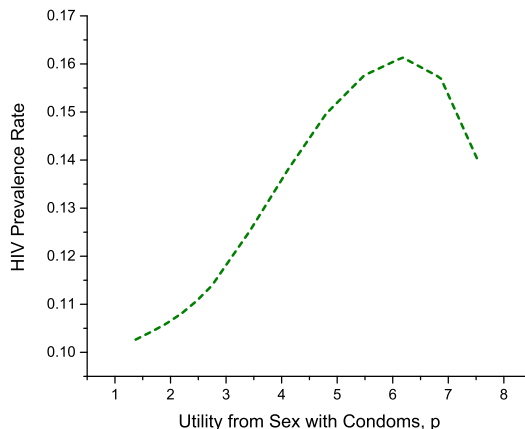


FIGURE 2.—Better condoms.

require essentially an information treatment, where some people are informed about increased access to ART while others are not.

³³While this might seem somewhat far-fetched, note that UNAIDS lists exactly such a policy in their recent report: “Develop new approaches to increase condom use and to enhance the positive perception of condoms

TABLE VII
BETTER CONDOMS

	Benchmark	G. E.	Small Field	G. E.	Small Field
p	1.4	5.5 (Better)		7.5 (Better Still)	
p/u	0.18	0.70		0.97	
HIV prevalence, %	10.3	15.8	15.6	14.0	18.8
Casual sex, % (of all)	15.7	34	31	32	41
Casual sex with condom, %	33.0	59	57	62	61
Singles who have casual sex, %	54.0	66	73	73	78
Single men, %	50	64	60	62	70
Single women, %	46	60	51	58	56
Price—protected	−6.5	246	−	260	−
Price—unprotected	279	264	−	244	−
Price—long term	125	134	−	138	−

where, starting from the benchmark, utility from sex with condoms is increased until it reaches the same period utility as unprotected sex. It turns out that the HIV rate displays a nonmonotonic pattern when increasing pleasure from condoms, p . The reason that increasing the utility from protected sex does not always lead to a lower 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.

Table VII gives the example of quadrupling condom pleasure from 1.4 to 5.5. In response, condom use increases tremendously, almost doubling from 33% to 59%. However, the percentage of singles also substantially increases (from 48% to 62%). Moreover, the percentage of singles who engage in short-term sex goes up from 54% to 66%. These two forces (safer sex versus more sex) push the prevalence rate in opposite directions. In the example of quadrupling pleasure, the latter force dominates, so that the overall HIV rate goes up by about 60% (from 10.3% to 15.8%). The HIV rate declines, though, when the pleasure from condoms is pushed up further from 5.5 to 7.5, at which point protected and unprotected sex give almost equal utility. This experiment highlights the potential of some policies to backfire and actually *increase* the overall prevalence rate. These effects can be quantitatively quite important: in the experiment, the HIV rate goes up by 40% as the utility gap between protected and unprotected sex disappears.

Implementing this particular policy as a field experiment gives surprising results. Depending on the exact increase in pleasure, the synthetic field experiment effects are larger or smaller than the general equilibrium effects—see Table VII. Consider the experiment where the pleasure from sex with condoms is increased from 1.4 to 5.5. The increase in the aggregate HIV rate in the general equilibrium experiment is slightly larger than the increase in the HIV rate among the treated in the field experiment (15.8% vs. 15.6%). The percentage of singles having casual sex is higher in the field experiment than in the general equilibrium one (73% vs. 66%). This transpires because the hike in HIV in the general equilibrium model dampens risky sexual behavior. This also results in a slightly higher percentage of casual sex with condoms in the general equilibrium model (59% vs. 57%). Also, there are more singles in the general equilibrium model, because at the higher HIV rate the benefit of unprotected sex within a marriage has dropped relative to

among the various populations in need,” p. 30 in UNAIDS (2016). Concerns about the acceptability of condoms have long been discussed in the literature; see Section 2.

short-term protected sex. Now, increase the pleasure from sex with condoms even further to 7.5. Again, the fraction of singles having casual sex is higher in the field experiment (78% vs. 73%). Since there is a larger percentage of sex with condoms, the HIV rate declines in the general equilibrium model, unlike in the field experiment, where the HIV rate rises. An individual's odds of catching HIV rise significantly in the field experiment because they are having so much more casual sex, both protected and unprotected. The drop in the aggregate HIV rate increases the benefit of marriage relative to short-term protected sex, so the number of singles drops in the general equilibrium experiment, unlike in the field experiment where it rises. Thus, the reaction in the field experiment can be amplified or mitigated relative to the general equilibrium one.

Finally, an epidemiological experiment would predict no effect of the condom policy. This is by construction, as epidemiological experiments assume no change in behavior, but without behavioral change, the increased condom pleasure by itself would not do anything. This is worth noting, since in the case of the medical policies, the lack of behavioral adjustments leads to an exaggeration of effects in the epidemiological experiments. Naturally, the opposite is the case in any experiment where the behavioral adjustments are needed for a policy to work—as in the case of increasing condom pleasure, where the hope is that more people would use them.³⁴

One potential concern about this exercise is the substantial increase in the number of singles, which might be at odds with intuition. People get married for many reasons, including the desire to have children, and increased condom pleasure might not change the determination for marriage for those to whom having children is very important. Additionally, fertility in Malawi is very high by Western standards. Modeling fertility explicitly goes beyond the scope of this paper, but an initial step in this direction might be taken by adding a value f for fertility in marriage that differs by type. Heterogeneity in this fertility benefit implies that those with strong preferences for children in marriage will not be enticed out of the marriage market, potentially reducing the responsiveness of marriage with respect to shifts in condom utility. This means that the per-period utility of marriage is now $u + l + f$. Otherwise the model is unchanged.

As a first attempt to gauge the promise of this channel, consider the following parameterization: Leave the number of types unchanged from the basic model to avoid computational complexity, but assume that those types with a high discount factor also have a higher value from fertility (i.e., have a higher f). Under the assumption that f has a quadratic form in β , the three parameters characterizing its distribution are chosen to keep the basic calibration targets roughly unchanged relative to the benchmark model. See Appendix D.2 for more details.

Table VIII shows how close this extended version is to the original benchmark. It also shows how this new economy reacts to increased pleasure with condoms. In line with intuition, when people marry partly to have children, their marriage decision is much less responsive to the introduction of more pleasurable condoms. Yet, interestingly, improved condoms continue to increase the HIV rate. This is driven by singles substantially increasing their sexual activity. Overall and as expected, however, the increase in the HIV rate is smaller (3.6 percentage points relative to 5.5 percentage points in the benchmark calibration).

³⁴The same logic would be present for policies aimed to work through the marriage market, as laid out in Greenwood et al. (2017).

TABLE VIII
CONDOMS AND FERTILITY

p	Benchmark	Better Condoms	Benchmark (w./Fertility)	Better Condoms (w./Fertility)
	1.4	5.5	1.4	5.5
HIV prevalence, %	10.3	15.8	10.0	13.6
Casual sex, % (of all)	15.7	34	18.5	26.9
Casual sex with condom, %	33.0	59	28.9	53.7
Singles who have casual sex, %	54.0	66	65.5	75.4
Single men, %	50	64	49.4	53.4
Single women, %	46	60	44.8	48.2

5.4. Treating Other Sexually Transmitted Diseases

The treatment of other sexually transmitted diseases (STDs) has been advocated in the fight against HIV/AIDS, based on the idea that the presence of other STDs makes a person more susceptible to contracting HIV. Thus, treating other STDs will decrease the transmission risk, for both men and women. For example, Grosskurth et al. (1995) found that improved STD treatment reduced HIV incidence by about 40% in rural Tanzania. Oster (2005) compared data from African countries to the United States and reached the conclusion that treating other STDs would be an effective policy. This conclusion is based on an epidemiological simulation, which shows that differences in transmission rates (due to the existence of other STDs) can explain much of the difference in HIV rates between the United States and Sub-Saharan Africa.

Table IX shows the simulation results for this policy in the model (see the second column of results). For this policy, the probability of infection is multiplied by a scaling factor $\lambda \in [0, 1]$, such that the new transmission rate is $\lambda(1 - \gamma)$ for all types of sex, genders, and circumcision types. As the transmission risk for both men and women declines by 15% ($\lambda = 0.85$), HIV prevalence decreases by almost one percentage point from 10.3% to 9.5%. This decrease in HIV prevalence masks the fact that, when faced with better transmission odds when having sex, people engage in riskier behavior. The fraction of sex that is casual increases from 15.7% to 18.7%. This is because there are more singles now, and singles have more sex. Moreover, among the singles having sex, condom usage falls from 33% to 28%. Despite the increase in risky behavior, the policy works in the sense that HIV does fall overall.

Yet the behavioral changes have non-trivial effects, which can be seen as follows. Compare the results from the general equilibrium experiment with the epidemiological version of the experiment in the third column of results of Table IX; see also Figure 3. In the epidemiological experiment, the decline in HIV prevalence is much larger, dropping to 7%. The reason for this difference is exactly the lack of behavioral changes described above. Thus, simulations based on epidemiological experiments may significantly overstate the beneficial effects of STD treatment. This casts some doubt on Oster (2005)'s finding that STD treatment alone is able to explain much of the U.S.-SSA difference in HIV, as the study was based on the assumption of constant sexual behavior across countries.³⁵

³⁵Oster (2005) argued that sexual behavior in the data is remarkably similar in the United States and Sub-Saharan Africa. However, her Table II seems to indicate that behavior is somewhat more risky in the United States, which would be in line with the model, where people in the country with the lower transmission risk engage in more risky behavior.

