Sexually Transmitted Infections with Semi-Anonymous Matching

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Abstract

We analyze the spread of sexually transmitted infections in an environment where individuals search for a sexual partner, and when found, cannot verify whether his partner is infected. Decisions are based on a variety of factors including the proportion infected, the likelihood of safe sex, the rate of detection and treatment, the cost of infection, and the length of search. The model demonstrates how directed search induces a separating equilibrium, and as a result, supports empirical evidence demonstrating the importance of sero-sorting. Furthermore, the model reinforces arguments that decreasing the costs of infection increases the infection rate. The model is calibrated and the policy implications are analyzed within the context of men who have sex with men.  

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1 Introduction

In economics, the law of demand says if the expected cost of sex decreases, then participation in the activity will increase, holding all else equal. Often referred to as “treatment optimism” in the public health literature (de Ven et al. [2000]), this simple and intuitive idea has been steadily gaining support. For instance, Dr. Richard Wolitski, the acting director of the CDC’s Division of HIV/AIDS Prevention, suggested one factor for a recent rise in HIV/AIDS diagnoses was because some men perceive it to be a less severe disease than it once was (Tuller [2008]). Furthermore, Kalichman et al. [2007] and Bruce et al. [2012] provide empirical support that decreasing costs have influenced beliefs which have increased activity among MSM groups. We support the argument by providing a structural model which demonstrates how “optimism” can lead to an increase in the incidence of HIV/AIDS and other sexually transmitted infections (STIs). In particular, we find incidence of infection increases from an increase in safe sex practices, an increase in the rate of detection, an increase in the rate of treatment, and a decrease in the daily cost of the infection.

Although the policy implications appear discouraging, the model also demonstrates how an alternative “economic” mechanism can reduce the incidence of STIs. Furthermore, we suggest how the mechanism can be improved to further reduce infections. Specifically, Dow and Philipson [1996] find empirical evidence that HIV infections are reduced by one-third due to sero-sorting: HIV/AIDS infected individuals match with other HIV/AIDS infected partners. We support the empirical evidence by demonstrating how individuals with asymmetric information who search

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2 Kalichman et al. [2007] findings are suggestive due to limitations of their data.
3 Lakdawalla et al. [2006] finds sexual activity of HIV positive individuals increased after the introduction of better treatments for HIV possibly due to higher quality of life. However, they find negative individuals reduced their activity. We investigate this in Section 6.1.
4 Refer to Morin et al. [2009] and Xia et al. [2006] for other studies regarding sero-sorting.
for sex can be partially separated into a group with a higher rate of infection and a group with a lower rate of infection. As a result, if individuals match within each group, then each encounter has a lower probability of spreading the infection. The separating equilibrium exists if individuals are able to direct their search to a variety of “centralized markets” rather than meeting a match randomly in the general population.

Two assumptions are key in proving how directed search creates a separating equilibrium and reduces the incidence of HIV/AIDS. First, individuals engage in semi-anonymous sex, i.e., they have limited information and cannot verify whether a partner is positive or negative for HIV/AIDS. Thus, someone who is not infected is willing to incur a cost (such as a fee or non-monetary hurdle) to insure himself against meeting and matching with someone who is infected. Second, individuals search for a sexual partner, and the larger the proportion of individuals who participate, the easier it is to find a match. As a result, a central market will attract participants because it reduces matching frictions. Combining these two assumptions implies if individuals were allowed to direct their search to a variety of locations (or markets), and a “market maker” created a barrier to entering one of the locations, then infected and uninfected individuals would search in separate markets and not match. In economic terms, directed search can be used to signal an agent’s type, a signal that results in a separating equilibrium as pioneered by Spence [1973] among others.

Besides building a model to highlight the importance of directed search, we show how the standard public health policies used to reduce morbidity and mortality among the infected decreases the costs of infection and in turn could increase participation, matching rates, and new infections. We analyze the issues both analytically and also use empirical evidence by calibrating the model.

5The same search framework has been used to understand other types of behavior including marriage (Mortensen [1988]), unemployment (Burdett and Mortensen [1980]), and illicit drug consumption (Galenianos et al. [2007]).
to men who have sex with men. The key assumption, both analytically and empirically, is individuals weigh the costs and benefits from participating and some do not participate because of the likelihood of infection.

In relation to the literature, several of our results are likely to occur in a simpler economic model. Philipson and Posner [1993] provides a good example of such an analysis. Both models predict an increase in the rate of infection if the day to day costs of the infection fall, i.e., medications become cheaper, societal stigma falls, or anti-viral drugs reduce the negative effects of being infected. However, the incorporation of search frictions differentiates our model in several key ways. Specifically, we find the introduction of safer sex increases the level of infection due to the higher level of participation and higher rate of matching. Frictions are also key in predicting an increase in infection if a cure for HIV is discovered and distributed. Finally, incorporating search is critical in determining why central markets exist and demonstrates how the markets create a separating equilibrium that can reduce infection.

The model’s predictions are important to the current public health debate for several reasons. First, the model’s results suggest many policies are inadequate and motivates the need for alternative ways to reduce HIV/AIDS and other STIs. Second, the model helps public health officials understand the role internet services, bars, and other centralized places play in facilitating semi-anonymous sexual encounters. A more thorough understanding of such places is important because they play a role in the spread of sexually transmitted infections (refer to Liau et al. [2006] for a survey). If one compares semi-anonymous matching with meeting prospective partners among

\footnotesize{Our model supports arguments by Lakdawalla et al. [2006] who find HIV/AIDS on the rise at the same time individuals become more vigilant about safe sex, i.e., during a period with increased condom usage.}

\footnotesize{Papers such as McFarlane et al. [2000] and Bolding et al. [2005] find roughly 15% of those interviewed have sought a sexual partner on the internet, while between 40% and 50% of men who have sex with men had done so.
more tightly linked networks of real life (as opposed to online) friends, the sexual encounters arranged in centralized locations are likely to be (1) geographically broader, (2) increasingly anonymous, and (3) more varied (i.e., more sexual partners). Together, these factors imply participants have less verifiable information when making decisions with disease transmission implications. Moreover, they significantly weaken traditional public health response tools such as contact tracing and partner notification, thereby undermining a critical core of measures that have been proven effective against HIV and other STIs. Therefore, the model provides a new perspective for public health officials as they try to understand the role centralized locations or markets play in the spread of STIs. Third, the model provides a variety of suggestions on how public health officials should respond to internet services and other markets that facilitate semi-anonymous sexual encounters. In particular, the model suggests that making new rapid-diagnostic tests available, much the way home pregnancy tests are now available, may enhance a separating equilibrium.

The following sections introduce the model’s environment, characterize its equilibria, calibrate the model, and analyze the effects of public health policies and the separating mechanism.

2 Environment

Assume individuals are risk-neutral and discount at rate $r > 0$ in a continuous time environment. Also, individuals search for sex and it takes time to find a willing partner. In determining the time it takes to find a partner, assume $Q$ agents choose to participate and accept a match. To obtain a match, let $Q_1$ agents solicit a sexual partner while $Q - Q_1$ wait for a solicitation. Agents choose whether to search actively or wait depending upon what action produces a higher chance of finding a partner, or matching. Given $M$ matches within the economy, the number of times
active and waiting agents match is on average $M/Q_1$ and $M/(Q-Q_1)$, respectively. It follows that $Q_1 = Q/2$ for agents to be indifferent between searching actively or waiting. In determining $M$, the probability a patient agent receives a particular solicitation is $1/(L - Q/2)$ given the population is made up of $L$ individuals.\footnote{Agents search uniformly over the entire population. Also, if one receives more than one potential match, then they randomly choose one with which to match.} In turn, the probability a waiting agent doesn’t receive any solicitations is $(1 - 1/(L - Q/2))^{Q/2}$, which equals $e^{-Q/(2L-Q)}$ as $L$ and $Q$ approach infinity and the ratio $q = Q/L$ is constant. Therefore, assuming a mass of agents results in a matching rate of

$$\alpha(q) = 1 - e^{-q/(2-q)}. \quad (1)$$

The key feature of the matching function described here is that as participation rises the matching rate rises.\footnote{The matching function is non-standard, but is taken to model an environment where participants do not know who is willing to participate. Alternative functional forms can be considered but the results do not change as long as the rate of matching increases with the rate of participation. Refer to Petrongolo and Pissarides [2001] for a survey on the matching function.} The functional form of the matching function is not particularly important to our results because the decision to participate in the market, the probability of infection, and the spread of the disease are affected by $\alpha$ and not $q$ directly. If an agent matches, then the agent receives utility $b$.

