

The effects of prevention and treatment interventions in a microeconomic  
model of HIV transmission

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## **Abstract**

A rational choice-based model for sexual transmission of HIV demonstrates behavioral and epidemiological effects of public health interventions. Susceptible individuals choose to protect or expose, both responding to and determining HIV prevalence. Interventions are modeled as exogenous shocks to the cost of protection, treatment coverage, and treatment quality. A prevention intervention improves social welfare through decreasing HIV prevalence, incidence, and HIV-related deaths in steady-state equilibrium. A treatment intervention improves social welfare if epidemiological effects outweigh behavioral effects. However, treatment interventions also increase the elasticity of behavior change with respect to the cost of protection. Therefore, a prevention intervention is more effective when infected individuals are better off. Complementary effects between different types of interventions may be important for determining an optimal public health response to HIV.

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## **I. Introduction**

The human immunodeficiency virus (HIV) epidemic emerged more than thirty years ago, and is still a pressing public health issue today. An estimated 34 million people were living with HIV in 2010, and millions will develop Acquired Immune Deficiency Syndrome (AIDS), the suite of diseases that HIV infection culminates to, without receiving life-saving antiretroviral (ARV) treatment (UNAIDS, 2010). In the absence of a cure or vaccine for HIV, public health policy-makers must strike a balance between preventing new infections and caring for those already infected. However, both prevention and treatment interventions may affect HIV-related risk behaviors. A rational choice-based model for sexual transmission of HIV that illustrates how prevention and treatment interventions affect individual risk behaviors can provide insight into the optimal public health response to HIV.

Incorporating rational behavior into an epidemiological model leads to different predictions from the standard mathematical approach. In a mathematical model of infectious disease, the rate of new infections (incidence) increases with the proportion of individuals who are infected (prevalence). In contrast, economic models predict that as prevalence increases, susceptible individuals respond to the higher risk of infection by adopting more protective behavior, thus limiting the rate of new infections. In other words, economic models of epidemics allow individual decision-making to both respond to and determine disease dynamics (Philipson, 2010). Sexually transmitted diseases, such as HIV, are candidates for economic modeling because exposure may be avoided through protective behavior. The dynamic demand for protection with respect to HIV prevalence has important policy implications. For example, a permanent change in the cost of

protection can alter incentives for risky behavior and lead to lower long-run disease prevalence.

Several leading papers in economic epidemiology apply a choice-based approach to models of HIV transmission (Philipson & Posner, 1993; Kremer, 1996; Geoffard & Philipson, 1996). However, public health and medical innovations have changed the nature of the HIV epidemic and the decision frameworks that are characterized in these existing models. First, education and rapid HIV testing technology has improved information of HIV risk and health status. Second, HIV prevention programs have directly affected risk behaviors through distributing contraceptives, especially male condoms, and through changing social norms and attitudes surrounding sexual behaviors. Third, and most importantly, highly active antiretroviral therapy (HAART), a treatment regimen that combines several classes of ARVs, has transformed the wellbeing and mortality rate of people living with HIV. Moreover, evidence from a recent clinical trial supports previous observations that individuals who receive HAART are less likely to transmit the virus to others (Cohen et al., 2011). The preventative effect of ARVs raises new questions about how treatment for HIV can affect risk behaviors. These new dimensions of the HIV epidemic call for an updated economic interpretation of HIV-related risk behaviors.

The aim of this paper is to incorporate representations of prevention and treatment interventions into a rational choice-based model of HIV transmission in order to illustrate their behavioral and epidemiological effects. Susceptible individuals may choose to use costly protection in order to avoid risking exposure to HIV. In maximizing lifetime expected utility, an individual's risk behavior will respond to changes in HIV prevalence

as well as health interventions. HIV prevalence is an endogenous and dynamic risk factor determined by the rate of susceptible individuals who choose protection. As HIV prevalence rises, the rate of risky behavior and the corresponding number of new infections decline such that the epidemic reaches a steady-state equilibrium.

Health interventions are modeled as exogenous shocks; a change in the cost of protection represents a prevention intervention, while changes in the coverage or effectiveness of ARVs represent treatment interventions. In addition to these two types of interventions, changes to underlying health may have behavioral effects if they favor susceptible or infected individuals. Finally, knowledge of the preventative effects of treatment will change behavior and HIV-related outcomes.

A prevention intervention reduces the rate of risky behavior so that HIV prevalence is lower in steady-state equilibrium. Alternatively, a treatment intervention incentivizes risky behavior of forward-looking susceptible individuals. However, treatment also increases the survival rate of infected individuals. A treatment intervention may improve social welfare depending on the opposing behavioral and epidemiological effects. Additionally, any improvement in the welfare of infected individuals through a treatment intervention has a complementary effect on prevention interventions. The behavioral effects of an intervention and the complementary effects between treatment and prevention interventions are larger when treatment has known preventative effects.

Results suggest that a lack of coordination between prevention and treatment interventions may lead to sub-optimal public health strategies. Future research of behavioral responses to HIV interventions should analyze public health strategies holistically in order to avoid overlooking possible complementary effects.