TABLE IX
TREATING OTHER STDs

Scaling Factor	Benchmark 1.00	G.E. 0.85	Epidem. 0.85	Small Field 0.85
HIV prevalence, %	10.3	9.5	7.0	10.1
Males	8.6	7.9	5.9	8.5
Females	12.1	11.3	8.2	11.9
Casual sex, % (of all)	15.7	18.7	—	16.7
Casual sex with condom, %	33.0	27.6	—	31.2
Singles who have casual sex, %	54.0	60	—	56
Single men, %	50	52	—	51
Single women, %	46	47	—	46
Price—protected	−6.5	10	—	—
Price—unprotected	279	286	—	—
Price—long term	125	127	—	—

The synthetic field experiment goes in the opposite direction: it predicts only a very small change in HIV prevalence compared to the benchmark. 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, the decrease does not feed back into lower infection rates for the treated population, something that is naturally part of the general equilibrium model.

The lack of a substantial HIV decline in the field experiment might actually be quite important. Note that eight of the nine studies of STD treatment for HIV prevention surveyed by Padian et al. (2010) delivered flat results. This seems quite puzzling, given that the theoretical effects of STD treatment are uncontroversial in the medical community. The simulations presented here highlight a novel reason for the lack of finding a large effect, namely, the missing equilibrium effects in randomized field experiments. Thus, treating STDs might actually be a promising policy measure, even though it is difficult to detect positive results in field experiments.

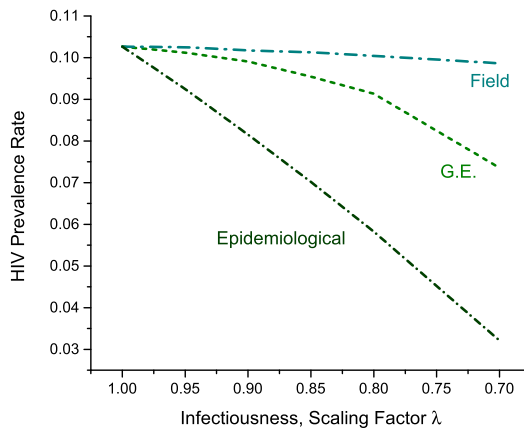


FIGURE 3.—Treating other STDs.

6. THE DIFFUSION OF BETTER INFORMATION

HIV prevalence in Malawi has fallen over the last two decades, from a peak of almost 17% in 1999 to less than 10% in 2016. Section 5 suggested that the introduction of ART was not the dominant driver of the falling HIV rate. So what explains this success story? From the policy experiments, male circumcision seems to work, yet there was no substantial change in circumcision rates in Malawi through 2010. Both the promotion of condoms and treatment of other STDs have been part of the government's plan to fight HIV (Government of Malawi (2003)), but it is unclear how widely such policies were implemented. Moreover, the policy experiments show that the condom policy may backfire and the treatment of other STDs may at most have a modest effect. Thus, both policies seem unlikely to be the main causes behind the HIV decline. This leaves a bit of a puzzle.

To what extent could better information have caused the decline in HIV? Clearly, if people are not aware of how HIV is transmitted, or how they can protect themselves, they will engage in more risky activities. As information improves, behavior becomes less risky, which lowers the HIV prevalence rate.³⁶ Indeed, evidence from the DHS shows that awareness of the HIV transmission mechanisms massively increased between 1992 and 2004 and has roughly stabilized since; see Figure 4.³⁷ Specifically, in 1992, only a quarter of the population correctly identified that abstinence reduces HIV risk, while by 2004, more than three quarters of the population did so. Similarly, in 1992, only about 10% knew that condom use reduces risk, while by 2004, 60% did. In 1992, approximately 60% erroneously believed that HIV can be transmitted through mosquito bites, which fell to about 20% by 2004. It is unclear to what extent improved awareness can be attributed to official information campaigns or whether it is the natural diffusion of information follow-

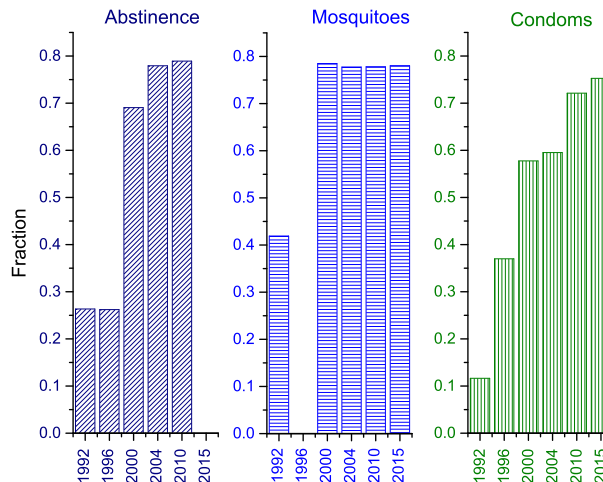


FIGURE 4.—HIV awareness in Malawi, 1992–2015, DHS data.

³⁶For example, Dupas (2011) found in a randomized field experiment that Kenyan teenage girls who are given information about the HIV status of different groups of men respond by shifting their sexual behavior to the lower-risk groups.

³⁷Figure 4 is based on six waves of the DHS. It is similar to Figure 3.3 in Fedor (2014), but includes both men and women and additional information for 2015. The two empty bars are due to the following: In 1996, the question on mosquitoes was not asked in a consistent way, and in 2015, no information related to abstinence was collected.

ing the discovery of a new disease.³⁸ Without taking a stand on the underlying reason for the better information, this section asks whether the improvement in information about transmission mechanisms can explain the drop in HIV in recent years in Malawi. This is done by allowing a fraction of the population in the model to be uninformed about HIV.

To model the presence of uninformed individuals, introduce a new variable $i \in \{0, 1\}$ in the permanent-type vector, x , characterizing each individual, with the convention that $i = 1$ stands for an informed individual and $i = 0$ for an uninformed one. Informed individuals know the true structure of transmission. Uninformed people do not. The survey evidence indicates the presence of individuals who are not aware that either condoms or abstinence can protect them from HIV infection. Therefore, assume that uninformed singles believe that the odds of contracting HIV are the same as in the short-term unprotected market, regardless of their sexual behavior. That is, individuals who either enter the short-term market for protected sex or who remain abstinent think that they are just as likely to catch HIV as someone in the short-term unprotected market. (In the absence of any other anchor for their beliefs about unprotected sex, for simplicity assume that they assign correct beliefs to unprotected sex.) Note that all sex in the long-term market is unprotected so nothing changes here. Finally, even though the survey did not directly question individuals about circumcision, it is unlikely that uninformed individuals were aware of its protective effects. So assume that uninformed individuals do not assign protective power to circumcision.

The adjustments to the model required to incorporate the presence of uninformed people are relatively modest. The value functions for uninformed individuals are similar to those for informed ones, with just a few changes, which imply different behaviors between them. Specifically, for a healthy uninformed old individual, the values of abstinence and short-term sex in equations (1) and (3) are adjusted by replacing the infection probabilities with those in the short-term unprotected market. Additionally, set $\chi(c) = 1$ for all c , so that individuals do not take circumcision into account. The modifications to the analogs for young healthy individuals are identical. For uninformed old individuals in the long-term market, equation (7) is adjusted so that they do not take the protective effects of circumcision into account for themselves or for their circumcised partner. Again, the same is done for young individuals in the long-term market. Given the decision rules for uninformed people, the determination of the equilibrium incidence and prevalence rates is governed by the correct infection probabilities. The exact equations are presented in Appendix D.1.

The model with uninformed people can be used to explore to what extent changes in information may have been responsible for the decline in HIV over time. Focus on two years: 1996 and (as before) 2004. Since awareness was relatively high and stable from 2004 onward, the full-information model is a good approximation of reality for that year. (Of course, as Figure 4 shows, even in 2004 there are still some uninformed people.) For 1996, it will be assumed that 60% of the population is uninformed, in line with the data.³⁹ Table X gives the results and compares them to the data for the same two years. The data moments are from two waves of the DHS (1996 and 2004) except for the HIV incidence

³⁸See Fedor (2014) for details on changes in HIV knowledge in Malawi and government policies that may have contributed to increased awareness.

³⁹As Figure 4 shows, in 1996, 63% of the population did not know that a condom is effective, 74% did not know that abstinence helps, and (in 1992) 58% erroneously believed that HIV could be transmitted through mosquitoes.