Other than the benefits from matching, agents can become infected, i.e., switch from being negative to positive. If an agent becomes positive, then he transitions to the positive state and discovers that he has the disease at rate $\rho$. Therefore, the model allows for the possibility that some agents are unaware of their infection. The likelihood the agent becomes infected is equal to the proportion of positive individuals with whom they match times the probability the disease is transmitted. If infected, an individual incurs a flow cost $\gamma$.\footnote{The cost $\gamma$ can easily be modeled as a lump sum cost at the time of detection.} However, positive agents exit the positive state because they are treated or die, which happen at rates $\lambda$ and $\delta$, respectively. If an
agent dies, we assume he is replaced by another agent who is negative. As a summary, a list of the model’s parameters are provided in Table 1.

Insert Table 1 Here

The environment proposed has been developed with a focus on men who have sex with men (MSM) as this is the largest infected group.\textsuperscript{11} Prostitution, IV drug use, superinfection, and other considerations are excluded in order to clearly understand the interactions between the rate of infection, medical treatment, and the incentives individuals face. Furthermore, policy makers often design alternative approaches to prevent infections in other populations. We discuss the implications of these issues in the conclusion.

3 Equilibria

The focus of this paper is on steady-state equilibria where the participation rate, the distribution of positive and negative agents, the likelihood of infection, and the matching rate are time invariant. To begin, a simple equilibrium is analyzed where agents always know their own type ($\rho = \infty$), and the disease does not result in death ($\delta = 0$). Afterward, delayed detection and mortality will be incorporated into the characterization of the model’s equilibria.

3.1 Immediate detection without mortality

In equilibrium, the proportion of participating agents in the negative and positive states, $n$ and $p$ respectively, are determined by the following steady state conditions

\textsuperscript{11}Men who have sex with men includes men who identify themselves as homosexual, bisexual, or heterosexual but have engaged in sexual activity with other men.
\[ \alpha(q)p = \lambda n, \]  
(2) 

\[ n + p = 1, \]  
(3)

where \( \pi = tp \). \( \pi \) represents the likelihood of a negative agent contracting the disease given they’ve matched. The probability equals the likelihood of matching with a positive agent, which equals \( p \) in this environment, times the likelihood of transferring the disease, \( t \). Overall the flow equations captures how agents are moving through the market in the steady state. From (2), the flows in and out of the positive state must be equal. Therefore, the negative agents being infected, or in other words, the product of the rate of matches and the likelihood of infection, is equal to the proportion of positive agents exiting the positive state. Equation (3) states that active agents are either positive or negative. As a result, the proportion of negative and positive agents who are active are

\[ n = \frac{\lambda}{t\alpha(q)}, \]  
(4) 

\[ p = 1 - \frac{\lambda}{t\alpha(q)}, \]  
(5)

given \( \lambda < t\alpha(q) \). Otherwise, the disease is eradicated in the steady state.

The flow Bellman equations are

\[ rN = \alpha(q)[b + \pi(P - N)], \]  
(6) 

\[ rP = -\gamma + \alpha(q)b + \lambda(N - P), \]  
(7)

where \( N \) and \( P \) is the asset value of the negative and positive state, respectively. Equations (6) and
demonstrate how agents discount the future at rate \( r \), match at the rate \( \alpha(q) \), and upon matching, enjoy utility \( b \). However, negative agents cannot tell their partner’s type but know the match can lead to infection with probability \( \pi \). If infected, the agent suffers disutility \( P - N \) and transitions to the positive state. Alternatively, a positive agent suffers disutility \( \gamma \) and transitions to the negative state at rate \( \lambda \). If he exits, the agent no longer pays the cost of the disease which is encompassed by the capital gain \( N - P \). To reiterate, it is assumed agents can choose to not participate, or retain their abstinence, which they do if \( N < 0 \). The proportion of agents who choose to search is \( q^* \).

**Proposition 1** An equilibrium with \( q^* > 0 \) exists. There may exist two steady-state equilibria.

1. **Interior:** A mass of agents, \( q^* \in (0, 1) \), search such that

\[
\alpha(q^*) = \left( t\gamma - rb - \sqrt{(t\gamma - rb)^2 - 4t\gamma b(\lambda + \delta)} \right) / (2tb)
\]

2. **Corner:** All agents search (\( q^* = 1 \)).

Intuitively, if \( \alpha(q)t < \lambda \), then \( p \) goes to zero, infection does not exist, and \( \frac{\partial N}{\partial \alpha(q)} = b \) as seen in Figure 1. Once the rate of infection outpaces treatment, participants begin to pay the costs as represented by the drop in \( N \) when \( \alpha(q) = \lambda / t \). However, they continue to participate if \( N \geq 0 \). If \( N \geq 0 \) and agents enter, then participation rises as well as the matching rate.\(^{12}\) If the matching rate increases, then an agent’s benefit from participating increases by \( b \) while the cost from infection increases at a decreasing rate (because the population is becoming saturated with positive individuals). Therefore, individuals enter until they drive the benefit from entering to zero or until everyone has entered. Figure 1 illustrates how the interior equilibrium can arise. The arrows in the figure

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\(^{12}\)This is the key property of the matching function described in (34).
represent when agents enter and exit and how this affects $\alpha(q)$ and $N$. An interior equilibrium exists because $N$ is non-monotonic in $\alpha(q)$ and agents stop entering when $N = 0$. In addition, the minimum root of $N$ is the only stable interior equilibrium because agents choose to enter at the other point where $N = 0$ (which drives the equilibrium to the corner). The proof is provided in the appendix.

Insert Figure 1 Here

3.2 Delayed detection with mortality

Roughly 25% of the infected population are unaware of their infection and a key component of sero-sorting is that the positive agent knows he has the disease. If agents know they are positive, then the infected may choose to separate from the uninfected given the correct incentives. In addition, we allow for mortality ($\delta > 0$) which will complicate the analysis.

In determining the effects of delayed detection, the model has been extended to include an unknowingly positive state. Assuming no recall on the number of matches since the agent was last tested,\(^{13}\) an agent is in one of three states: negative, unknowingly positive, and knowingly positive where the asset value of each state is denoted as $N$, $P_1$, and $P_2$, respectively. The Bellman equations can be represented as

\[^{13}\text{If agents recalled the number of matches, then $\rho$ would be determined endogenously as agents would face the benefit of being cured versus the cost of being tested. Therefore, as the number of matches an agent receives increases, his probability of having the disease increases as well as the return to getting tested. At some point, the benefits outweigh the costs and the agent would choose to be tested. The length of time this takes would be equal to $\rho$.}\]
\[ rN = \alpha(q)[b + \pi(P_1 - N)], \quad (9) \]

\[(r + \delta)P_1 = -\gamma + \alpha(q)b + \rho(P_2 - P_1), \quad and \quad (10) \]

\[(r + \delta)P_2 = -\gamma + \alpha(q)b + \lambda(N - P_2). \quad (11) \]

Equations (9)-(11) demonstrate how positive and negative agents discount the future at rate \( r + \delta \) and \( r \), respectively. In addition, they match at the rate \( \alpha(q) \), and upon matching, enjoy utility \( b \). However, negative agents cannot tell their partner’s type but know the match can lead to infection with probability \( \pi \). If infected, the agent suffers disutility \( P_1 - N \) and transitions to the unknowingly positive state. Alternatively, the unknowingly positive agents suffer disutility \( \gamma \) and find out they have the disease at rate \( \rho \). Finally, the knowingly positive agents face the same costs and benefits as those who do not know but are treated at rate \( \lambda \).\(^{14}\) If they exit, the agent no longer pays the cost of the disease which is characterized by \( N - P_2 \).