The remainder of the paper is organized as follows: section II provides a brief update on the HIV epidemic and the public health response in addition to a review of economic contributions to HIV modeling; section III describes the framework for a model of sexual transmission of HIV with dynamic demand for protection; section IV analyzes the effects of exogenous health interventions; section V incorporates the preventative effects of treatment to the model; and section VI concludes with a discussion of the results.

## **II. Literature Review**

### *The state of the HIV epidemic*

The spread of HIV has slowed in all but a few countries; the estimated 2.7 million new infections in 2010 was one fifth of the rate of new infections at the peak of the epidemic in 1999 (UNAIDS, 2010). However, the stabilization of the epidemic does not diminish the scale of the public health challenges still ahead. Forty-nine countries have generalized epidemics, where adult HIV prevalence is over 1%, and nine countries in southern Africa have adult HIV prevalence over 10% (Ibid.).

Most new infections are caused through sexual transmission (UNAIDS, 2010), underscoring the importance of prevention efforts that target sexual risk behaviors. Behaviors surrounding condom use, multiple partners, and age at sexual debut are the most common targets of prevention interventions. Often cited examples of successful programs are Thailand's "100% condom program" which mandated condom use in the commercial sex industry, and Uganda's "zero grazing" campaign which emphasized abstinence and being faithful to a single partner in addition to condom use (Hearst & Chen, 2004). Although HIV prevention efforts have curbed the growth of the epidemic,

there is opportunity for improvement through increasing access to female-initiated forms of protection and more engagement with high-risk populations (UNAIDS, 2010).

In North America and Europe, AIDS-related mortality declined immediately after the introduction of the first antiretroviral drugs in 1996. Though there is still no cure for HIV, pharmaceutical innovations have drastically improved health for those living with HIV. However, access to drugs has been slow to reach many parts of the world that have the highest burden of disease; the corresponding decline in AIDS-related mortality in sub-Saharan Africa did not occur until 2005 (UNAIDS, 2010).

In addition to improving the life expectancy and wellbeing of individuals living with HIV, ARV treatment was shown to decrease an infected individual's likelihood of transmitting the virus in a recent randomized control (Cohen et al., 2011). The idea that treatment can act as prevention is not new, and many have promoted the strategy of accelerating ARV coverage as the most promising way to control the HIV epidemic (Lima et al., 2008; Montaner et al., 2006). However, others express concern that the private and public benefits of HAART may be offset by an increase in risky behavior, eventually leading to a resurgent HIV epidemic (Bezemer, 2008; Dieffenbach & Fauci, 2009). The behavioral responses to treatment will be of critical importance as more information on the preventative effects of HAART accumulates.

### *Economic models of HIV epidemics*

Incorporating rational choice into a model of infectious disease leads to different predictions compared to traditional epidemiological models. The key departure of an economic model from a mathematical model is that higher disease prevalence is



associated with a lower rate of new infections because individuals adopt more protective behavior. Whereas HIV prevalence increases indefinitely in a mathematical model, HIV prevalence is limited by prevalence-elastic risk behavior in a model based on rational choice (Philipson, 2000).<sup>2</sup>

Three leading studies in economic epidemiology model HIV transmission and are particularly relevant to this paper. In a model where individuals optimize the rate of partner change, Kremer (1996) shows that an imperfect vaccine may cause an increase in HIV prevalence. Often, economic theory applied in epidemiology focuses on vaccination because it is a discrete health input that has important policy implications (see for example Bauch & Earn, 2004). In Kremer's model, the perception that a vaccine lowers HIV risk may increase the rate of partner change such that the number of new infections outweighs those avoided from the vaccine. Kremer also compares the behaviors of high-risk and low-risk populations. Among the high-risk population, the marginal probability of infection from an additional partner is smaller or possibly negative. As HIV prevalence increases, a high-risk individual is more likely to already be infected may behave fatalistically, or increase the rate of partner change. Kremer concludes that the timing of interventions and the targeting high-risk groups are critical for effective HIV prevention efforts.

Philipson and Posner (1993) formalized the tradeoff between HIV risk and the choice of unprotected and protected sex. Geoffard and Philipson (1996) extend this model and show that the demand for a protective behavior, such as condom use, increases

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<sup>2</sup> A discussion of mathematical models, including the SI (susceptible-infected) compartmental model that represents HIV, is found in Anderson and May (1991). Philipson (2000) summarizes the distinctions between economic and mathematical epidemiology, and Funk et al. (2010) reviews different categories of behavioral models of infectious diseases.

in response to rising HIV prevalence and leads to a steady state prevalence. A government can improve welfare by introducing a permanent change to the incentives for protection (lowering costs) so that susceptible individuals choose to use protection at a lower HIV prevalence threshold. They also stress the importance of timing; a prevention intervention may be “too late” to have any effect on behavior if HIV prevalence is past a certain threshold (Philipson, 1996). However, in contrast to Kremer’s emphasis on targeting high-risk populations, Philipson’s model suggests that susceptible individuals who face the highest consequence of becoming infected are more responsive to prevention interventions than individuals with high risk.