TABLE X
IMPROVED INFORMATION—MODEL VERSUS DATA

	Data			Model		
	1996	2004	Δ in p.p.	1996	2004	Δ in p.p.
HIV prevalence, %	14.6	13.5	-1.1	14.4	10.3	-4.1
HIV incidence per 1000 healthy	20.4	11.1	-9.3	17.3	11.7	-5.6
Sex that is casual, %	24	18	-6	20	16	-4
Condom use in casual sex, %	29	39	10	28	33	5
Fraction of singles, %	31	33	2	47	48	1
Singles who had casual sex, %	60	47	-13	67	54	-13

and prevalence numbers.⁴⁰ Since the decline in HIV is not targeted in the benchmark calibration, this gives an additional opportunity to examine the performance of the model.⁴¹

The table shows that accounting for the lack of awareness about HIV transmission mechanisms indeed raises the overall prevalence and incidence rates quite a bit. The implied decline in the HIV rate is in fact larger than the one observed in the data. This is not surprising since the model results are based on a steady-state comparison, while in reality the transitional dynamics would likely take some time to work out. On this, note that the prevalence rate simply cannot fall very quickly in the real world because it is a stock variable. It takes time for the reduction in new infections (incidence) to cumulate through the economy. Incidence rates actually fell very quickly over this time period, from 20.4 new infections annually per 1000 healthy people in 1996 to 11.1 by 2004. The model implies that a reduction in the fraction of the population that is uninformed from 60% to zero will decrease incidence rates by a similar order of magnitude from 17.3 to 11.7 per 1000 healthy people. Therefore, improved information may well have played a key role in reducing HIV in Malawi.

The fall in incidence rates is due to safer sexual choices. The adjustments in terms of sexual behavior are also remarkably close to the data. In the model, the fraction of casual sex falls by 4 percentage points (p.p.), condom use increases by 5 p.p., the fraction of singles increases by 1 p.p., and among them, 13 p.p. fewer singles have casual sex. The data show a very similar trend toward less risky behavior. From 1996 to 2004, the fraction of casual sex fell by 6 p.p., condom use increased by 10 p.p., the fraction of singles rose by 2 p.p., and among them, 13 p.p. fewer singles had casual sex.

Finally, given that in 2004 not all Malawians were perfectly informed, one may question whether the assumption of perfect awareness in the benchmark model biases the policy experiments in Section 5. Put differently, one may want to know what the effect of policies would have been in a world with more uninformed people. This question is addressed by implementing the same policy experiments for the 1996 model (i.e., the one with 60% uninformed people). This is reported in Table XI in Appendix D.1. The table shows that results are qualitatively similar to those in the full-information model. At the same time, all policies are quantitatively somewhat more effective in the model with uninformed people. For example, circumcising all men would lower the HIV rate by 68%, relative to only 46% in the baseline model. In the case of ART, the positive effect is seen earlier;

⁴⁰The HIV incidence and prevalence numbers are from UNAIDS; see also footnote 25. This departure from Table II is due to the fact that the 1996 DHS did not collect biomarker information and hence no HIV prevalence rate can be calculated. Further, none of the DHS has incidence information.

⁴¹In fact, this section was motivated by a suggestion from a referee. The calibration predates the suggestion.

that is, treating only a third of the population ($q = 0.01$) leads to a decline in the HIV rate. The higher effectiveness of the policies is not that surprising, though. Recall that in the model with perfect information, people dampen the effectiveness of the policies by responding with riskier behavior. Uninformed people do not react as much because they do not understand the true trade-offs between different sexual behaviors. Thus, the higher the fraction of uninformed people, the better all policies work in reducing HIV.

7. CONCLUSIONS

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 men and women rationally search for the type of sexual activity they desire to engage in, taking into consideration the risks of this activity. Some people select stable long-term relationships; others may choose more fleeting ones. Condoms may or may not be used in 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 calibrated to capture some of the salient features of the Malawian HIV/AIDS epidemic. The framework can match both targeted and non-targeted statistics regarding sexual behavior and HIV/AIDS in Malawi, as well as some cross-country data. The benchmark simulation is then used to undertake some policy interventions. The quantitative results suggest that policy analysis of HIV/AIDS interventions is complicated; in particular, some policies may backfire and actually increase HIV. The simulations also rationalize some puzzling results in previous works.

Specifically, it is shown that ART is unlikely to have been the main driver behind the decline in HIV over time. ART can work well, but only if a large fraction of the population is treated. Second, male circumcision seems a useful policy with effects that are likely much larger than one would expect based on field experiments alone. Third, better condoms can potentially backfire and lead to more HIV. Fourth, the beneficial effects of treating other STDs are much smaller than suggested by some epidemiological studies. At the same time, the analysis resolves a puzzle regarding the lack of positive effects in field experiments, due to their difficulty in capturing the salubrious spillover effects on partners that operate to reduce the equilibrium HIV prevalence rate. Finally, the research also explores the likelihood that the Malawian populace was not fully knowledgeable in the early stages of the epidemic about how HIV is transmitted. As a result, people who were uninformed did not move into safer sexual activity. It is shown that as information spread about the HIV transmission mechanism, the rate of HIV fell. Thus, the diffusion of better information appears to be an important driver of the HIV decline in Malawi over time.

The framework developed here contains several dimensions of heterogeneity. One can, however, think of different margins that were not explored in the current paper. For example, HIV prevalence rates in urban areas tend to be higher than in rural regions. This may have to do with different search costs in the two localities.⁴² Furthermore, the impact of migration between the rural and urban areas may be important to consider. Additionally, the theoretical model does not allow for concurrent relationships, while in reality, extramarital affairs and polygyny may matter for the spread of the virus. Such questions are left for future research.

⁴²Indeed, running a counterfactual in which search costs in the model are increased leads to a lower HIV prevalence rate.

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SUPPLEMENT TO “AN EQUILIBRIUM MODEL OF THE AFRICAN HIV/AIDS EPIDEMIC”

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APPENDIX A: TIMING OF EVENTS

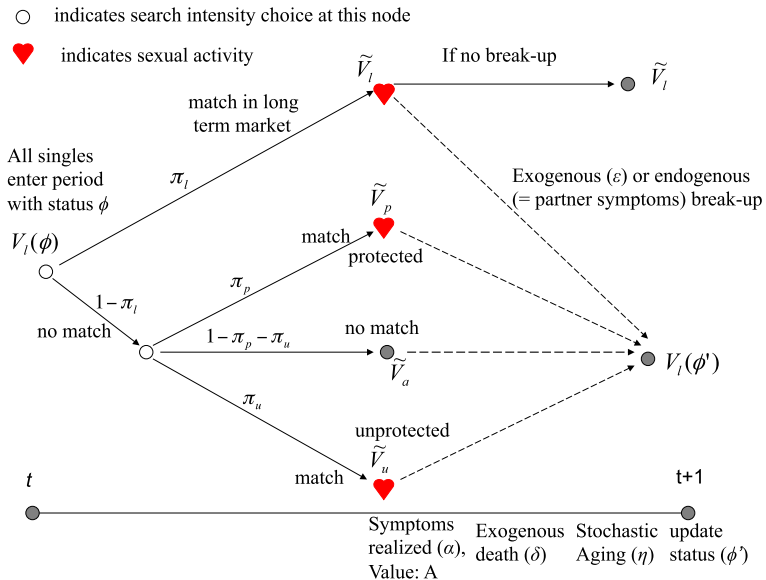


FIGURE 5.—Timing of events.

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APPENDIX B: DATA

Most of the empirical moments are based on information collected from the interviews of individuals conducted for the Malawi Demographic and Health Survey (MDHS) in 2004, carried out by the Malawi National Statistical Office. In this nationally representative survey, 11,698 women aged 15 to 49 and 3261 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 1a: HIV is defined as “Prevalence of HIV, total (% of population ages 15–49).” ART is defined as “Antiretroviral therapy coverage (% of people living with HIV).” HIV data come from UNAIDS and ART coverage is taken from the World Development Indicators.

- Figure 4 is based on the MDHS for the years 1992, 1996, 2000, 2004, 2010, and 2015. The question asked is “What can a person do (to avoid getting AIDS)?” and then various options are given. For 1996, the question on mosquitoes was not asked in a consistent way, and in 2015, no information related to whether abstinence reduces HIV risk was collected. Fractions were computed based on the entire sample (males and females); sample weights were used. The exact list of variables used is as follows. Abstinence: women: s808a (1992), qa509a (1996), v754b (2000–2010), men: mv754b (1992), qma509a (1996), mv754b (2000–2010). Condom use: women: s808c (1992), qa504 (1996), v754c (2000–2015) men: mv754c (1992), qma504 (1996), mv754c (2000–2015). Mosquitoes: women: s804g (1992), v754jp (2000–2015) men: sm504g (1992), mv754jp (2000–2015).

- Figure 6: HIV rate—men versus women, model versus 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: 2,404 men and 2,864 women). The resulting HIV rates are smoothed using a third-order polynomial.

- Figure 7: Fraction ever married—model versus data. The fraction of people who have ever been married is derived by dividing the number of people who either 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 8: Sexual behavior by age—model versus data. Singles: Those men and women 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).

Casual sex: 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). These 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 9: Deaths by HIV/AIDS by age—model versus data. The data on deaths caused by HIV/AIDS are taken from [Bowie \(2006\)](#), pp. 31–42. He reported the frac-

tion of HIV/AIDS related deaths by age groups, based on the WHO Global Burden of Disease Malawi from 2002.