In determining the probability of contracting the disease, the proportion of agents who are positive and active is determined by the steady-state flows. These are

\[ \alpha(q)\pi n = (\rho + \delta)P_1, \quad (12) \]

\[ \rho P_1 = (\lambda + \delta)P_2, \quad and \quad (13) \]

\[ n + P_1 + P_2 = 1, \quad (14) \]

where \( \pi = t(P_1 + P_2) \) and represents the likelihood a negative agent contracts the disease given they match. In words, (12) demonstrates the number flowing into the unknowingly positive state

\(^{14}\) The disutility \( \gamma \) is assumed to be the same in each state. However, this can easily be relaxed.
equals those flowing out. Equation (13) states the number who flow into the knowingly positive state equals those flowing out. Finally, (14) is the identity. The proportion of negative agents can be derived from (12)-(14) to get

\[ n = \frac{(\rho + \delta)(\lambda + \delta)}{(\rho + \lambda + \delta)\alpha(q)t}. \]  

(15)

Thus, the probability of contracting the disease is

\[ \pi = t \left( 1 - \frac{\kappa_\pi}{\alpha(q)t} \right). \]  

(16)

where \( \kappa_\pi = \frac{(\rho + \delta)(\lambda + \delta)}{(\rho + \lambda + \delta)} \).

As before, the equilibria hinge on the agent’s decision of whether to participate. With complete information, individuals enter whenever the benefits from a match \( b \) outweighs or equals the expected costs. Analytically, agents participate if \( \pi\gamma/(\alpha(q^*)\pi + r + \lambda) \leq b \) for the immediate detection case without mortality. If mortality and delayed detection are incorporated, then the costs change and the condition to enter becomes

\[ rN = \alpha(q) \left( b - \pi \frac{\alpha(q)\delta + r\gamma}{\alpha(q)\pi(r + \delta) + \kappa_N} \right) \geq 0, \]  

(17)

where \( \kappa_N = \frac{r(r+\rho+\delta)(r+\lambda+\delta)}{r+\rho+\lambda+\delta} \).

Note, if the rate of detection is instantaneous and mortality is not modeled \( (\rho = \infty \text{ and } \delta = 0) \), then the probability of being positive, negative, and contracting the disease is the same as described in Section 3.1.
such an event is increasing. This will result in another type of equilibrium. However, the spirit of
the equilibria with delayed detection and mortality is very similar to Proposition 1.

**Proposition 2** An equilibrium with \( q^* > 0 \) exists. There may exist three steady-state equilibria.

1. **Low Interior**: A mass of agents, \( q^* \in (0, 1) \), search such that

\[
\alpha(q^*) = \frac{-b(\kappa_N - \kappa_{\pi}) + t\gamma - \sqrt{(b\kappa_N - b\kappa_{\pi})^2 - 2tb\gamma(\kappa_N + \kappa_{\pi}) + t^2\gamma^2}}{2rtb}.
\]  

(18)

2. **High Interior**: A mass of agents, \( q^* \in (0, 1) \), search such that

\[
\alpha(q^*) = \frac{-b(\kappa_N - \kappa_{\pi}) + t\gamma + \sqrt{(b\kappa_N - b\kappa_{\pi})^2 - 2tb\gamma(\kappa_N + \kappa_{\pi}) + t^2\gamma^2}}{2rtb}.
\]  

(19)

3. **Corner**: All agents search \( (q^* = 1) \).

Almost the same intuition described in Proposition 1 applies here. Agents enter until either they
have all entered (a corner equilibrium), or agents enter until \( N = 0 \), which implies that only a
fraction participate. Also, \( N \) can increase or decrease as \( \alpha(q) \) changes and has at most two non-
trivial roots because \( \frac{\partial^2 N}{\partial \alpha(q)^2} \) is either strictly positive or strictly negative for all \( \alpha(q) \).\(^{16}\) Therefore, \( N \)
could take the shape shown in Figure 2. If it does, then agents could enter and drive the equilibrium
to what we refer to as the high interior equilibrium.\(^{17}\) The new type of equilibrium (high interior)
can be shown to arise because mortality occurs \( (\delta > 0) \). It is not due to a lag in detection \( (\rho < \infty) \).
The proof is provided in the appendix.

\(^{16}\)With mortality, an increase in \( \alpha(q) \) could increase the costs of infection at an increasing rate because agents do
not attain the benefits of an increasing \( \alpha(q) \) after they die.

\(^{17}\) A necessary condition for the high interior equilibrium to exist is for \( N < 0 \) at the corner.
4 Calibration

To predict the effects of different public health policies, we assign values to the model’s parameters by calibrating it to fit important observations. The calibration is primarily for illustrative purposes as we do not incorporate directed search.\(^\text{18}\) Section 5 discusses the model’s predictions analytically as well as quantitatively using the calibrated parameters.

We calibrate the model to fit the data for HIV/AIDS among MSM within the U.S. We focus on this group because the CDC [2008] reports half of new HIV infections in the U.S. occur within this group and that approximately the same number of new infections occurred annually between 2001-2006 (a key to calibrating to a steady-state equilibrium). In calibrating the model, we assume the unit of time corresponds to one week; set the rate of time preference to be 5% annually or \(r = 1.05^{1/52} - 1\) weekly; without loss of generality we normalize the benefit from a match to \(b = 1\).

In estimating the rate of diagnoses once infected, the CDC reports in 2006 that an estimated 1.1 million persons have HIV/AIDS of which 25% are undiagnosed. In addition, roughly 56,000 were diagnosed. Therefore, \(\rho = 56/(0.25 \times 1,100) = 0.2\) annually or 0.0039 weekly.

In estimating the survival rate, individuals cannot transition from the positive to the negative state which implies \(\lambda = 0\). In addition, the CDC reports roughly 15 thousand fatalities from AIDS occurred in 2006. Hence, \(\delta = 15/1,100\) per annum or \(2.6 \times 10^{-4}\) per week.

The remaining parameters are deduced through a simple calibration procedure. In determining the number of active individuals, we take the MSM population to be 2.5% of the U.S. male popula-
tion, or roughly 2,750 thousand.\textsuperscript{19} In addition, the CDC reports that roughly half of the 1.1 million cases of HIV/AIDS were obtained from men having sex with men. Therefore, the proportion of MSM who are positive \((1 - n)\) equals the number with HIV/AIDS (550 thousand) divided by the active population \((2,750q)\). To solve for \(q\), we use the Powers et al. [2008] finding that the risk of infection per contact, \(\pi\), is roughly equal to 1 in 1000 contacts. Therefore, plugging the values for \(\delta\), \(\rho\), and \(\pi\) into (15) to find \(n\), and using the fact that \(1 - n = 550/(2,750q)\), we find \(q = 0.42\). From this, we estimate \(t = 0.002\) using the fact that \(\pi = t(1 - n) = 1/1000\).

Notice we estimate an interior equilibrium \((q^* < 1)\) from the calibration. However, up to this point it is unclear whether the market is at a low or high interior equilibrium. To deduce the type of equilibrium, it must be true that \(\gamma = 1.44\) for \(N = 0\) given the calibrated flows. As a result, we find \(\frac{\partial^2 N}{\partial \alpha (q)} > 0\) from (36) which only allows for a low interior equilibrium as characterized in Proposition 2. However, the comparative statics of a high interior equilibrium closely resemble those of the low interior. Furthermore, we consider a corner equilibrium in Section 5.1.2. Table 2 summarizes the calibrated parameters.

\textbf{Insert Table 2 Here}

\section{5 Policy}

The effects of public health policies such as increasing safer sex or reducing the costs of infection depends on the type of equilibrium. Due to the results from the calibration, we consider carefully the low interior equilibrium where HIV/AIDS is deterring participation. In this case, we investigate quantitatively and qualitatively how policies affect participation and the incidence of HIV. After\textsuperscript{19} The estimated number of MSM comes from the CDC’s report on Sexual Behavior and Selected Health Measures. Purcell et al. [2010] also estimates 2.6\% of the male population engaged in MSM in the last 12 months.
the low interior equilibrium is considered, we provide the effects of public health policies when
the market is at a corner equilibrium.

We are investigating the results of public health policies within the context of the model for
two reasons. First, we want to highlight how incentives can play an important role in predicting
the results of a certain policy. More importantly, the investigation stresses the relevance of a sepa-
rating equilibrium, i.e., how directed search induces sero-sortinght. The separating equilibrium is
discussed in Section 5.2.