In both models, the policy implications for targeting and timing of prevention depend on the assumption that individuals are unaware of their HIV status. It is important to note that informational aspects of a disease, in particular the time it takes for symptoms to appear, affects disease dynamics in economic models and not in mathematical models (Philipson, 2000). However, the informational aspects of HIV have changed since the first economic models of HIV were created. Thanks to rapid-testing technology, voluntary counseling and testing centers, opt-out testing at primary healthcare services, and educational campaigns, the average time from infection to diagnosis has decreased significantly (UNAIDS, 2010). Chen (2008) examines the effects of prevention interventions with varying degrees of health information in an extension of Geoffard and Philipson’s model, and finds that more universal knowledge of HIV status decreases HIV prevalence. In general, the simplifying assumption that all infected individuals are aware of their health status may be more representative of current epidemics than the assumption that no one knows their status.

In a discrete-time model for the rate of partners and HIV, Auld (2001) finds that pessimistic beliefs about the epidemic in the future may increase risky behaviors in the present time period. Mannberg (2011) introduces the possibility of exogenous stochastic health shocks in a model of safe and unsafe sex in order to determine the effect of health uncertainty on risky sexual behavior. In situations with high uncertainty about future health, individuals are less likely to invest in protection and HIV prevalence increases. Mannberg includes access to ARV treatment as one dimension of uncertainty, and concludes that low access to treatment in addition to poor underlying health help to explain the persistence of high-risk sexual behaviors in generalized epidemics of HIV. Poor underlying health may also be important in explaining HIV-risk behavior according to theories of competing risks in health (Dow, Sala, & Sala-i-Martin, 1999). If an investment in protection from HIV does not improve a more immediate health risk, then HIV risk behaviors may not respond to interventions until the immediate health risks are addressed. Taken together, treatment for HIV and future healthcare in general may be important to the success of HIV prevention interventions.

The structure of the model that follows is similar to that of Geoffard & Philipson (1996) and Mannberg (2011) in that the rate of new infections is determined by an individual's decision to have safe or unsafe sex. However, this model takes a closer look at the effects of changes to welfare in the infected state by including both treatment coverage and quality, as well as three different survival rates for susceptible, infected, and treated individuals. Finally, the analysis of the model focuses on treatment and prevention interventions that represent the two main arms of current policy options, rather than on the effects of a hypothetical HIV vaccine.

### III. The Model

In this section, a model for the spread of HIV through sexual transmission depends on the behavior of rational individuals who are susceptible to infection. HIV prevalence is both endogenous to sexual risk-behaviors and a result of the decision to protect or expose.

There are two health states in the model, susceptible and infected. Once an individual becomes infected, they cannot return to the susceptible state. However, an individual's level of health in the infected state depends on whether or not they receive treatment. A government or agency provides treatment for a constant proportion of infected individuals. In each time period  $t$ , individuals randomly match with a sexual partner. An individual is aware of their own health state and the overall HIV prevalence in the population,  $\alpha$ , but is unaware of their partner's health state. Of the total population,  $N=1$ , a proportion  $\alpha$  are infected with HIV in the initial time period ( $\alpha(t_0)>0$ ).

A susceptible individual can avoid exposure to HIV by choosing to use a costly form of protection, for example a condom.<sup>3</sup> The decision to protect or expose depends on the tradeoff between the cost of protection and the risk of becoming infected. There are two dimensions to this risk: the likelihood and the consequence of becoming infected.

The likelihood of a susceptible individual becoming infected depends on the HIV prevalence and transmission rates. A higher HIV prevalence increases the chance that a susceptible individual matches with an infected individual. If a susceptible and infected individual do match, the transmission rate is the probability that a new infection occurs.

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<sup>3</sup> Male condoms are the most widely accessible and effective form of protection from sexual transmission of HIV. In many cases, condoms are distributed for free, but there still may be other transactional or social costs, and sex with a condom is often valued less, as documented in studies of commercial sex in India (Rao et al., 2003). The model also would apply to less common forms of protection such as female condoms and antiretroviral microbial gels (in clinical trials).

The transmission rate is lower if a susceptible individual uses protection or if an infected individual receives treatment. Therefore, the decision to protect or expose also determines the rate of new infections, and HIV prevalence will be a dynamic risk factor. In contrast, the consequence of becoming infected in this model is a static risk factor. Welfare in the infected state depends on the exogenous availability and quality of ARV treatment. In addition, other health inputs that are unrelated to HIV may affect susceptible and infected individuals differently. These exogenous determinants of the welfare gap between infected and susceptible individuals will affect risk-behavior and HIV prevalence.

In order to elicit how the preventative effects of treatment affect risk-taking behavior and HIV prevalence, the model will be analyzed in two stages: first with a natural transmission rate between infected and susceptible partners; and second with a transmission rate that is tied to treatment coverage. Comparative analysis of exogenous shocks to prevention costs, treatment coverage, treatment quality, and underlying health will illustrate how HIV-related health interventions affect risk taking behaviors and HIV prevalence.

### **Susceptible individual value function**

The first task is to model a representative susceptible individual's decision to protect against HIV infection. In each time period, a forward-looking susceptible individual can pay a cost  $c$  to prevent infection from a sexual partner with unknown health status, or they can risk exposure. Assuming there are no altruistic incentives, infected individuals do not use protection. Therefore, an individual that chooses not to use protection may be more likely to be infected. Observing a partner's decision before

choosing to expose or protect could reveal information about the partner's health status and affect risk behavior. Schroeder and Rojas (2002) offer a game theoretic model of sexual risk behavior that takes health status signaling into consideration. Here, a simplifying assumption is made so that a susceptible individual decides whether to protect or expose at the beginning of each time period, before matching with a sexual partner.