- Table I: Parameters Chosen Outside the Model. All sources are described in the text.
- Table III: 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 nor cohabiting, 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 7. The [World Health Organisation \(2008\)](#) reports that 29% of all deaths in Malawi in 2004 were due to HIV/AIDS.
- Table IV: The cross-country circumcision data come from [Ahuja, Wendell, and Werker \(2009\)](#). The statistics for HIV rates come from UNAIDS, while the numbers for GDP per capita and ART coverage come from the World Bank Development Indicators. The rates for syphilis seropositivity relate to data among antenatal care attendees from the WHO Global Health Observatory. The fractions of populations of different religions are given by the Global Religious Futures Project of the Pew Research Center. Condom prices for different countries are reported in the Global Directory of Condom Social Marketing Projects and Organisations (UNAIDS).
- Table X: Incidence and prevalence numbers are taken from UNAIDS. All numbers on sexual behavior are computed from the MDHS. The 2004 numbers are identical to those in Table III and were calculated as described above. The numbers for 1996 were computed in exactly the same way using data from the MDHS 1996 instead. Model numbers are based on the model simulations.

APPENDIX C: THEORY

C.1. Value Functions for Young Individuals, $d = \iota$

The value functions for young individuals follow a similar structure as those for old individuals, namely, equations (1) to (6). The required adjustments are outlined in the main body in connection with (1).

In particular, for young abstinent individuals of health status ϕ , the analog to (1) replaces the high discount factor, β , with the low discount factor, ι , and treats continuation values as the average of the continuation with a low and a high discount factor, so that

$$\begin{aligned} \tilde{V}_a^\iota(\phi, x) = & \ln(y) + \alpha_\phi \iota A \\ & + (1 - \alpha_\phi) \iota \left\{ \frac{Q(\phi)[\eta V_i^\beta(t, x) + (1 - \eta)V_i^\iota(t, x)]}{+ [1 - Q(\phi)][\eta V_i^\beta(\phi, x) + (1 - \eta)V_i^\iota(\phi, x)]} \right\}. \end{aligned} \quad (9)$$

Similarly, for short-term sex, either for infected or treated individuals ($\phi = 0, t$), the analog to (2) is

$$\begin{aligned} \tilde{V}_s^t(\phi, x) &= \ln(y - z_s) + pI(s) + u[1 - I(s)] + \alpha_\phi \iota A \\ &+ (1 - \alpha_\phi) \iota \left\{ \frac{Q(\phi)[\eta V_l^\beta(t, x) + (1 - \eta)V_l^t(t, x)]}{+ [1 - Q(\phi)][\eta V_l^\beta(\phi, x) + (1 - \eta)V_l^t(\phi, x)]} \right\}, \end{aligned} \quad (10)$$

for $s = p, u$. For young healthy individuals ($\phi = 1$), the analog to (3) is

$$\begin{aligned} \tilde{V}_s^t(1, x) &= \ln(y - z_s) + pI(s) + u[1 - I(s)] \\ &+ \sum_{\hat{\phi}} R_s(\hat{\phi})[1 - \gamma_s(\hat{\phi})] \\ &\times \chi(c) \iota \left\{ \frac{q[\eta V_l^\beta(t, x) + (1 - \eta)V_l^t(t, x)]}{+ (1 - q)[\eta V_l^\beta(0, x) + (1 - \eta)V_l^t(0, x)]} \right\} \\ &+ \left\{ 1 - \sum_{\hat{\phi}} R_s(\hat{\phi})[1 - \gamma_s(\hat{\phi})]\chi(c) \right\} \\ &\times \iota[\eta V_l^\beta(1, x) + (1 - \eta)V_l^t(1, x)]. \end{aligned} \quad (11)$$

For long-term sex, note that the transition probabilities $Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c})$ in Appendix C.2 are not affected by the discount factor, and therefore the young individual's analog of (6) is

$$\begin{aligned} \tilde{V}_l^t(\phi, \hat{\phi}, \hat{c}, x) &= \ln(y - z_l) + u + l + \alpha_\phi \iota A \\ &+ (1 - \alpha_\phi)(1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}}) \iota \\ &\times \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c}) \left[\frac{\eta \tilde{V}_l^\beta(\phi', \hat{\phi}', x)}{+ (1 - \eta) \tilde{V}_l^t(\phi', \hat{\phi}', x)} \right] \\ &+ (1 - \alpha_\phi)[1 - (1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}})] \iota \\ &\times \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c}) \left[\frac{\eta V_l^\beta(\phi', x)}{+ (1 - \eta)V_l^t(\phi', x)} \right]. \end{aligned} \quad (12)$$

C.2. Transition Probabilities in Long-Term Relationships

The transition probabilities, $Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, c, \hat{c})$, from the situation where a relationship is currently characterized by the quadruple $(\phi, \hat{\phi}, c, \hat{c})$ to one where the couple's health status next period is $(\phi', \hat{\phi}')$, are now presented. Start with the situation where the person is currently healthy ($\phi = 1$) but his partner is infected ($\hat{\phi} \in \{0, t\}$). The following

lists all possible cases for this situation:

$$\begin{aligned}
Y(1, t|1, \widehat{\phi}, c, \widehat{c}) &= \{1 - [1 - \gamma_u(\widehat{\phi})]\chi(c)\}Q(\widehat{\phi}); \\
Y(1, 0|1, \widehat{\phi}, c, \widehat{c}) &= \{1 - [1 - \gamma_u(\widehat{\phi})]\chi(c)\}[1 - Q(\widehat{\phi})]; \\
Y(0, t|1, \widehat{\phi}, c, \widehat{c}) &= [1 - \gamma_u(\widehat{\phi})]\chi(c)(1 - q)Q(\widehat{\phi}); \\
Y(0, 0|1, \widehat{\phi}, c, \widehat{c}) &= [1 - \gamma_u(\widehat{\phi})]\chi(c)(1 - q)[1 - Q(\widehat{\phi})]; \\
Y(t, t|1, \widehat{\phi}, c, \widehat{c}) &= [1 - \gamma_u(\widehat{\phi})]\chi(c)qQ(\widehat{\phi}); \\
Y(t, 0|1, \widehat{\phi}, c, \widehat{c}) &= [1 - \gamma_u(\widehat{\phi})]\chi(c)q[1 - Q(\widehat{\phi})].
\end{aligned} \tag{13}$$

The chance that the individual remains healthy is given by $\{1 - [1 - \gamma_u(\widehat{\phi})]\chi(c)\}$, while the odds that they will not are $[1 - \gamma_u(\widehat{\phi})]\chi(c)$. In the latter case, the person will get treated with probability q and not with $1 - q$. The term $Q(\widehat{\phi})$ reflects the odds of the partner being treated, while the one $1 - Q(\widehat{\phi})$ gives the odds that the companion is not.

The symmetric probabilities for when the partner is healthy ($\widehat{\phi} = 1$) but the individual is infected or treated ($\phi = 0, t$) are

$$\begin{aligned}
Y(t, 1|\phi, 1, \widehat{c}, c) &= \{1 - [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})\}Q(\phi); \\
Y(0, 1|\phi, 1, \widehat{c}, c) &= \{1 - [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})\}[1 - Q(\phi)]; \\
Y(t, 0|\phi, 1, \widehat{c}, c) &= [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})(1 - q)Q(\phi); \\
Y(0, 0|\phi, 1, \widehat{c}, c) &= [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})(1 - q)[1 - Q(\phi)]; \\
Y(t, t|\phi, 1, \widehat{c}, c) &= [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})qQ(\phi); \\
Y(0, t|\phi, 1, \widehat{c}, c) &= [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})q[1 - Q(\phi)].
\end{aligned} \tag{14}$$

In the above equations, the term $[1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})$ gives the odds that the partner will become infected.

Next, both partners might be infected ($\phi \in \{0, t\}$ and $\widehat{\phi} \in \{0, t\}$), in which case a healthy future is no longer an option. The only question that remains is whether the future sees treatment or not, so that

$$Y(\phi', \widehat{\phi}'|\phi, \widehat{\phi}, c, \widehat{c}) = \begin{cases} [1 - Q(\phi)]Q(\widehat{\phi}), & \text{for } (\phi', \widehat{\phi}') = (0, t); \\ [1 - Q(\phi)][1 - Q(\widehat{\phi})], & \text{for } (\phi', \widehat{\phi}') = (0, 0); \\ Q(\phi)Q(\widehat{\phi}), & \text{for } (\phi', \widehat{\phi}') = (t, t); \\ Q(\phi)[1 - Q(\widehat{\phi})], & \text{for } (\phi', \widehat{\phi}') = (t, 0). \end{cases} \tag{15}$$

The last remaining case is where both partners are currently healthy. Here, $Y(1, 1|1, 1, c, \widehat{c}) = 1$, implying that $Y(\phi', \widehat{\phi}'|1, 1, c, \widehat{c}) = 0$, when $\phi' \in \{0, t\}$ and/or $\widehat{\phi}' \in \{0, t\}$.