In addition to the comparative statics, we incorporate the effect public health policies have
on welfare. While there are costs associated with matching due to the likelihood of infection,
individuals participate because of the utility from matching. Therefore, we consider a welfare
function that captures the benefits from a match, the expected costs of an individual contracting
the disease, as well as the increased likelihood of others contracting the disease from matches
that occur in the future. Note the welfare function is based purely upon the model’s environment.
The model excludes other potential costs related to semi-anonymous matching such as morality,
abortions, divorce, etc. These are inherently linked to the discussion of STI prevention. However,
we are excluding them from our analysis. To capture welfare within the model, it is defined as the
weighted sum of individuals’ future expected utility. Analytically,

\[ W = q^*(nN + p_1P_1 + p_2P_2). \]  

(20)

In words, welfare is equal to the weighted average of the positive and negative agents’ well-being.
5.1 Public health policies

5.1.1 The interior equilibrium

To reiterate, the interior equilibrium is a result of HIV/AIDS acting as a deterrent. When HIV/AIDS is acting as a deterrent, the welfare function is simplified because $N = 0$ in equilibrium. Algebraically,

\[ W = -q^* \frac{b}{t}, \quad (21) \]

when detection is immediate and does not result in death. The welfare function simplifies to

\[ W = -q^* \frac{b (\delta + \lambda)(r + \delta + \lambda + \rho) + \rho (\delta + \lambda + \rho)}{(r + \delta + \lambda + \rho)(\delta + \lambda + \rho)}, \quad (22) \]

when $0 < \delta$ and $\rho < \infty$. Welfare is necessarily negative because the uninfected cannot verify whether their partner has a disease, thereby creating asymmetric information and a situation where sexual activity has a negative effect on average welfare. This is a strong statement about matching semi-anonymously. It is not in the best interest of overall welfare even when other issues are excluded. To reiterate, we are focusing on welfare not of the individual in a particular state but welfare on average within our model. Some individuals may experience positive shocks to utility due to finding a match. However, the average agent’s utility will increase as participation falls. Similarly, HIV treatment increases individuals’ well-being on a daily basis by alleviating pain. However, medical intervention could decrease aggregate welfare given an interior equilibrium as treatment can increase the overall number of infections.

The results might seem counterintuitive at first glance. However, the model predicts the facts described by Kenshaw [2008] which reports that HIV rates have increased for MSM under 30 by
32% between 2001 and 2006. The article goes on to describe the subtle intuition of the model, an effect referred to as treatment optimism. Specifically, the article quotes Dr. Frieden “People who grew up watching their friends die of AIDS are a lot more careful than those who didn’t.” The argument is supported with a rigorous econometric approach in Lakdawalla et al. [2006]. To summarize, successful treatment reduces costs while increasing risky behavior and the incidence of HIV/AIDS.

The first result finds that reducing the disutility of infection can decrease welfare. In this environment, reducing the day to day cost of the disease is equivalent to reducing $\gamma$.\(^{20}\)

**Proposition 3** Assume a low interior equilibrium. If the cost $\gamma$ decreases, then

1. the participation rate $q^*$ rises,

2. welfare falls, and

3. the proportion of infected individuals increases.

To understand the intuition, if $\gamma$ falls, then the cost of being sick falls. A fall in $\gamma$ (which could be interpreted as increasing the subsidies for treatment or reducing the burden of a drug regimen) reduces the costs of participation and therefore the level of participation rises. If participation rises, then infection rises and welfare falls (proof in appendix). Table 3 provides the quantitative results when detection is delayed.

To reiterate, if $\gamma$ falls, then the cost of the disease decreases and in turn an individual’s expected lifetime utility is higher, all else equal. However, all else is not equal. The lower cost induces more

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\(^{20}\)An example of this is use of treatments which increase well-being of an infected individual such as Highly Active Antiretroviral Therapy (HAART).
individuals to participate, which raises the rate of matching, the likelihood of infection and lowers the expected lifetime utility. As a result, we find social welfare within our model is decreasing in $\gamma$.

**Proposition 4** Assume a low interior equilibrium. If the transfer probability $t$ falls, then

1. the participation rate $q^*$ rises,

2. welfare falls if and only if

$$\frac{\partial q^*}{\partial t} > 1, \text{ and}$$

(23)

3. the proportion of infected individuals increases if and only if

$$\frac{\partial \alpha(q^*)}{\partial t} < -1.$$  

(24)

To understand the intuition, as $t$ rises the probability of contracting the disease falls. As the probability falls, the costs of participation falls and the level of participation increases. When participation rises, the rate of matching increases which could increase the proportion of positive agents given the rate of matching grows faster than $t$ falls. On the positive side, welfare is a function of $t$ as seen in (22). Therefore, welfare could rise while the proportion of infected individuals rises.
This result occurs because a decrease in $t$ reduces the costs from the asymmetric information which is inherent in the market (proof in appendix).

Table 4 demonstrates the quantitative results when detection is not immediate. The table shows (23) and (24) are satisfied. Therefore, our quantitative results suggest if the probability of transferring the disease increases, then participation and the proportion of infected individuals rises while welfare falls.

Insert Table 4 Here

Innovations in detection have taken place in HIV testing. Rapid testing for HIV allows for same day results while Western Blot or other methods can take weeks. Therefore, we investigate what happens if a policy is implemented that favors rapid testing, a policy which is equivalent to increasing the detection rate $\rho$ in our model.

**Proposition 5** Assume a low interior equilibrium. If the detection rate $\rho$ increases, then

1. the participation rate $q^*$ rises if $\lambda > 0$, and
2. the proportion of infected individuals increases if

$$\frac{\alpha(q^*)\lambda}{(\rho + \lambda + \delta)(\delta + \rho)} < \frac{\partial \alpha(q^*)}{\partial \rho}.$$  \hspace{1cm} (25)

Again, the results predict rising participation because a higher $\rho$ reduces the length and cost of infection. Furthermore, infections increase if the increase in detection ($\rho$) and effective treatment is out paced by the increase in matches $\alpha(q^*)$ and in turn the rate of infection. Welfare is highly dependent upon the parameters of the model and the analytical results have been excluded.
What is potentially surprising is the special case when $\lambda = 0$. In this case, participation is unchanged by increasing $\rho$ as agents bear the same cost of the disease whether they know it or not and knowing does not enable them to transition to the negative state.\textsuperscript{21} Table 5 presents the quantitative results which shows participation and welfare is unaffected by $\rho$.

\textbf{Insert Table 5 Here}

Deducing the effects from a change in $\delta$ is significantly more complicated because $\delta$ affects both the discount rate of those infected as well as the number infected. Hence, the analytical results are highly dependent upon the parameters of the model and are uninformative. Table 7 presents the quantitative results. The table shows how prolonging life increases the costs of participation. Therefore, we find fewer participating and welfare rising.

\textbf{Insert Table 7 Here}

To conclude, we consider the effects if treatment became available for HIV ($\lambda > 0$).

\textbf{Proposition 6} Assume a low interior equilibrium. If the treatment rate $\lambda$ increases, then

1. the participation rate $q^*$ rises if $\rho > 0$, and

2. the proportion of infected individuals increases if

$$\frac{\alpha(q^*)\rho}{(\rho + \lambda + \delta)(\delta + \lambda)} < \frac{\partial \alpha(q^*)}{\partial \lambda}.$$ \hspace{1cm} (26)

The intuition is similar to the previous results (proof in appendix). Table 6 presents the quantitative results and shows how very small changes in $\lambda$ would substantially change key aspects of the market.

\textsuperscript{21} This result would change if the disutility from infection changed when an agent discovered he had the disease.
5.1.2 The corner equilibrium

The calibration determined a low interior equilibrium. However, the conclusion that the market is at an interior equilibrium is contingent upon the functional form of $\alpha(q)$. Therefore, we find it informative to include the results for a corner equilibrium. At the corner, STIs do not act as a deterrent. Therefore, public health policies do not change an individual’s decision to participate, which in the previous sections lead to an increase the incidence of infection.

**Proposition 7** Assume a corner equilibrium. If detection ($\rho$), death ($\delta$), or treatment ($\lambda$) increases, or the transfer probability $t$ decreases, then the proportion of infected individuals decreases.

The proof is straightforward. The number of negative agents is \( \frac{(\rho + \delta)(\lambda + \delta)}{\alpha(1)\bar{t}(\rho + \lambda + \delta)} \). Therefore, if $\rho$, $\delta$, or $\lambda$ increases, or $t$ decreases, then the fraction of negative agents increases.

5.2 Economic policy

Medical treatment is very costly in terms of employing doctors, researching new ways to treat patients, or purchasing existing medicine. This is true whether the market is at an interior equilibrium and medical intervention is potentially increasing infection, or the market is at a corner and intervention is decreasing the aggregate number of STIs. In the face of this problem, we show how an economic mechanism, or a separating equilibrium, can arise and reduce the incidence of STIs as well as public health expenditures.