In each time period  $t$ , a susceptible individual maximizes the value function  $V_s$  with the control variable  $a=\{\text{protect, expose}\}$  given the following exogenous parameters: the full cost of using protection,  $c$ ; utility per time period,  $u$ ; HIV prevalence in the population,  $\alpha$ ; transmission rate,  $\beta$ ; and survival rates in susceptible and infected health states,  $p_s$  and  $p_i$ . This fits into a discrete-choice dynamic programming framework, where the susceptible individual maximizes utility in the current time period based on the probability of transitioning to the infected state in the next period. The condition for optimality is clear when expressed as a Bellman equation below<sup>4</sup>:

$$V_{s_t} = \max \{ u - c + p_s V_{s_{t+1}}, u + \alpha_t \beta p_i V_{i_{t+1}} + (1 - \alpha_t \beta) p_s V_{s_{t+1}} \}$$

The left side of the Bellman equation represents the decision to protect; if the individual protects then they gain a net utility  $u-c > 0$  and remain susceptible in the next time period. The next period's utility is discounted by the susceptible individual's survival rate,  $p_s \in [0,1]$ .

On the right side of the Bellman equation, the decision to expose yields utility  $u$  in time  $t$ , and introduces the risk of contracting HIV. A new infection occurs with

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<sup>4</sup> See Bertsekas (1995) for an overview of sequential decision making under uncertainty, or the original work of Bellman (1957) on dynamic programming.

probability  $\beta$  when a susceptible individual chooses expose and matches with an infected individual. If a susceptible individual becomes infected, they survive to the next time period with probability  $p_i < p_s$ , and experience utility  $V_i$  for the remaining time periods.

This equation requires several simplifying assumptions. First, protection is one hundred percent effective at preventing HIV, or the transmission rate is zero with protected sex. Measuring transmission rates is difficult because it requires data on sexual behavior before HIV infection occurs. In a study of serodiscordant couples in Uganda, the overall probability of transmission through heterosexual contact was estimated at 0.0011 (Gray et al., 2001). Condom use is estimated to reduce transmission rates by 80% (Weller & Davis-Beaty, 2007).

Second, both partners have the ability to choose to use protection. Realistically, the power to choose or negotiate safe sex may not be shared equally between sexual partners. The question of agency, and specifically how gender disparities may affect sex-related risk behaviors, will be discussed further in section VI.

Third, HIV infection leads to an immediate drop in survival rate. In reality, the different phases of HIV infection have distinct health effects; an acute phase of flu-like symptoms is followed by a prolonged asymptomatic period and finally a rise in opportunistic infections that cause AIDS and death, if untreated. However, a simplified representation of the loss of health is not expected to change the qualitative results of this model, since susceptible individuals make risk-behavior decisions based on lifetime expected utility, effectively smoothing the health effects of infection.

Fourth, all individuals are aware of their health status. Although improved technology and education has increased the accessibility and usage of HIV testing,

unrecognized HIV infection is a persistent problem and it is possible for people to remain unaware that they are infected with HIV for several years after contracting the virus.

Within the framework of this model, one could interpret the length of a time period as the time an individual takes to realize their health status. Alternatively, if the realization of a transition from the susceptible to infected health state were delayed by a length of time, then infected individuals may act as if they were susceptible and choose to protect.

Therefore, imperfect knowledge of HIV status would possibly decrease the rate of new infections. This counterintuitive result is dependent on the absence of any altruistic incentives.

Returning to the Bellman equation for the susceptible value function, if  $\alpha=0$ , susceptible individuals choose expose, avoiding cost  $c$  without taking on any risk of lower lifetime expected utility. In order to observe how HIV prevalence evolves, the initial HIV prevalence is restricted so that susceptible individuals also choose expose at the initial HIV prevalence:

$$\alpha_{t_0} < \frac{c}{\beta(p_s V_s - p_i V_i)}$$

A susceptible individual will continue to choose expose as HIV prevalence increases, until HIV prevalence reaches a critical value,  $\alpha^*$ , where the individual is indifferent between choosing to protect or expose. Above this threshold, when  $\alpha_t > \alpha^*$ , the risk of infection outweighs the cost of protection, all susceptible individuals choose protect, and no new infections occur. Therefore, HIV prevalence will be constant at  $\alpha^*$  when susceptible individuals choose to expose at a rate such that new infections equal deaths from HIV ( $\alpha_t = \alpha_{t+1} = \alpha^*$ ).



### Infected individual value function

Unlike the susceptible value function, utility in the infected state,  $V_i$ , is independent of HIV prevalence and is static. In each period, all infected individuals gain a net utility  $w$  and a constant proportion  $k \in [0,1]$  receive ARV treatment. An infected individual who receives treatment survives at a higher probability rate,  $p_r$ . With probability  $(1-k)$ , an infected individual receives no treatment and survives at a rate  $p_i$ :

$$V_{i_t} = w + kp_r V_{i_{t+1}} + (1 - k)p_i V_{i_{t+1}}$$

The infected value function implies that ARV drug supply is perfectly elastic, and that treatment is provided at no cost to the infected individual. This represents the case where ARV treatment is supplied by a government or public health agency.