C.3. Stationary Equilibrium

A stationary equilibrium for the developed framework is formulated now. First, the equilibrium 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 type- x singles that have health status ϕ and discount factor d . Similarly, let $\mathcal{L}^d(\phi, \widehat{\phi}; x, \widehat{x})$ stand for the measure of long-term relationships for type- x individuals with health status ϕ and discount factor d who are coupled with a partner of type \widehat{x} and health status $\widehat{\phi}$. Given some distributions \mathcal{S} and \mathcal{L} of singles and married people, the sexual behavior of individuals according to their decision rule Π [$\Pi_r^d = \Pi_r^d(\phi, x)$ for each status and 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. In steady state, the distributions of singles and married people remain constant, and are determined by a fixed point of this operator:

$$(\mathcal{S}^\beta, \mathcal{L}^\beta, \mathcal{S}^\iota, \mathcal{L}^\iota) = \mathbf{T}(\mathcal{S}^\beta, \mathcal{L}^\beta, \mathcal{S}^\iota, \mathcal{L}^\iota; \Pi). \quad (16)$$

Next, the expectations over the fraction of types in each market have to be consistent with the aggregation of individual choices in equilibrium. 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 , type- x with status ϕ , and discount factor d , is given by

$$\mathcal{M}_{g,r}^d(\phi, x) \equiv \begin{cases} \Pi_{g,l}^d(\phi, x) \mathcal{S}_g^d(\phi; x), & \text{if } r = l, \\ [1 - \Pi_{g,l}^d(\phi, x)] \Pi_{g,r}^d(\phi, x) \mathcal{S}_g^d(\phi; x), & \text{if } r = p, u. \end{cases} \quad (17)$$

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. Then the fraction $R_{s,r}(\phi)$ of agents with health status ϕ in market s of gender g is given by

$$R_{g,s}(\phi) = \frac{\sum_d \sum_x \mathcal{M}_{g,s}^d(\phi, x)}{\sum_d \sum_x \sum_{\phi'} \mathcal{M}_{g,s}^d(\phi', x)}, \quad \text{for all } g \text{ and } s \in \{p, u\}. \quad (18)$$

For the long-term market, the relevant fraction is given by

$$R_{g,l}(\phi, c) = \frac{\sum_d \sum_x \mathcal{M}_{g,l}^d(\phi, x) \mathcal{I}(c(x) = c)}{\sum_d \sum_x \sum_{\phi'} \mathcal{M}_{g,l}^d(\phi', x)}, \quad \text{for all } g, \quad (19)$$

where $c(x)$ is a slight abuse of notation that denotes the circumcision status of the agent that is contained in his or her type x . The function $\mathcal{I}(\cdot)$ is an indicator function that takes the value of 1 if its argument is true. Note that $R_{f,s}(\phi)$ and $R_{f,l}(\phi, c)$ denote the distributions among women, which are relevant for *men* when determining their odds of getting infected. Similarly, $R_{m,s}(\phi)$ and $R_{m,l}(\phi, c)$ refer to the odds among men, but are relevant for *women* when making their decisions.

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

$$\sum_d \sum_x \sum_\phi \mathcal{M}_{f,r}^d(\phi, x) = \sum_d \sum_x \sum_\phi \mathcal{M}_{m,r}^d(\phi, x), \quad \text{for all } r. \quad (20)$$

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

$$z_{f,r} + z_{m,r} = 0, \quad \text{for all } r. \quad (21)$$

This leads to the following formal definition of equilibrium.

DEFINITION: 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, $z_{g,r}$, a set of stationary distributions, $\mathcal{S}_g^d(\phi; x)$ and $\mathcal{L}_g^d(\phi, \hat{\phi}; x, \hat{x})$, and status/type prevalence in each market, $R_{g,s}(\phi)$ and $R_{g,l}(\phi, c)$, for all $d = \{\iota, \beta\}$, $g \in \{f, m\}$, $r \in \{l, p, u\}$, $s \in \{p, u\}$, 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 (4) and (8), taking as given transfer payments and HIV/AIDS prevalence rates.
2. The stationary distributions, $\mathcal{S}_g^d(\phi; x)$ and $\mathcal{L}_g^d(\phi, \hat{\phi}; x, \hat{x})$, solve the appropriately gender subscripted version of (16).
3. The distributions over health status for each market, $R_{g,s}(\phi)$ and $R_{g,l}(\phi, c)$, are given by (18) and (19) using (17).
4. The transfer payments, $z_{r,g}$, are such that the markets for all types of relationships clear according to (20). Additionally, the flow of transfers across the genders must balance as specified by (21).

C.4. Stationary Distributions

The transition operator \mathbf{T} defined in Section C.3 is now fully characterized. Before starting, recall that $\mathcal{I}(\cdot)$ is an indicator function that takes the value of 1 if its argument is true, and 0 otherwise. Focus on a particular gender so that the gender subscript can be omitted. Again, $\mathcal{S}^d(\phi; x)$ denotes the beginning-of-period mass of singles with discount factor d , health status ϕ , and type x . Next, $\mathcal{L}^d(\phi, \hat{\phi}; x, \hat{x})$ represents the beginning-of-period measure of long-term relationships for individuals of type x with health status ϕ and discount factor d who are coupled with a partner of type \hat{x} and health status $\hat{\phi}$. Finally, \mathcal{A} is the mass of individuals with the final symptoms of AIDS. The sexual behavior of individuals is governed by their decision rules, $\pi_r^d = \Pi_r^d(\phi, x)$.

Assume temporarily that only people who are of health status $\phi = t$ will be treated next period. Moreover, suppose that the individual's discount factor does not change. Given the beginning of period distributions \mathcal{S}^d , \mathcal{L}^d , and \mathcal{A} , one can compute the distributions at the beginning of next period under these assumptions. Call these \mathcal{S}'^d , \mathcal{L}'^d , and \mathcal{A}' . These will be adjusted subsequently for changing treatment status and discount factors. Before proceeding, define the following variable to represent the infectiousness of each short-term market:

$$\hat{\theta}_s = \sum_{\hat{\phi}} R_s(\hat{\phi})[1 - \gamma_s(\hat{\phi})], \quad \text{for } s \in \{p, u\}. \quad (22)$$

First consider *next period's distribution of single individuals*. Take up first the distribution of healthy singles next period:

$$\begin{aligned} \mathcal{S}'^d(1, x) &= (1 - \delta) \\ &\times \left\{ \mathcal{S}^d(1, x)[1 - \Pi_l^d(1, x)] \right\} \left\{ 1 - \Pi_p^d(1, x) - \Pi_u^d(1, x) \right\} \end{aligned}$$

$$\begin{aligned}
& + \sum_s \Pi_s^d(1, x) [1 - \widehat{\theta}_s \chi(c)] \Big\} \\
& + \sum_{\widehat{\phi}, \widehat{x}} [\mathcal{L}^d(1, \widehat{\phi}; x, \widehat{x}) + R_l(\widehat{\phi}, \widehat{c}) \Pi_l^d(1, x) \mathcal{S}^d(1, x)] \{1 - [1 - \gamma_u(\widehat{\phi})] \chi(c)\} \\
& \times [1 - (1 - \delta)(1 - \alpha_{\widehat{\phi}})(1 - \varepsilon)] \Big\} \\
& + \mu(x) \mathcal{I}(d = \iota). \tag{23}
\end{aligned}$$

Singles survive into the next period with probability $(1 - \delta)$, as captured by the first line. The second and third lines account for healthy singles this period that continue as healthy singles next period. There are $\mathcal{S}^d(1, x)$ such singles this period. They remain healthy singles if they do not successfully enter the long-term market, which is represented by the term in the first bracket, and if they either do not enter the short-term market or enter but do not get infected, as presented by the terms in braces. The fourth and fifth lines account for those who exit from marriage as healthy singles. The terms in the first bracket give the stock of individuals married to a partner of status $\widehat{\phi}$ at the start of the period plus those singles who newly marry such a partner this period. They remain healthy with probability $\{1 - [1 - \gamma_u(\widehat{\phi})] \chi(c)\}$, but the marriage breaks up with the probability in the bracket on the fifth line, $[1 - (1 - \delta)(1 - \alpha_{\widehat{\phi}})(1 - \varepsilon)]$. The final line is the inflow of newborns.

Consider next the distribution of infected individuals without treatment next period:

$$\begin{aligned}
\mathcal{S}^{dI}(0, x) & = (1 - \delta) \\
& \times \left\{ \mathcal{S}^d(1, x) [1 - \Pi_l^d(1, x)] \sum_s \Pi_s^d(1, x) \widehat{\theta}_s \chi(c) \right. \\
& + \sum_{\widehat{\phi}, \widehat{x}} [\mathcal{L}^d(1, \widehat{\phi}; x, \widehat{x}) + R_l(\widehat{\phi}, \widehat{c}) \Pi_l^d(1, x) \mathcal{S}^d(1, x)] [1 - \gamma_u(\widehat{\phi})] \chi(c) \\
& \times [1 - (1 - \delta)(1 - \alpha_{\widehat{\phi}})(1 - \varepsilon)] \\
& + \mathcal{S}^d(0, x) (1 - \alpha_0) [1 - \Pi_l^d(0, x)] \\
& + (1 - \alpha_0) \sum_{\widehat{\phi}, \widehat{x}} [\mathcal{L}^d(0, \widehat{\phi}; x, \widehat{x}) + R_l(\widehat{\phi}, \widehat{c}) \Pi_l^d(0, x) \mathcal{S}^d(0, x)] \\
& \left. \times [1 - (1 - \delta)(1 - \alpha_{\widehat{\phi}})(1 - \varepsilon)] \right\}. \tag{24}
\end{aligned}$$

The first four lines detail the same elements as in the previous equation, but now healthy individuals only transit to the untreated infected state, $\phi = 0$. Line five captures currently infected singles, who do not develop final-stage symptoms with probability $1 - \alpha_0$ and who do not enter the long-term market with probability $1 - \Pi_l^d(0, x)$, and therefore survive as infected singles. Lines six and seven account for individuals that either started in marriage or got married, similar to lines three and four, except now these individuals are currently infected. Again, they return as infected singles, if they do not develop final-stage symptoms, and if the marriage does not survive.