The idea is an “economic” mechanism can direct different types of agents (positive or negative) towards those of the same type. We show incentives can easily arise and create separation. The key
is participants cannot verify whether a partner is positive or negative for HIV/AIDS. Thus, someone who is not infected is willing to incur a cost (such as a fee or non-monetary hurdle) to insure himself against meeting and matching with someone who is infected. As a result, if individuals can direct their search to a variety of locations (or markets), and a “market maker” created a minimal barrier to entering one of the locations, then the uninfected would pay the cost while those infected would not. Therefore, the mechanism would separate the populations and reduce the spread of infection. Dow and Philipson [1996] finds empirical evidence of the mechanism by documenting how individuals sero-sort and how sero-sorting reduces the spread of HIV/AIDS.

To be specific, directed search assumes multiple markets can exist, called sub-markets, and agents choose to search in one of them. In such an environment, a central authority (or market maker) opens centralized markets where individuals can search for a match. In general, the mass of individuals in each market is $q_i$ for $i \in \{1, 2\}$ (two is sufficient), and those searching face the same coordination friction as described in (34). However, if the market is opened and contains only agents searching for a match, then the probability of receiving an offer is $\alpha(1) = (1 - e^{-1})$.\textsuperscript{22}

Hence, centralized markets can increase the rate of matching and sexual activity. As a result, the markets could increase the incidence of infection if positive and negative agents are not separating in sufficient quantities.

For separation to occur, the mechanism must encourage each type of agent to choose to enter a different market. To do this, the market maker must differentiate the two markets by imposing a cost to entering one of the markets such as a screening process (or imposing an entry fee). However

\textsuperscript{22} It could be assumed that the matching technology is improved in the centralized market and the rate of matches an agent engages in is only constrained by time. This will simply require $\alpha(1)$ to be redefined and equal to a different constant. For example, five per week on average or $\hat{\alpha} = 5$. We discuss this extension below.
levied, label the barrier to entry $\tau$. This brings us to a critical contribution of the paper.

**Proposition 8** If the cost of entry satisfies

$$
\tau \leq \alpha(1) \left( b - \frac{t \left( \frac{\lambda + \delta}{\lambda + \rho + \delta} \right) \left( 1 - \frac{\rho + \delta}{\alpha(1)t} \right) (\alpha(1)\delta + r\gamma)}{\alpha(1)t \left( \frac{\lambda + \delta}{\lambda + \rho + \delta} \right) \left( 1 - \frac{\rho + \delta}{\alpha(1)t} \right) (r + \delta) + \kappa N} \right),
$$

then negative agents are willing to participate if agents separate. The agents separate if the cost of entry satisfies

$$
0 < \tau < \left( \delta \alpha(1) + r\gamma \right) \frac{(\alpha(1)t\rho + (\delta + \rho)(\delta + \lambda))\kappa N}{(\alpha(1)t(r + \delta) + \kappa N)((r + \delta)(\alpha(1)t - \rho - \delta) + \kappa N)},
$$

where knowingly positive agents enter the free market and the rest enter the market with the barrier to entry.

Equation (27) insures $\tau$ is not so high that negative agents do not participate in the centralized market at all. The left hand inequality in (28) insures positive agents do not search in the “negative” market while the right hand side guarantees negative agents do not search in the positive market.

In other words, $\tau$ is bounded from above by the cost of contracting the disease.

The primary point of the model is to propose how the incidence of HIV/AIDS is reduced with sero-sorting and how such sero-sorting occurs even when individuals retain private information regarding their type. The answer we propose is directed search. However, the extent of the reduction from directed search depends upon the time it takes to detect the disease.

**Proposition 9** Assume two active sub-markets exist and $\tau$ satisfies (27) and (28).

1. If detection is immediate, then directed search eliminates STIs.

2. If detection is delayed, and $\alpha(1)t < \rho + \delta$, then directed search eliminates STIs.
3. If detection is delayed, $q^* = 1$, and $\alpha(1)t > \rho + \delta$, then directed search reduces the proportion infected by

$$\frac{\rho(\rho + \delta)}{\alpha(1)t(\lambda + \rho + \delta)}.$$  \hfill (29)

4. If detection is delayed, $q^* < 1$, and $\alpha(1)t > \rho + \delta$, then directed search reduces the proportion infected by

$$\frac{\delta + \rho}{\alpha(1)t} \left(1 - \frac{\alpha(1)}{\alpha(q^*)} \frac{(\delta + \lambda)}{\lambda + \rho + \delta}\right).$$  \hfill (30)

In the base case with immediate detection, the separation eradicates HIV because the negative market is free of positive agents. More realistically, if there exists a lag in detection, then the negative market contains some positive agents. If detection is lagged and positive agents leave the negative market faster than those entering, or $\alpha(1)t < \rho + \delta$, then the mass of positive agents in the negative market is zero in the steady state. Therefore, the disease is still eradicated. If this condition does not hold, then the mechanism might or might not reduce the number of infected agents. On one hand, if the market was originally at the corner equilibrium, then the rate of matching does not increase with the introduction of the centralized market and the likelihood that a negative agent matches with a positive agent is reduced. So, the proportion of infected individuals falls. On the other hand, if the market was originally at an interior equilibrium and the introduction of our mechanism increases the rate of contact, then the proportion of individuals infected in the steady state could increase if detection is relatively slow (proof in appendix).

Quantitatively, the calibration estimates $\alpha(1)t < \rho + \delta$. Specifically, $\alpha(1)t = 1.33 \times 10^{-3} < 4.18 \times 10^{-3} = \rho + \delta$. Therefore, our numerical example would suggest $p_1 + p_2 = 0$ in the steady-state, and HIV/AIDS should be eradicated with directed search. Essentially, the detection rate is
much faster than the transmission rate and the number of infected individuals would decrease to zero if directed search was introduced. However, our calibration is primarily for illustrative purposes as it does not determine the amount of directed and random search that occurs. Therefore, we suggest a slightly different calibration. Specifically, what if the market does not contain the same inherent frictions and the matching rate is much higher than predicted by the urn-ball matching problem? To answer the question, we examine a variety of potential matching rates to see the effects of directed search on HIV/AIDS. The results are in Table 8 where column 1 is the calibrated model without a centralized market. Column 2 demonstrates the effects of a centralized market that faces the frictions encompassed by (34). Finally, columns 3 and 4 highlight how the results change if the matching rate is determined by some exogenous factor due to the inherent nature of the centralized market.

Insert Table 8 Here

The quantitative examples show the model’s measure of welfare increasing with directed search. However, notice the proportion of infected agents could rise if matching occurs at a very fast rate. The results highlight the tradeoff with opening up a “hookup” market. Specifically, the matching rate becomes much faster because individuals are searching among those who are participating. Therefore, participants might find matches at a faster rate, and if they do, the rate and incidence of HIV might rise relative to the benchmark. Essentially, the reduction in the incidence of HIV/AIDS is hampered by delayed detection through imperfect separation.

In defence of directed search as an approach to reducing HIV/AIDS, notice that if public health officials increased the rate of detection $\rho$ by a sufficient amount, then directed search would un-
ambiguously reduce HIV/AIDS. Therefore, we suggest policies should be targeted to improve the rate of detection.\textsuperscript{23}

6 Extensions

The theory being proposed to model semi-anonymous matching is analogous to job matching in the labor market. This class of models has been extensively developed in the economics literature to investigate many features of labor markets.\textsuperscript{24} As such, the model we employ is quite flexible; it can be extended in numerous ways by either adding to or relaxing the assumptions. In this section we consider two such extensions that are quite different in practice but are similar in terms of the model. The first extension addresses how anti-viral drugs have enabled HIV positive individuals to increase their sexual activity (Lakdawalla et al. [2006]). The second extension considers an environment where multiple norms exist within one population. Both extensions are similar in that they affect the matching function.