Both utility and survival rates are health state-dependent and restricted such that lifetime expected utility in the susceptible state is greater than lifetime expected utility in the infected state:  $0 < w < (u - c)$  and  $0 < p_i < p_r < p_s < 1$ .

### Growth of HIV prevalence

The growth of HIV prevalence over time depends on survival rates of infected individuals, the proportion of infected individuals receiving treatment, the transmission rate of HIV, and the rate of exposure among susceptible individuals,  $\phi$ . A new infection occurs if a susceptible individual matches with an infected individual, chooses expose, and if the virus transmits. New infections and infected individuals without treatment survive at the rate  $p_i$ , while  $k\alpha$  infected individuals survive at the rate  $p_r$ . The growth equation for HIV prevalence follows:

$$\alpha_{t+1} = (kp_r + (1 - k)p_i)\alpha_t + p_i\phi_t\beta(1 - \alpha_t)\alpha_t$$

When  $\alpha_t = \alpha_{t+1}$ , HIV prevalence is constant and the system is said to be in epidemiological equilibrium. In order for this to occur, individuals expose at a rate  $\phi^*$  such that the amount of new infections per period, or the incidence of HIV, is equal to the deaths of infected individuals in the same time period. Setting  $\alpha_t = \alpha_{t+1} = \alpha^*$ :

$$\phi^* = \frac{1 - p_i - k(p_r - p_i)}{p_i \beta (1 - \alpha^*)}$$

Recall that when  $\alpha < \alpha^*$  all individuals expose ( $\phi=1$ ), and when  $\alpha > \alpha^*$  all individuals protect ( $\phi=0$ ). Epidemiological equilibrium,  $\phi^*$ , only occurs when  $\alpha$  satisfies the susceptible individual's indifference condition. The next step is to solve for  $\alpha^*$  in terms of the parameters of susceptible and infected state value functions.

### **Behavioral Equilibrium**

Behavioral equilibrium occurs at  $\alpha^*$  when a susceptible individual is indifferent between the choices to protect or expose:

$$u - c + p_s V_{s_t} = u + \alpha_t^* \beta p_i V_{i_t} + (1 - \alpha_t^* \beta) p_s V_{s_t}$$

Given that in behavioral equilibrium  $V_{i_{t+1}} = V_{i_t}$  and  $V_{s_{t+1}} = V_{s_t}$ , the indifference condition can be solved explicitly by substituting the following values for susceptible and infected utility:

$$V_s = \frac{u - c}{1 - p_s}$$

$$V_i = \frac{w - d}{1 - p_i - k(p_r - p_i)}$$

After rearranging, the indifference condition yields HIV prevalence in behavioral equilibrium:

$$\alpha^* = \frac{c}{\beta \left( \frac{p_s}{1-p_s} (u - c) - \frac{p_i}{1-p_i-k(p_r-p_i)} w \right)}$$

### Steady-state equilibrium

A steady-state equilibrium occurs when the rate of exposure is in equilibrium ( $\phi_t = \phi_{t+1} = \phi^*$ ) given HIV prevalence, and when HIV prevalence is in equilibrium ( $\alpha_t = \alpha_{t+1} = \alpha^*$ ) given the rate of exposure (as in Kremer, 1996). In other words, steady-state equilibrium requires both epidemiological and behavioral equilibrium.

Substituting  $\alpha^*$  into the condition for epidemiological equilibrium yields an expression for  $\phi^*$  in terms of exogenous parameters:

$$\phi^* = \left( \frac{1 - p_i - k(p_r - p_i)}{p_i} \right) \left( \beta - \frac{c}{\left( \frac{p_s}{1-p_s} (u - c) - \frac{p_i}{1-p_i-k(p_r-p_i)} (w - d) \right)} \right)^{-1}$$

For further analysis, it is useful to have notation for the survival odds ratios in each state,  $\sigma_s = p_s / (1 - p_s)$  and  $\sigma_i = p_i / (1 - p_i - k(p_r - p_i))$ , and for the lifetime expected welfare differential between susceptible and infected states,  $X = \sigma_s(u - c) - \sigma_i w > 0$ . HIV prevalence in steady-state equilibrium ( $\alpha^*$ ) is equal to the ratio of the cost of protection ( $c$ ) to the risk of infection, where risk is equal to the product of the likelihood of contracting the virus from an infected individual ( $\beta$ ) and the lifetime expected loss in welfare ( $X$ ). Exposure rate in steady state equilibrium ( $\phi^*$ ) is equal to the ratio of the odds of death

among infected individuals ( $1/\sigma_i$ ) to the rate of new infections among susceptible individuals ( $\beta(1-\alpha^*)$ ).