Finally, the distribution of treated individuals next period is given by

$$\begin{aligned}
\mathcal{S}^d(t, x) &= (1 - \delta) \\
&\times \left\{ \mathcal{S}^d(t, x)(1 - \alpha_t)[1 - \Pi_l^d(t, x)] \right. \\
&+ (1 - \alpha_t) \sum_{\widehat{\phi}, \widehat{x}} [\mathcal{L}^d(t, \widehat{\phi}; x, \widehat{x}) + R_l(\widehat{\phi}, \widehat{c})\Pi_l^d(t, x)\mathcal{S}^d(t, x)] \\
&\left. \times [1 - (1 - \delta)(1 - \alpha_{\widehat{\phi}})(1 - \varepsilon)] \right\}. \tag{25}
\end{aligned}$$

The four lines here correspond to lines one, five, six, and seven in the previous expression. The reason the intermediate lines are dropped is the temporary assumption that only individuals who were already in treatment at the beginning of the period are eligible for treatment next period. This will be adjusted later.

The mass of individuals with final-stage symptoms next period is

$$\mathcal{A} = \sum_{d, \phi, x} (1 - \delta) \left\{ (1 - \delta_2)\mathcal{A} + \left[\mathcal{S}^d(\phi, x) + \sum_{\widehat{\phi}, \widehat{x}} \mathcal{L}^d(\phi, \widehat{\phi}, x, \widehat{x}) \right] \alpha_{\phi} \right\}. \tag{26}$$

It comprises those that started the period in the final stage and neither died of natural causes nor of AIDS-related reasons. It also includes all other individuals who develop final-stage symptoms, which occurs with probability α_{ϕ} .

Now consider the *distribution of long-term marriages next period* for type- x individuals with health status ϕ and discount factor d who are coupled with a type- \widehat{x} partner with health status $\widehat{\phi}$. Start with a marriage between two healthy individuals. The marriage survives if neither spouse dies of natural causes or the marriage does not break up exogenously. The stock of marriages next period includes marriages in the current period made up from both old and new ones. The mass of such marriages next period is

$$\begin{aligned}
&\mathcal{L}^d(1, 1; x, \widehat{x}) \\
&= (1 - \delta)^2(1 - \varepsilon) \\
&\times \left\{ \mathcal{L}^d(1, 1; x, \widehat{x}) + [1 - R_l(0, \widehat{c}) - R_l(t, \widehat{c})]\Pi_l^d(1, x)\mathcal{S}^d(1, x) \right\}. \tag{27}
\end{aligned}$$

Next, move onto the case where the partner is infected or treated. The terms are similar to before, only now marriages break up for one additional reason, namely, the partner develops AIDS (probability $\alpha_{\widehat{\phi}}$). The person stays healthy with probability $\{1 - [1 - \gamma_u(\widehat{\phi})]\chi(c)\}$. So,

$$\begin{aligned}
\mathcal{L}^d(1, \widehat{\phi}; x, \widehat{x}) &= (1 - \delta)^2(1 - \varepsilon)(1 - \alpha_{\widehat{\phi}}) \{1 - [1 - \gamma_u(\widehat{\phi})]\chi(c)\} \\
&\times [\mathcal{L}^d(1, \widehat{\phi}; x, \widehat{x}) + R_l(\widehat{\phi}, \widehat{c})\Pi_l^d(1, x)\mathcal{S}^d(1, x)]. \tag{28}
\end{aligned}$$

A similar expression obtains for partnerships where the individual under consideration is infected or treated but the partner is healthy:

$$\begin{aligned}
\mathcal{L}^d(\phi, 1; x, \widehat{x}) &= (1 - \delta)^2(1 - \varepsilon)(1 - \alpha_{\phi}) \{1 - [1 - \widehat{\gamma}_u(\phi)]\chi(\widehat{c})\} \\
&\times [\mathcal{L}^d(\phi, 1; x, \widehat{x}) + R_l(1, \widehat{c})\Pi_l^d(\phi, x)\mathcal{S}^d(\phi, x)]. \tag{29}
\end{aligned}$$

In the case where both spouses are infected or treated, there is no longer the need to take into account the transmission of the disease. Now there is a chance that either person will develop symptoms. So,

$$\begin{aligned} \mathcal{L}^{nd}(\phi, \widehat{\phi}; x, \widehat{x}) &= (1 - \delta)^2(1 - \varepsilon)(1 - \alpha_\phi)(1 - \alpha_{\widehat{\phi}}) \\ &\quad \times [\mathcal{L}^d(\phi, \widehat{\phi}; x, \widehat{x}) + R_l(\widehat{\phi}, \widehat{c})\Pi_l^d(\phi, x)\mathcal{S}^d(\phi, x)]. \end{aligned} \quad (30)$$

Finally, introduce the *adjustments for a changing discount factor and changing treatment status*, which are mechanical parts of the model that do not involve any choices. Incorporate discount factor changes first. To do this, let D^{nd} represent generic auxiliary distributions that result from incorporating the transitions from the previous D^d due to changing discount factors. High-discount-factor individuals stay with a high discount factor, but low-discount-factor people switch to a high discount factor with probability η . Hence,

$$D^{n\beta}(\phi, \dots) = D^{\beta}(\phi, \dots) + \eta D^u(\phi, \dots), \quad (31)$$

$$D^{nu}(\phi, \dots) = (1 - \eta)D^i(\phi, \dots). \quad (32)$$

To give examples, $\mathcal{S}^{n\beta}(\phi, x) = \mathcal{S}^{\beta}(\phi, x) + \eta\mathcal{S}^u(\phi, x)$ counts the number of type- x singles of health status ϕ that end up with the high discount, β . Another example would be $\mathcal{L}^{nu}(\phi, \widehat{\phi}; x, \widehat{x}) = (1 - \eta)\mathcal{L}^u(\phi, \widehat{\phi}; x, \widehat{x})$.

The analysis focuses on steady states for the model. Therefore, the fixed point of the operator \mathbf{T} in (16) is being sought. The stationary distributions for singles and marrieds can now be recovered by taking into account changes in treatment status, since infected individuals with status 0 change to a treated status t with probability q :

$$\mathcal{S}^d(1, x) = \mathcal{S}^{nd}(1, x),$$

$$\mathcal{S}^d(0, x) = \mathcal{S}^{nd}(0, x)(1 - q),$$

$$\mathcal{S}^d(t, x) = \mathcal{S}^{nd}(0, x)q + \mathcal{S}^{nd}(t, x),$$

$$\mathcal{L}^d(1, 1, x, \widehat{x}) = \mathcal{L}^{nd}(1, 1, x, \widehat{x}),$$

$$\mathcal{L}^d(1, 0, x, \widehat{x}) = \mathcal{L}^{nd}(1, 0, x, \widehat{x})(1 - q),$$

$$\mathcal{L}^d(0, 1, x, \widehat{x}) = \mathcal{L}^{nd}(0, 1, x, \widehat{x})(1 - q),$$

$$\mathcal{L}^d(0, 0, x, \widehat{x}) = \mathcal{L}^{nd}(0, 0, x, \widehat{x})(1 - q)^2,$$

$$\mathcal{L}^d(1, t, x, \widehat{x}) = \mathcal{L}^{nd}(1, 0, x, \widehat{x})q + \mathcal{L}^{nd}(1, t, x, \widehat{x}),$$

$$\mathcal{L}^d(t, 1, x, \widehat{x}) = \mathcal{L}^{nd}(0, 1, x, \widehat{x})q + \mathcal{L}^{nd}(t, 1, x, \widehat{x}),$$

$$\mathcal{L}^d(0, t, x, \widehat{x}) = \mathcal{L}^{nd}(0, t, x, \widehat{x})(1 - q) + \mathcal{L}^{nd}(0, 0, x, \widehat{x})(1 - q)q,$$

$$\mathcal{L}^d(t, 0, x, \widehat{x}) = \mathcal{L}^{nd}(t, 0, x, \widehat{x})(1 - q) + \mathcal{L}^{nd}(0, 0, x, \widehat{x})q(1 - q),$$

$$\mathcal{L}^d(t, t, x, \widehat{x}) = \mathcal{L}^{nd}(t, t, x, \widehat{x}) + \mathcal{L}^{nd}(0, t, x, \widehat{x})q + \mathcal{L}^{nd}(t, 0, x, \widehat{x})q + \mathcal{L}^{nd}(0, 0, x, \widehat{x})q^2.$$

The right-hand sides of these equations together with (23) to (32) fully describe the fixed point of the operator \mathbf{T} in (16).