6.1 Health and search intensity

Lakdawalla et al. [2006] finds the introduction of better treatments for HIV have increased the sexual activity of those with HIV and an increase in the incidence of HIV.\textsuperscript{25} The model predicts both results. However, they also find some evidence that the introduction of a better HIV treatment has reduced the sexual activity of those without HIV.\textsuperscript{26} They suppose this is due to a drastic increase

\begin{flushleft}
\textsuperscript{23}Note that current technologies focus on detecting antibodies to the HIV virus and can take up to several months to turn positive after an individual has become infected.
\textsuperscript{24}Rogerson et al. [2005] has an excellent survey of the labor search literature.
\textsuperscript{25}Kalichman et al. [2007] finds increases in sexual activity for both those with and without HIV as a result of treatment related beliefs.
\textsuperscript{26}There is ongoing debate regarding the sexual activity of the uninfected as a result of better treatment options. For example, MacKellar et al. [2011] study the beliefs of the HIV-negative regarding changes in susceptibility of HIV due to the introduction of HAART. They find evidence of treatment optimism and increased high risk sexual activity in those who have beliefs in higher levels of HAART-related efficacy. Bruce et al. [2012] also finds that uninfected
\end{flushleft}
in the activity of those with HIV. As improved treatments have allowed those with HIV to search with a higher level of intensity, the chance of a match over a given period of time increases for those with HIV. By introducing the assumption that search intensity rises as the cost of the disease falls, our model predicts the aforementioned results. Specifically, if search intensity is negatively correlated with the cost of the disease, then we find increased activity among positive individuals, decreased activity among negative individuals and the incidence of HIV rises.

When search intensity is introduced in our environment, the matching function changes. As before, we assume individuals search for sex and it takes time to find a willing partner. However, if a positive agent’s ability to participate is effected by his health, as measured by the cost of infection $\gamma$, then we propose a link between their participation and $\gamma$. Specifically, we’ll assume a negative relationship where the probability a positive agent can participate is $f(\gamma)$ per period and $df(\gamma)/d\gamma < 0$. In other words, the probability $(1 - f(\gamma))$ represents the likely a positive agent is too sick to participate at any point in time. If their ability to participate is limited, then their likelihood of matching changes as well. Specifically, $Q$ still represents the agents who search at any particular point. Furthermore, half search actively while the other half wait patiently. As a result, the rate a patient or active agent matches is still $\alpha(q) = 1 - e^{-q/(2-q)}$. However, the difference is a positive agent’s matching rate is $f(\gamma) \times \alpha(q)$ as they do not participate in certain periods due to their illness. Furthermore, $Q$ does not contain all the positive agents who want to match. Some are not searching due to illness. Thus, the new matching rate of the positive individual does not alter the flow equations represented in (12)-(14). However, it does change the likelihood of a negative agent contracting the disease given he matches. The likelihood decreased individuals’ beliefs in undetectable viral loads of the infected result in more high risk activity of the uninfected.
because the proportion of positive individuals in the pool of people actively searching is no longer equal to $p_1 + p_2$. Rather, it is $f(\gamma)(p_1 + p_2)$. So, the likelihood of contracting the disease given a negative individual matches becomes

$$\pi = t \left( \frac{f(\gamma)(p_1 + p_2)}{f(\gamma)(p_1 + p_2) + n} \right).$$

(31)

In words, $t$ is still the likelihood of transmission given someone matches with a positive agent. The likelihood of matching with a positive agent is now equal to the proportion of positive agents who feel well enough to search divided by the pool who are searching. The proportion of positive people in the pool who are well enough to search is $f(\gamma)(p_1 + p_2)$ while those searching is $(f(\gamma)(p_1 + p_2) + n)$. To reiterate, equations (12)-(14) do not change as they are written. However, the $\pi$ in the equations changes.

The Bellman equations are left fairly unchanged as well. Equation (9) is unchanged while (10) and (11) change to

$$(r + \delta)P_1 = -\gamma + f(\gamma)\alpha(q)b + \rho(P_2 - P_1), \text{ and}$$

(32)

$$(r + \delta)P_2 = -\gamma + f(\gamma)\alpha(q)b + \lambda(N - P_2).$$

(33)

In words, the rate of matching falls from $\alpha(q)$ to $f(\gamma)\alpha(q)$ while everything else is the same.

To demonstrate how the model can replicate the findings in Lakdawalla et al. [2006], we have provided a numerical example in Table 9. Rather than specify the functional form of $f(\gamma)$, we have provided a few example values. In this example, we have consider $f(\gamma) < 1$ as the starting parameter, rather than $f(\gamma) = 1$.\(^{27}\) This correlates to higher cost of infection associated with HIV/AIDS.

\(^{27}\)Recall $f(\gamma) = 1$ is the value calibrated in Section 4.
prior to widespread HAART availability. This also lines up with the fact that we have calibrated the model to data that comes after the introduction of the new treatments.

To reiterate, this example demonstrates that if the cost of the infection decreases and search intensity increases, then the number of partners for the infected rises, the number of partners of the uninfected falls and the likelihood of infection rises. In comparison to the previous results where $\gamma$ changes, note welfare increases as $\gamma$ decreases. This is distinctly different than the results in Table 3. These results are driven by the falling participation of negative agents.

**Insert Table 9 Here**

### 6.2 Multiple sexual norms

In this sub-section, we analyze the implications of a market that has two types of individuals. Specifically, assume individuals searching for sex have two different norms: A and B. The number of norms can easily be extended. In the case of two types, assume if a type A individual matches with a type B, then the match is disbanded without the realization of utility $b$. The difference between having two types of individuals versus one type comes in the form of the matching function. Following Section 2, assume $Q^j$ for $j \in \{A, B\}$ agents choose to participate and accept a match. To obtain a match, let $Q^j_1$ agents solicit a sexual partner while $Q^j - Q^j_1$ wait for a solicitation. Agents choose whether to search actively or wait depending upon what action produces a higher chance of finding a partner, or matching. Given $M^j$ matches within the economy, the number of times active and waiting agents match is on average $M^j/Q^j_1$ and $M^j/(Q^j - Q^j_1)$, respectively. It follows that $Q^j_1 = Q^j/2$ for agents to be indifferent between searching actively or waiting. In determining $M^j$, the probability a patient agent of type $j \in \{A, B\}$ receives a particular solicitation is $\frac{1}{(L - Q^A/2 - Q^B/2)}$.
given the population is made up of $L$ individuals.\footnote{As before, agents search uniformly over the entire population. Also, if one receives more than one potential match, then they randomly choose one with which to match.} In turn, the probability a waiting agent of type $j$ doesn’t receive any solicitations from their type is $(1 - (L - Q^A / 2 - Q^B / 2))^{Q^A / 2}$, which equals $e^{-Q^j / (2L - Q^A - Q^B)}$ as $L$ and $Q$ approach infinity and the ratio $q^j = Q^j / L$ for $j \in \{A, B\}$ is constant. Therefore, assuming a mass of agents results in a matching rate for a type A to be

$$a(q^A) = 1 - e^{-q^A / 2q^A-q^B}.$$  \hspace{1cm} (34)

The matching rate for a type B agent is identical except the superscripts are switched. Again, the key feature of the matching function described here is that if participation rises, then the matching rate rises. Note we are assuming an agent’s type is fixed and different types do not occasionally match or match and receive a lower level of utility. The model could be easily extended to allow for such considerations.

The multiple sexual norms problem follows the single norm problem except there are two of everything - two sets of Bellman equations (9)-(11), two sets of steady state flows (12)-(14), and the proportion of infected individuals who are of Type A and B.\footnote{For brevity, we do not provide a formal discussion of the steady state flows, the Bellman equations, nor a proof of the equilibrium.} The interior equilibrium is determined where both Type A and Type B asset values from participating, $N$, is zero.

Rather than an algebraic analysis of the multi-norm equilibrium, we have included a numerical example to demonstrate the extended model’s equilibrium and tie our work to the related literature. Specifically, the literature related to MSM suggests some types of sexual interactions face a higher likelihood of transmitting HIV. In our model, this translates into different transmission rates for the different types which we label $t_A$ and $t_B$. Taking from the calibration, we assume all the parameters
for each type are unchanged except the transmission rates. The interior equilibrium with multiple norms is presented in Table 10 along with the varying transmission rates.

The results show the benchmark case for the multi-norm model looks nearly identical to the single norm benchmark, i.e., we observe an equivalent proportion of those with STIs. The reason is due to the zero entry condition. The arrival rates are the only structural difference in the model with and without multiple norms. Thus, the arrival rates of matches in both models are equivalent in equilibrium given identical parameter values. The difference between the single norm and multi-norm models is the number of participants. The aggregate number is higher when multiple norms are present as the multiple norm model makes it significantly harder to match via an increased coordination friction. Thus a larger portion of agents participate before the matching rates and infection rates deters further entrants.