To summarize, there are two conditions for a steady-state equilibrium,  $(\alpha^*, \phi^*)$ :

$$\begin{aligned} 1) \quad \alpha^* &= \frac{c}{\beta X} \\ 2) \quad \phi^* &= \frac{1}{\sigma_i \beta (1 - \alpha^*)} \end{aligned}$$

#### IV. HIV interventions

##### HIV interventions and welfare analysis

Governments and health agencies seek to improve social welfare with health interventions that lower HIV prevalence, HIV incidence (number of new infections per time period), and HIV-related death rates. In equilibrium, a lower exposure rate  $\phi^*$  indicates that fewer new infections are needed to replace HIV-related deaths. In other words, for any given level of HIV prevalence, as  $\phi^*$  decreases, a larger proportion of infected individuals are survivors from the previous period and a smaller proportion are new infections. Therefore, an intervention that decreases both  $\alpha^*$  (HIV prevalence) and  $\phi^*$  (HIV incidence, and death rate) will improve social welfare, an intervention that raises  $\alpha^*$  and  $\phi^*$  will worsen social welfare, and the impact of an intervention that has opposing effects on  $\alpha^*$  and  $\phi^*$  will depend on the welfare function  $W$ :

$$W = f(\alpha^*, \phi^*) \quad \frac{\partial W}{\partial \alpha^*} < 0 \quad \frac{\partial W}{\partial \phi^*} < 0$$

Public health interventions are modeled as exogenous shocks to certain parameters. A drop in  $c$  represents a prevention intervention, a rise in  $k$  (treatment

coverage) and a rise in  $p_r$  (treatment quality) represent two types of treatment interventions. Finally, a change in  $\sigma_s$  or  $\sigma_i$  represent changes to underlying health that affect susceptible and infected populations differently.

Parameters that affect  $\alpha^*$  are said to have a behavioral effect, and parameters that affect  $\phi^*$  independent of  $\alpha$ , are said to have an epidemiological effect on steady-state equilibrium. Since  $\phi^*$  is a function of  $\alpha^*$ , the behavioral and epidemiological effects together determine the net effect of an intervention on  $\phi^*$ . Table 1 Summarizes the existence and direction of behavioral and epidemiological effects as well as the total effects on  $\alpha^*$  and  $\phi^*$  for each type of intervention.

*Table 1. Summary of behavioral and epidemiological effects of exogenous shocks to steady-state equilibrium*

Parameter ( $x$ )	Behavioral Effect		Epidemiologic al Effect		$\partial\alpha^*/\partial(x)$	$\partial\phi^*/\partial(x)$
$c$	yes	+	no		+	+
$k$	yes	+	yes	-	+	+/-
$p_r$	yes	+	yes	-	+	+/-
$\sigma_i$	yes	+	yes	-	+	+/-
$\sigma_s$	yes	-	no		-	-

### Prevention intervention

The aim of a prevention intervention is to lower  $c$ , either through procurement and distribution of contraceptives, or through decreasing non-monetary costs and social barriers to choosing protection. The cost of protection affects the tradeoff that susceptible individuals face, and that determines  $\alpha^*$ :

$$\frac{\partial\alpha^*}{\partial c} = \frac{\sigma_s u - \sigma_i w}{\beta(\sigma_s(u - c) - \sigma_i w)^2} > 0$$

When  $c$  decreases, a susceptible individual chooses to protect at a lower level of risk, and HIV prevalence declines. On the other hand, if  $c$  were to rise, the marginal cost of protection and the marginal benefit of exposure both increase; a susceptible individual is willing to take on more risk and switches from expose to protect at a higher HIV prevalence. The second order partial derivative of  $\alpha^*$  with respect to  $c$  indicates that as  $c$  increases, equilibrium HIV prevalence increases at a faster rate:

$$\frac{\partial^2 \alpha^*}{\partial c^2} = \frac{\sigma_s(\sigma_s u - \sigma_i w)}{\beta(\sigma_s(u - c) - \sigma_i w)^3} > 0$$

Because the cost of protection does not affect the transmission or the survival rates of infected individuals, there is no epidemiological effect of a prevention intervention. Only the behavioral effect of a change in  $c$  acts on  $\phi^*$ . Since  $\partial \phi^* / \partial \alpha^* = 1 / (\sigma_i \beta (1 - \alpha^*)^2) > 0$ , both  $\alpha^*$  and  $\phi^*$  will change in the same direction:

$$\begin{aligned} \frac{\partial \phi^*}{\partial c} &= \frac{\partial \phi^*}{\partial \alpha^*} \frac{\partial \alpha^*}{\partial c} = \frac{\sigma_s u - \sigma_i w}{\sigma_i \beta^2 (1 - \alpha^*)^2 (\sigma_s(u - c) - \sigma_i w)^2} \\ \frac{\partial \phi^*}{\partial c} &= \frac{\sigma_s u - \sigma_i w}{\sigma_i (\beta(\sigma_s(u - c) - \sigma_i w) - c)} = \frac{X + \sigma_s c}{\sigma_i \beta X - \sigma_i c} > 0 \end{aligned}$$

A prevention intervention that lowers the costs of protection will improve social welfare because both  $\alpha^*$  and  $\phi^*$  decrease. All three goals of an intervention are met: lower HIV prevalence, lower incidence of new infections, and lower number of deaths.