APPENDIX D: DETAILS ON MODEL EXTENSIONS

D.1. *The Diffusion of Better Information*

To capture uninformed individuals, consider a type- x person with $i = 0$. Let $\hat{s} \in \{a, p, u\}$ and define $\hat{I}(\hat{s}) = 1$, for $\hat{s} = p$, and $\hat{I}(\hat{s}) = 0$, otherwise. Likewise, define $J(\hat{s}) = 0$, if $\hat{s} = a$, and $J(\hat{s}) = 1$, otherwise. If healthy, the value from short-term sex, for $\hat{s} = \{a, u, p\}$, is now given by

$$\begin{aligned} \tilde{V}_s^\beta(1, x) &= \ln(y - z_s) + \{p\hat{I}(\hat{s}) + u[1 - \hat{I}(\hat{s})]\}J(\hat{s}) \\ &\quad + \sum_{\hat{\phi}} R_u(\hat{\phi})[1 - \gamma_u(\hat{\phi})]\beta[qV_i^\beta(t, x) + (1 - q)V_i^\beta(0, x)] \\ &\quad + \left\{1 - \sum_{\hat{\phi}} R_u(\hat{\phi})[1 - \gamma_u(\hat{\phi})]\right\}\beta V_i^\beta(1, x), \end{aligned} \quad (33)$$

where $z_a = 0$. Compared to the value function for informed individuals (3), uninformed people perceive all sex as being as risky as unprotected sex without circumcision. In the case of abstinence, the uninformed now worry about infection even when they do not have sex—cf. (1).

In the long-term market, an uninformed individual thinks that transmissions are governed as if people are not circumcised. That means that, for a uninformed type- x individual (so that $i = 0$),

$$\begin{aligned} \tilde{V}_i^\beta(\phi, \hat{\phi}, \hat{c}, x) &= \ln(y - z_i) + u + l + \alpha_\phi \beta A \\ &\quad + (1 - \alpha_\phi)(1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}})\beta \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, 0, 0) \tilde{V}_i^\beta(\phi', \hat{\phi}', \hat{c}, x) \\ &\quad + (1 - \alpha_\phi)[1 - (1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}})]\beta \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, 0, 0) V_i^\beta(\phi', x). \end{aligned}$$

(Note that c and \hat{c} have been set to 0 in the transition probability Y .) Similar adjustments need to be made for young uninformed type- x individuals. Now,

$$\begin{aligned} \tilde{V}_s^\iota(1, x) &= \ln(y - z_s) + \{p\hat{I}(\hat{s}) + u[1 - \hat{I}(\hat{s})]\}J(\hat{s}) \\ &\quad + \sum_{\hat{\phi}} R_u(\hat{\phi})[1 - \gamma_u(\hat{\phi})]\iota \left\{ \begin{aligned} &q[\eta V_i^{\beta\iota}(t, x) + (1 - \eta)V_i^{\iota\iota}(t, x)] \\ &+ (1 - q)[\eta V_i^{\beta\iota}(0, x) + (1 - \eta)V_i^{\iota\iota}(0, x)] \end{aligned} \right\} \\ &\quad + \left\{1 - \sum_{\hat{\phi}} R_u(\hat{\phi})[1 - \gamma_u(\hat{\phi})]\right\}\iota [\eta V_i^{\beta\iota}(1, x) + (1 - \eta)V_i^{\iota\iota}(1, x)], \end{aligned}$$

for $\hat{s} = \{a, u, p\}$. Last, the value function for a young uninformed individual in a long-term relationship is

$$\begin{aligned} \tilde{V}_i^\iota(\phi, \hat{\phi}, \hat{c}, x) &= \ln(y - z_i) + u + l + \alpha_\phi \iota A \\ &\quad + (1 - \alpha_\phi)(1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}})\iota \end{aligned}$$

TABLE XI
IMPROVED INFORMATION—MODEL VERSUS DATA

	Bench.	Condoms	Circumcision	ART		STDs
		$p = 5.5$	100%	$q = 0.01$	$q = 0.075$	$\gamma_u^m = 0.9$
Panel (a): 1996 benchmark						
HIV prevalence, %	14.4	20.7	4.5	12.7	7.0	11.1
Sex that is casual, %	19.8	34.6	26.2	21.6	35.0	20.2
Condom use (singles), %	27.9	48.8	25.8	26.4	26.0	26.4
Fraction of singles, %	47.4	58.9	50.3	47.5	58.1	46.2
Non-abstinent singles, %	66.8	72.2	82.1	73.1	86.1	71.7
Panel (b): 2004 benchmark						
HIV prevalence, %	10.3	15.8	5.6	11.3	8.1	9.5
Sex that is casual, %	15.7	34.0	28.9	20.5	41.5	18.7
Condom use (singles), %	32.8	59.2	22.4	27.2	24.1	27.6
Fraction of singles, %	48.0	62.4	56.7	51.1	65.7	49.6
Non-abstinent singles, %	53.6	66.4	75.6	62.5	83.6	60.3

$$\begin{aligned}
& \times \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, 0, 0) \left[\begin{array}{c} \eta \tilde{V}_l^\beta(\phi', \hat{\phi}', x) \\ + (1 - \eta) \tilde{V}_l^\beta(\phi', \hat{\phi}', x) \end{array} \right] \\
& + (1 - \alpha_\phi) [1 - (1 - \epsilon)(1 - \delta)(1 - \alpha_{\hat{\phi}})] \iota \\
& \times \sum_{\phi', \hat{\phi}'} Y(\phi', \hat{\phi}' | \phi, \hat{\phi}, 0, 0) \left[\begin{array}{c} \eta V_l^\beta(\phi', x) \\ + (1 - \eta) V_l^\beta(\phi', x) \end{array} \right].
\end{aligned}$$

Table XI reports the results of selected policy experiments using the environment with uninformed individual as a starting point—Panel (a): 1996 benchmark. For ease of comparison, the table also reports the results using the benchmark calibration—Panel (b): 2004 benchmark. As discussed in Section 6, in general, policies implemented when there are uninformed agents tend to have stronger effects. The reason is that uninformed agents do not change their behavior in response to the policy change.

D.2. Fertility

This little appendix describes how the model is parameterized when there is heterogeneous utility for fertility within marriage, as described in Section 5.3. Note that, conditional on an individual's circumcision status, there is only one source of permanent heterogeneity in the benchmark model: the time discount factors, β and ι . Since there is a one-to-one correspondence between β and ι , think about β as summarizing this heterogeneity. To keep the computational complexity fixed, leave the number of types constant. That is, for each type- x person with a different discount factor, β , assign a different level of utility from fertility $f(x)$; again, $f(x)$ is really just changing with β . Now, the instantaneous utility from a long-term relationship is given by $u + l + f(x)$.

Assume a quadratic form for the utility from fertility such that $f(x) = \theta_0 + \theta_1\beta + \theta_2\beta^2$, regardless of the value for c and ι . Note that θ_0 and l cannot be separately identified given the linear form of utility. Thus, three values must be calibrated: $\theta_0 + l$, θ_1 , and θ_2 . These are picked such that the new model fits the baseline data targets as closely as possible. The resulting parameters are: $\theta_0 = 28,207$, $\theta_1 = -58,380$, and $\theta_2 = 30,201$. The

TABLE XII
CALIBRATION—FERTILITY

Observation	Data	Model <i>Benchmark</i>	Model <i>w./Fertility</i>
HIV prevalence, %	11.8	10.3	10.0
Males	10	8.6	8.3
Females	13	12.1	11.8
Sex that is casual, % (of all)	18	16	18
Condom use for casual sex, %	39	33	29
Singles that had casual sex in past year, %	47	54	65
Singles, %	33	48	47
Married by age 22, %			
Males	58	57	54
Females	90	63	58
Married by age 50, %			
Males	100	98	78
Females	100	98	88
Deaths related to HIV, %	29	25	32

fertility benefit $f(x)$ ranges from -1.55 to 21.23 . Table XII reports results from this new parameterization.

APPENDIX E: LIFE-CYCLE IMPLICATIONS

Figure 6 plots HIV/AIDS prevalence by age.⁴³ Both the data and model agree on a hump-shaped infection pattern, despite the fact that individuals in the model become sexually active earlier than is observed in the Malawian 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 been sexually active for a longer period of time. Therefore, a larger percentage of the older population is infected with HIV/AIDS. Second, 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 6 also illustrates the differential patterns of infection between the sexes. The figure shows that women get infected earlier than men, in both the data and model.

Figure 7 compares the fraction of the population that has ever married in the model versus the data. The model generates the earlier marriage of women (relative to men) via their higher infection risk. Men eventually “catch up,” and by age 50 almost everyone is married, in both the data and model.

The model also does a very nice job of matching the decline in risky activity over the life cycle. Older people are less likely to be single; see Figure 8. As people age, they are thus less likely to engage in casual sex; this is also reported in Figure 8. The fact that the discount factor stochastically rises with age helps to generate this pattern.