Moving away from the benchmark case, we investigate two cases where the transmission rates are unequal. We see in Case 1 an increase in the transmission rates of Type B individuals coupled with a decrease in the proportion of those with STIs. The counter-intuitive result is nearly identical to the case with a single sexual norm as seen in Table 4. The more counter-intuitive result is the decrease in welfare of Type A individuals. This is due to the fact type A participation rises. If participation rises, then welfare will fall as suggested in (22). Participation is rising because the matching rate of Type A agents falls when Type B agents exit the search process. This happens because active Type As are more likely to match incorrectly with the non-participating type B agents. Therefore, an interior equilibrium with a lower probability of matching encourages increased participation and lowers welfare. Case 2 reinforces the result that an increase in the transmission rates could decrease infection.
7 Conclusion

HIV treatment has greatly extended the life span and increased the quality of life for persons living with HIV. Unfortunately, our model supports empirical evidence that these interventions lead to “treatment optimism.” Hence, we predict future public health interventions that reduce the cost of infection will increase the rate of new infections. Compounding the problem, online hookup sites, and semi-anonymous sex in general, have been implicated in the spread of STIs and cause a breakdown in the traditional approaches to managing STI outbreaks like contact tracing. However, we find a semi-anonymous environment with directed search creates an incentive structure where credible signaling occurs. Due to the signal, these markets may reduce HIV/AIDS depending upon how much the markets increase the rate of matching. Whether the markets increase or decrease infection, we find they reduce the limited information problem, and as a result, can make individuals safer. Although the signaling mechanism may seem abstract, we point out centralized markets already exist and sero-sorting has been documented as a major factor in reducing the spread of infection (Cassels et al. [2009]). In addition, some of these markets have barriers to entry such as small monetary or non-monetary hurdles.

Given directed search with sero-sorting, one can think of two realistic policy prescriptions. First, we suggest that making new rapid-diagnostic tests available, much the way home pregnancy tests are now available, may provide a realistic way to enhance a separating equilibrium by reducing or eliminating “sero-guessing.” The suggestion is plausible as the tests are already available in clinical outpatient settings and are easily administered using saliva. Obviously, much development
and validation work would need to be done on the test, but our results would support subsidizing the development and more widespread deployment of such a test. Second, public health officials have discussed the shutting down of central markets as they are thought to increase the incidence of HIV/AIDS and other STIs. As demonstrated by the model, such a policy might reduce the incidence of STIs. However, our work suggests it would be better, assuming our limited welfare function, for health organizations to test individuals for their status rather than eliminating the places where they search. Besides, enforcing such a ban would likely be difficult if not impossible to impose.

It should be noted that our model focuses on the MSM population and the policy implications should be viewed accordingly. However, we have demonstrated that our model can be extended to include a broader range of issues. Future research extensions include prostitution, recall on the side of agents searching for a match, IV drug use, superinfection, differing transmission probabilities, marriage, coercion, analysis of different STIs with known cures such as syphilis, etc. While some of these extensions further complicate the findings, we expect the main results to be robust with respect to how directed search creates sero-sorting.
Appendix. Proofs of Propositions

Proof of Propositions 1 and 2  The proof of Proposition 1 is nearly identical to Proposition 2. Thus, the proof of the more general case has been provided. An equilibrium exists if there exists a point where the benefit from entry \((N)\) is non-negative in equilibrium. Such a point exists because \(0 < N\) for all \(0 < \alpha(q) \leq \kappa/3\) and \(N\) is continuous for all \(\alpha(q)\) (the only possibility for discontinuity is when \(\alpha(q) = \kappa/3\) but \(rN = \alpha(q)b\)) at this point because \(\pi = 0\). Therefore, there exists a point where either \(N = 0\) and \(0 < q^* \leq 1\) or \(0 < N\) and \(q^* = 1\).

Three different equilibrium may arise because \(N\) is a non-monotonic function of \(\alpha(q)\). Specifically, \(N\) can be manipulated to be

\[
rN = \alpha(q) \left( b - t \left( 1 - \frac{\kappa}{\alpha(q)t} \right) \frac{\alpha(q) \delta + r\gamma}{\alpha(q)t \left( 1 - \frac{\kappa}{\alpha(q)t} \right) (r + \delta) + \kappa N} \right). \tag{35}
\]

which implies

\[
\frac{\partial^2 N}{\partial \alpha(q)^2} = 2t \kappa N \frac{b \delta ((\delta + r) \kappa - \kappa N) + \gamma r (r + \delta)}{(\alpha(q) - \kappa) (r + \delta) + \kappa N}^3. \tag{36}
\]

Therefore, if \(N\) has a minimum or maximum when \(\alpha(q) \in [\kappa/3, \infty]\), then it is unique. At this point, the derivation of the equilibrium must be broken into cases.

Case 1  If \(\frac{\partial^2 N}{\partial \alpha(q)^2} < 0\) and \(N > 0\) when \(q = 1\), then the only steady-state equilibrium is when \(q^* = 1\). This is because there doesn’t exist an \(N = 0\) for any \(\alpha(q) \in [\kappa/3, \alpha(1)]\) as \(N\) has a unique maximum and \(N > 0\) when \(\alpha(q) = \kappa/3\) and \(q = 1\).

Case 2  If \(\frac{\partial^2 N}{\partial \alpha(q)^2} < 0\) and \(N < 0\) when \(q = 1\), then the only steady-state equilibrium is when \(N = 0\). This is because \(N\) is positive when \(\alpha(q) = \kappa/3\), is continuous, and has a unique maximum. Using the quadratic formula, the equilibrium \(\alpha(q^*)\) is determined by (19) and \(q^*\) from (34).
Case 3  If $\frac{\partial^2 N}{\partial \alpha(q)^2} > 0$ and $N > 0$ when $q = 1$, then two steady-state equilibrium may exist. First, a corner equilibrium where $q^* = 1$. Second, a low interior where $N = 0$. This is the only interior equilibrium because $N$ has only two non-trivial roots. $N$ has only two roots because it is positive when $\alpha(q) = \kappa \pi / t$, is continuous, and has a unique minimum. Only the minimum of the two roots is an equilibrium because the larger root is not stable (agents would choose to enter). Using the quadratic formula, the equilibrium $\alpha(q^*)$ is determined by (18) and $q^*$ from (34).

Case 4  If $\frac{\partial^2 N}{\partial \alpha(q)^2} > 0$ and $N < 0$ when $q = 1$, then the only steady-state equilibrium is when $N = 0$. This is because $N$ is positive when $\alpha(q) = \kappa \pi / t$, is continuous, and has a unique minimum. Using the quadratic formula, the equilibrium $\alpha(q^*)$ is determined by (18) and $q^*$ from (34).

Proof of Proposition 3  Assuming a low interior equilibrium implies participation rises because

$$\frac{\partial N}{\partial \gamma} = -\frac{\alpha(q) \pi}{\alpha(q) \pi (r + \delta) + \kappa N} < 0. \quad (37)$$

Therefore, if $\gamma$ falls, then $N$ rises and in equilibrium $\alpha(q^*)$ must increase for $N$ to equal zero. In turn, if $\alpha(q^*)$ rises, then participation $q^*$ must rise. Inspecting (22), one can see welfare necessarily falls because participation is rising. Finally, the proportion (and number) of positive agents rises as $\alpha(q^*)$ rises. This can be seen indirectly from (15).

Proof of Proposition 4  Assuming a low interior equilibrium implies participation rises because

$$\frac{\partial rN}{\partial t} = -\alpha(q)\kappa N \frac{r \gamma + \delta \alpha(q) b}{((\alpha(q) t - \kappa \pi)(\delta + r) + \kappa N)^2} < 0. \quad (38)$$
Therefore, if $t$ falls, then $N$ rises and in equilibrium $\alpha(q^*)$ must increase for $N$ to equal zero. In turn, if $\alpha(q^*)$ rises, then participation $q^*$ must rise. Inspecting (22), one can see welfare falls if and only if $q^*$ rises faster than $t$. Finally, the proportion (and number) of positive agents is rising if the increase in $\alpha(q^*)$ rises faster than the fall in $t$. This can be seen indirectly from (15).