### **Treatment coverage intervention**

Increasing treatment coverage will shift the average survival rate of infected individuals and improve lifetime expected utility in the infected state. Therefore, a

change in  $k$  has epidemiological and behavioral effects. First, the behavioral effect of a treatment coverage intervention:

$$\frac{\partial \alpha^*}{\partial k} = \frac{\partial \alpha^*}{\partial \sigma_i} \frac{\partial \sigma_i}{\partial k} = \frac{cw}{\beta(\sigma_s(u - c) - \sigma_i w)^2} \left( \frac{p_i(p_r - p_i)}{(1 - p_i - k(p_r - p_i))^2} \right) > 0$$

When treatment is more widely available, susceptible individuals choose to expose at higher levels of HIV prevalence, because the consequence of infection is improved. The behavioral effect also increases the equilibrium exposure rate, since  $\partial \phi^* / \partial \alpha^* > 0$ . However, as  $k$  increases, more infected individuals survive longer at the rate  $p_r$ , so there are fewer new infections required to maintain a given level of HIV prevalence. In this way, an increase in  $k$  has a negative epidemiological effect on  $\phi^*$ . To analyze the total effect on the rate of exposure, it is useful to break  $\phi^*$  into the product of the epidemiological effect,  $\phi^*_E = 1/(\sigma_i \beta)$ , and behavioral effect,  $\phi^*_B = 1/(1 - \alpha^*)$ :

$$\frac{\partial \phi^*}{\partial k} = \frac{\partial \phi^*_E}{\partial k} \phi^*_B + \frac{\partial \phi^*_B}{\partial k} \phi^*_E$$

$$\frac{\partial \phi^*_E}{\partial k} = \frac{-(p_r - p_i)}{\beta p_i}$$

$$\frac{\partial \phi^*_B}{\partial k} = \frac{\partial \phi^*_B}{\partial \alpha^*} \frac{\partial \alpha^*}{\partial k} = \frac{1}{\beta(1 - \alpha^*)^2} \left( \frac{cw}{\beta(\sigma_s(u - c) - \sigma_i w)^2} \right) \left( \frac{p_i(p_r - p_i)}{(1 - p_i - k(p_r - p_i))^2} \right)$$

At this point, the signs of the opposing behavioral and epidemiological effects are clear.

The total partial effect on  $\phi^*$  simplifies to:

$$\frac{\partial \phi^*}{\partial k} = \frac{1}{\beta(1 - \alpha^*)} \left( \frac{p_r - p_i}{p_i} \right) \left( -1 + \frac{cw\sigma_i}{(\beta X)^2 - c\beta X} \right)$$

Then there is a minimum welfare differential,  $X_k$  such that the epidemiological effects outweigh the behavioral effects, or when  $\partial\phi^*/\partial k < 0$ :

$$cw\sigma_i < (\beta X)^2 - c\beta X \implies \frac{\partial\phi^*}{\partial k} < 0$$

$$X_k = \frac{1 + \sqrt{1 + 4cw\sigma_i}}{2\beta}$$

As  $k$  increases, the welfare differential  $X$  decreases and susceptible individuals have lower incentive to protect. Eventually the positive behavioral effect overtakes the negative epidemiological effect and  $\phi^*$  increases.

Although a treatment coverage intervention increases HIV prevalence, social welfare may improve if new infections make up a smaller share of the infected population. This would indicate that the individuals who survive longer because of treatment outnumber the new infections caused by an increase in risky behavior. In particular, social welfare improves with an increase in  $k$  if the epidemiological effects outweigh the behavioral effects,  $X > X_k$ , and if the effect of a fall in  $\phi^*$  outweighs the effect of an increase in  $\alpha^*$  in the social welfare function,  $\partial W/\partial\phi^* > \partial W/\partial\alpha^*$ .

### **Treatment quality intervention**

Pharmaceutical or healthcare delivery innovations that improve the effectiveness of ARV treatment increase  $p_r$ . A change in  $p_r$  will have behavioral and epidemiological effects. The effect of a rise in  $p_r$  on  $\alpha^*$  is similar to the effect of a change in  $k$ :

$$\frac{\partial\alpha^*}{\partial p_r} = \frac{\partial\alpha^*}{\partial\sigma_i} \frac{\partial\sigma_i}{\partial p_r} = \frac{cw}{\beta(\sigma_s(u - c) - \sigma_i w)^2} \left( \frac{p_i k}{(1 - p_i - k(p_r - p_i))^2} \right) > 0$$



As with treatment coverage, treatment effectiveness has a positive behavioral effect; an improvement in treatment quality causes  $\alpha^*$  to rise. Note that the behavioral effects of an intervention in treatment quality will be larger than an intervention in treatment coverage if  $k > p_r - p_i$  :

$$\frac{\partial \alpha^* / \partial p_r}{\partial \alpha^* / \partial k} = \frac{k}{(p_r - p_i)}$$

Intuitively, if only a small proportion  $k$  of individuals receive treatment, then improving the effectiveness of treatment ( $p_r - p_i$ ) will have a smaller effect on behaviors than expanding access to existing treatment. When both treatment interventions occur concurrently, there is greater behavioral elasticity:

$$\frac{\partial^2 \alpha^*}{\partial p_r \partial k} = \frac{\partial^2 \alpha^*}{\partial k \partial p_r} = \frac{cw\sigma_i}{\beta X^2 p_i} + \frac{k(p_r - p_i)}{(1 - p_i - k(p_r - p_i))^2} \left( \frac{2cw\sigma_s(u - c)}{\beta X^3} \right) > 0$$

The behavioral elasticity of HIV prevalence with respect to an increase in treatment quality is amplified as more individuals have access to that treatment. Similarly, behavioral effects of a change in treatment coverage are amplified when treatment is known to be more effective.