An additional prediction of the model relates to the causes of death, since individuals may die either due to HIV/AIDS or due to other natural causes. Figure 9 compares the

⁴³The data are fitted with a third-order polynomial. The somewhat choppy raw data are due to the small sample sizes.

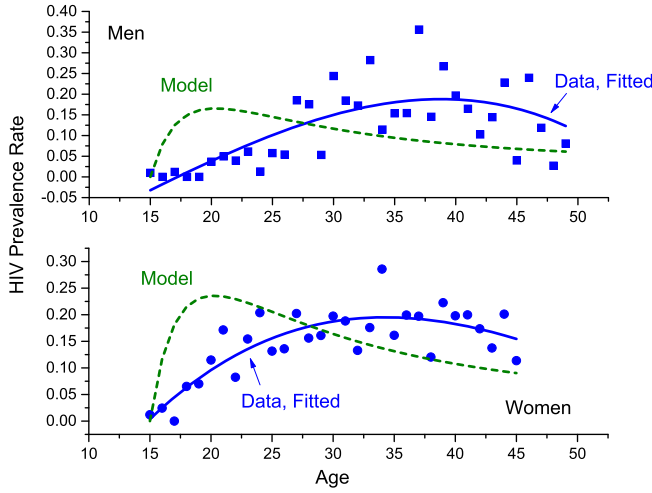


FIGURE 6.—HIV prevalence rate—men versus women, model versus data.

model prediction over the life cycle with its data counterpart. Both the data and model exhibit a hump-shaped pattern of HIV/AIDS-caused deaths; this is consistent with the hump-shaped pattern of infection rates.

APPENDIX F: ROBUSTNESS

This appendix provides some sensitivity analysis regarding the parameters estimated in Section 4. Recall that 11 parameters were chosen by fitting the model to a specific set of data moments from Malawi. These are listed on the different rows of Tables XIII and XIV. Each of these two tables has three columns besides the first that lists the parameters. The column labeled “HIV—Benchmark” provides the HIV prevalence rate when the pa-

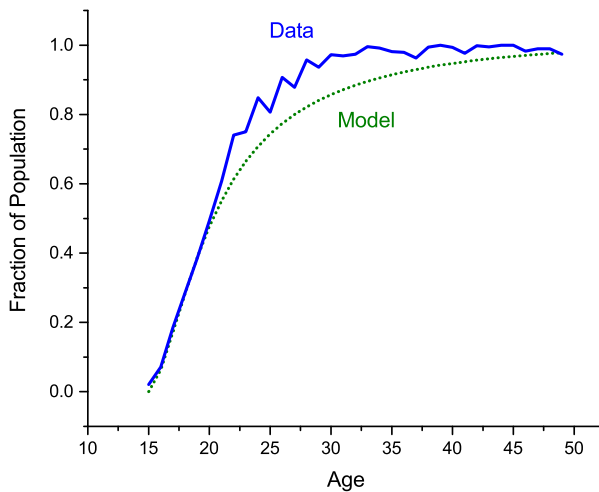


FIGURE 7.—Fraction of population ever married—model versus data.

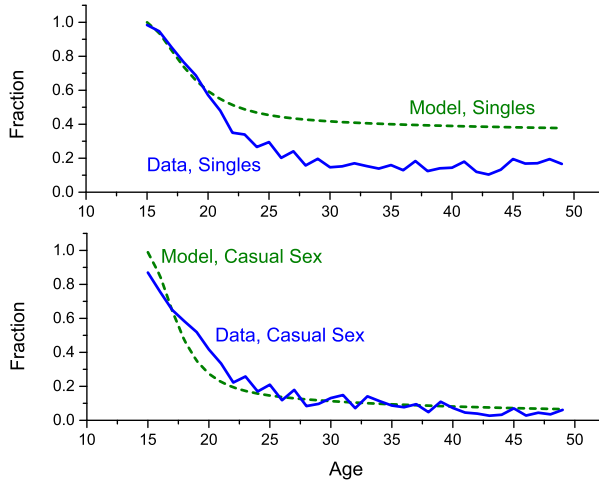


FIGURE 8.—Sexual behavior by age—model versus data.

parameter of each corresponding row is changed by 1% (Table XIII) or 10% (Table XIV).⁴⁴ The column “ Δ HIV—Circumcision (50%)” reports the change in the percentage HIV rate under the intervention that circumcises 50% of the males in the economy. Finally, the last column (Δ HIV—ART ($q = 5\%$)) presents the change in the percentage HIV rate when the infected have a 5% probability of receiving ART in each period.

Table XIII shows that the benchmark is quite robust when the parameters are changed by 1%. The HIV prevalence rate is always remarkably close to the 10.3% found in the benchmark calibration. Moreover, the results from the two main policy experiments (male circumcision and ART) are also very close to the changes found in the benchmark. Jux-

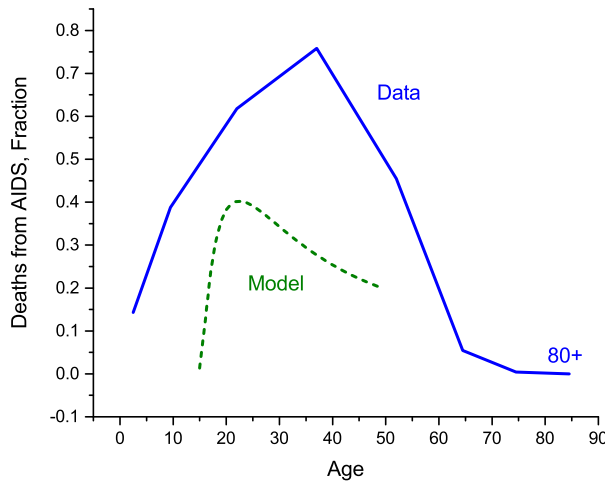


FIGURE 9.—Deaths by HIV/AIDS by age, fraction—model versus data.

⁴⁴To be precise, the rows for the discount factors (β_{\max} and β_{\min}) report changes on the discount rates $\rho = (1 - \beta)/\beta$.

TABLE XIII
ROBUSTNESS—1%

	HIV—Benchmark	Δ HIV—Circumcision (50%)	Δ HIV—ART ($q = 5\%$)
Main calibration	10.3	-1.2	-0.8
p	10.3	-1.2	-0.7
u	10.2	-1.2	-1.1
l	10.1	-1.2	-1.0
β_{\max}	10.3	-1.2	-0.8
β_{\min}	10.4	-1.3	-0.8
ι_{change}	10.2	-1.2	-0.7
A	10.3	-1.2	-0.9
η	10.2	-1.2	-0.7
ω_s	10.2	-1.2	-0.9
ω_l	10.3	-1.2	-0.7
κ	10.3	-1.2	-0.8

tapose these numbers with the ones reported in Table XIV, in which each parameter is changed by 10%. The percentage change now is considerably larger. Correspondingly, the HIV prevalence rate now changes compared with the main calibration. This suggests that, in order to fit the moments targeted in the calibration, the parameters should be close to the ones found in the estimation. At the same time, the percentage changes in the policy experiments are remarkably similar, even if individual parameters are changed by 10%.

APPENDIX G: COMPUTATIONAL DETAILS

A capsule summary of the numerical algorithm used to solve the benchmark model is provided here. There are two key steps. The first step involves solving the model for a given set of parameter values. In the second step, the algorithm picks the parameter values to match the model's output with the data targets as closely as possible. The first step proceeds as follows:

1. The static problems (4) and (8) that yield the meeting probabilities are solved. The solution to these problems implies that each π can be implicitly written as a nonlinear

TABLE XIV
ROBUSTNESS—10%

	HIV—Benchmark	Δ HIV—Circumcision (50%)	Δ HIV—ART ($q = 5\%$)
Main calibration	10.3	-1.2	-0.8
p	10.3	-1.2	-0.8
u	9.1	-1.5	0.0
l	8.8	-1.3	0.3
β_{\max}	10.3	-1.2	-0.8
β_{\min}	11.4	-1.5	-1.8
ι_{change}	9.0	-0.5	0.5
A	10.3	-1.2	-0.8
η	9.8	-1.1	-0.3
ω_s	9.8	-1.3	-0.5
ω_l	10.9	-1.3	-1.3
κ	10.1	-1.2	-0.7

function of the difference between either two \tilde{V} 's or a \tilde{V} and V . Given a grid of values for the \tilde{V} 's and V 's, the π 's can be computed at each grid point using the bisection method. The values for the π 's when the \tilde{V} 's and V 's lie off the grid can be obtained using an interpolation scheme.

2. One outer loop solves for the market-clearing prices using the NEWUOA algorithm. This algorithm picks the prices to minimize excess demand in the three relationship markets.

3. In an inner loop, the value functions and stationary distributions are determined computationally, given prices, using standard iterative procedures. First, the "matched" value functions (the \tilde{V} 's) are computed for each type of individual. Then, the ex ante value functions (the V 's) are calculated using a linear interpolation scheme for the π 's that employs the results from 1. The stationary distributions are computed using the formulas in Appendices C.3 and C.4.

In the second step, the parameter values are calibrated using a Pattern Search algorithm. The calibration algorithm and the solutions to the static problems in 1 are implemented in MATLAB, while the more computationally demanding loops in 2 and 3 are coded in FORTRAN.

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