**Proof of Proposition 5**  Assuming a low interior equilibrium, it can be shown

$$\frac{\partial rN}{\partial \rho} > 0$$

(39)

after some substantial algebra which is omitted here. Therefore, if $\rho$ rises, then $N$ rises and in equilibrium $\alpha(q^*)$ must increase for $N$ to equal zero. In turn, if $\alpha(q^*)$ rises, then participation $q^*$ must rise. The proportion (and number) of positive agents is rising if the increase in $\alpha(q^*)$ rises sufficiently fast. The precise change must satisfy (25) which is deduced by solving for when $\frac{\partial n}{\partial \rho} < 0$ where $n$ is determined by (15).

**Proof of Proposition 6**  Assuming a low interior equilibrium, it can be shown

$$\frac{\partial rN}{\partial \lambda} > 0$$

(40)

after some substantial algebra which is omitted here. Therefore, if $\lambda$ rises, then $N$ rises and in equilibrium $\alpha(q^*)$ must increase for $N$ to equal zero. In turn, if $\alpha(q^*)$ rises, then participation $q^*$ must rise. The proportion (and number) of positive agents is rising if the increase in $\alpha(q^*)$ rises sufficiently fast. The precise change must satisfy (25) which is deduced by solving for when $\frac{\partial n}{\partial \lambda} < 0$ where $n$ is determined by (15).
Proof of Proposition 8  The value of entering the negative market for a negative agent, if it exists, is

\[ rN_n = \alpha(1) \left( b - \frac{t}{\alpha(1)T} \left( \frac{\lambda + \delta}{\lambda + \rho + \delta} \right) \left( 1 - \frac{\rho + \delta}{\alpha(1)T} \right) \left( \alpha(1)\delta + r\gamma \right) \right) - \tau, \]  

(41)
as \[ \tau = t \left( \frac{\lambda + \delta}{\lambda + \rho + \delta} \right) \left( 1 - \frac{\rho + \delta}{\alpha(1)T} \right) \] while the value of entering the positive market is

\[ rN_p = \alpha(1) \left( b - \frac{t}{\alpha(1)T} \left( \frac{\alpha(1)\delta + r\gamma}{\alpha(1)T} \right) \right), \]  

(42)as \[ \tau = t. \] Therefore, a negative agent will enter the negative market if \( N_p \leq N_n \). This is true if (28) holds. Also, agents will search in the centralized market if \( 0 \leq rN_n \) which is true if (27) holds. As for \( \tau > 0 \), note the value of the positive agent is identical in either market as the matching rate is the same. Therefore, \( \tau \) need only be greater than zero. ■

Proof of Proposition 9  For part 1 of the proposition, the result follows directly from the fact (28) is satisfied. If so, agents separate and no interaction occurs between negative and positive agents.

To see part 2 of the proposition, it is necessary to note \( p_1 = \left( \frac{\lambda + \delta}{\lambda + \rho + \delta} \right) \left( 1 - \frac{\rho + \delta}{\alpha(1)T} \right) \) in the separated market. Therefore, if \( \rho + \delta > \alpha(1)T \), then \( p_1 \) goes to zero and positive agents do not mix with negative agents. Part 3 of the proposition is true because the fraction of infected individuals equals

\[ 1 - \frac{(\lambda + \delta)(\rho + \delta)}{\alpha(1)T(\rho + \alpha + \delta)} \] prior to the separating equilibrium while it becomes \( 1 - \frac{\rho + \delta}{\alpha(1)T} \) afterward. Similarly for part 4, the change in the proportion of infected individuals can be deduced by realizing it is

\[ 1 - \frac{(\lambda + \delta)(\rho + \delta)}{\alpha(q^*)T(\rho + \alpha + \delta)} \] prior to the separating equilibrium given an interior equilibrium. ■

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References


SF Morin, SB Shade, WT Steward, AW Carrico, RH Remien, MJ Rotheram-Borus, JA Kelly,


Figure 1: Search Decision
Figure 2: Search Decision
Table 1: Variables used in model

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tr>
<td>$q$</td>
<td>participation rate</td>
</tr>
<tr>
<td>$\alpha(q)$</td>
<td>matching rate</td>
</tr>
<tr>
<td>$b$</td>
<td>utility matching</td>
</tr>
<tr>
<td>$t$</td>
<td>transmission probability</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>disutility from infection</td>
</tr>
<tr>
<td>$\rho$</td>
<td>rate of diagnoses</td>
</tr>
<tr>
<td>$\delta$</td>
<td>rate of mortality</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>rate of treatment</td>
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<tr>
<td>$r$</td>
<td>discount rate</td>
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Table 2: Parameters of MSM

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<td>$b$</td>
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<td>utility from a match</td>
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<tr>
<td>$t$</td>
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<tr>
<td>$\rho$</td>
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<tr>
<td>$\delta$</td>
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<tr>
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Table 3: Effects of Reducing the Cost of Infection ($\gamma$)

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<th>1.36</th>
<th>1.44</th>
<th>1.51</th>
<th>1.58</th>
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<tbody>
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<td>Sexually Active (%)</td>
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<td>45.3</td>
<td>42.3</td>
<td>40.1</td>
<td>38.5</td>
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</table>
| Unknowingly Positive (%) | 3.5 | 3.2 | 3 | 2.8 | 2.6 |}
| Knowingly Positive (%) | 52.8 | 47.9 | 44.3 | 41.4 | 38.9 |
| Negative (%) | 43.7 | 48.9 | 52.7 | 55.8 | 58.5 |
| Change in Welfare (%) | -18.3 | -7.1 | —    | 5.1  | 9    |
Table 4: Effects of Changing the Transmission Probability ($t$)

<table>
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<tr>
<th>$t$ (%)</th>
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<td>2.6</td>
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<td>52.8</td>
<td>47.9</td>
<td>44.3</td>
<td>41.4</td>
<td>38.9</td>
</tr>
<tr>
<td>Negative (%)</td>
<td>43.7</td>
<td>48.9</td>
<td>52.7</td>
<td>55.8</td>
<td>58.5</td>
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<tr>
<td>Change in Welfare (%)</td>
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<td>-18</td>
<td>—</td>
<td>13.5</td>
<td>24.2</td>
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Table 5: Effects of Increasing Detection ($\rho$)

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<th>0.43</th>
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<td>42.3</td>
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<td>2.97</td>
<td>2.83</td>
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<td>44.3</td>
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<tr>
<td>Negative (%)</td>
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<td>52.7</td>
<td>52.7</td>
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<td>52.7</td>
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<tr>
<td>Change in Welfare (%)</td>
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<td>0</td>
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Table 6: Effects of Changing the Treatment Rate ($\lambda$)

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<td>67.8</td>
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<td>47.8</td>
<td>53.4</td>
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<td>48.4</td>
<td>41.7</td>
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<td>-57.4</td>
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Table 7: Effects of Changing the Survival Rate ($\delta$)

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<th>0.025</th>
<th>0.026</th>
<th>0.028</th>
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<td>45.3</td>
<td>46.4</td>
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<td>-13.9</td>
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Table 8: Effects from Directed Search

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<tr>
<td>Matching rate</td>
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<td>0.63 2.5 5.0</td>
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<td>Sexual Participation (%)</td>
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<tr>
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<td>0.0   1.3   3.8</td>
</tr>
<tr>
<td>Knowingly Positive (%)</td>
<td>44.3</td>
<td>0.0    19.7  56.7</td>
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<tr>
<td>Negative (%)</td>
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<td>100.0  79.0  39.5</td>
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<td>Welfare $\times r$</td>
<td>-0.19</td>
<td>(0,0.63) (0,1.30) (0,1.37)</td>
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<td>Barrier to entry</td>
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<td>(0,0.63) (0,1.30) (0,1.37)</td>
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Table 9: Effects of Linking Health and Search Intensity

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<thead>
<tr>
<th></th>
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<th>2.8</th>
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<td>1</td>
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<td>-33</td>
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### Table 10: Effects from Multiple-Norms

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<td>Type B</td>
<td>Type A</td>
<td>Type B</td>
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<tr>
<td>Transmission Rates</td>
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<td>0.21</td>
<td>0.3</td>
<td>0.3</td>
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<tr>
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<td>34.9</td>
<td>38.2</td>
<td>19.2</td>
<td>22</td>
<td>22</td>
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<td>1.9</td>
<td>1.9</td>
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<td>44.3</td>
<td>44.3</td>
<td>28.6</td>
<td>28.6</td>
<td>28.6</td>
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<td>52.7</td>
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