As with treatment coverage, treatment quality has a positive behavioral effect on  $\phi^*$  and a countering negative effect due to the higher survival rate of infected individuals on treatment. The same method of breaking  $\phi^*$  into behavioral and epidemiological effects is used to solve the net effect of treatment quality on the rate of exposure:

$$\frac{\partial \phi^*}{\partial p_r} = \frac{-k}{\beta p_i} \phi_B^* + \frac{1}{(1 - \alpha^*)^2} \left( \frac{cw}{\beta X^2} \right) \left( \frac{p_i k}{(1 - p_i - k(p_r - p_i))^2} \right) \phi_E^*$$

This simplifies to:

$$\frac{\partial \phi^*}{\partial p_r} = \frac{k}{p_i} \left( \frac{1}{\beta(1 - \alpha^*)} \right) \left( -1 + \frac{cw\sigma_i}{\beta(\beta X - c)} \right)$$

As before, there is a critical welfare differential,  $X_{pr}$ , above which the net effect on  $\phi^*$  from a change in  $p_r$  is negative:

$$cw\sigma_i < \beta^2 X - \beta c \implies \frac{\partial \phi^*}{\partial p_r} < 0$$

$$X_{p_r} = \frac{cw\sigma_i + \beta c}{\beta^2}$$

The minimum welfare differentials that ensure a negative effect on  $\phi^*$  may be different for treatment coverage and quality interventions. For treatment coverage, the minimum welfare differential  $X_k$  satisfies  $cw\sigma_i < (\beta X)^2 - \beta cX$ , and the analogous  $X_{pr}$  for treatment quality satisfies  $cw\sigma_i < \beta^2 X - \beta c$ . Therefore,  $X_k < X_{pr}$  when  $u$  is large relative to  $c$  and  $w$  such that  $X > 1$ . This may be included as an additional parameter restriction on utility in the susceptible state:

$$u > \frac{1 + \sigma_i w + \sigma_s c}{\sigma_s} \implies X_k < X_{p_r}$$

Whether or not this condition holds depends on the arbitrary magnitude of the loss in welfare from infection within time periods ( $u - c - w$ ) compared to the loss in welfare from lower life expectancy ( $\sigma_s - \sigma_i$ ). However, it makes intuitive sense that living with the physical, mental health, and social effects of HIV would cause a loss in welfare to satisfy

this restriction, even if the loss in life expectancy were completely eliminated through treatment.

The epidemiological effects outweigh the behavioral effects for a larger range of welfare differential  $X$  when ARVs are made more accessible than when ARVs are made more effective. If the environment is such that  $X_{pr} < X < X_k$ , then a treatment *quality* intervention would *increase*  $\phi^*$ , while a treatment *coverage* intervention would *decrease*  $\phi^*$ . Therefore, a treatment coverage intervention improves welfare under a wider set of circumstances than a treatment quality intervention.

### **Changes to underlying health**

Health interventions that are not targeted at HIV or underlying health shocks may affect susceptible and infected populations differently. In the model, changes to underlying health that affect  $\sigma_s$  and  $\sigma_i$  differently will have behavioral and epidemiological effects. This analysis could be done with the survival rates  $p_s$  and  $p_i$ , with the same qualitative results (  $\partial\sigma_s/\partial p_s > 0$  ;  $\partial\sigma_i/\partial p_i > 0$  ). However, it is less intuitive for a single exogenous shock to affect infected individuals differently depending on treatment status (e.g., a change in  $p_i$  but not in  $p_r$ ), so underlying health is analyzed as a change in overall survival odds ratios for susceptible or infected states.

Beginning with a change in an infected individual's survival odds, those with worse health may gain a larger marginal benefit from basic public health improvements. For example, because individuals infected with HIV have compromised immune systems, they may experience a larger increase in survival odds from improvements to sanitation. In the other direction, individuals with worse health are also likely to be more vulnerable

to catastrophic events such as conflict or natural disaster. Events that affect individuals infected with HIV exclusively or more than susceptible individuals are modeled as an exogenous change in  $\sigma_i$ .

$$\frac{\partial \alpha^*}{\partial \sigma_i} = \frac{cw}{\beta(\sigma_s(u - c) - \sigma_i w)^2} = \frac{cw}{\beta X^2} > 0$$

$$\frac{\partial \phi^*}{\partial \sigma_i} = \frac{1}{\sigma_i \beta (1 - \alpha^*)} \left( \frac{1}{\sigma_i} - \frac{cw}{\beta (1 - \alpha) X^2} \right)$$

The results are similar to the treatment interventions, since both treatment interventions also increase  $\sigma_i$ . If the following condition is satisfied then there will be a negative net effect on  $\phi^*$ :

$$cw\sigma_i < \beta X^2 - cX \implies \frac{\partial \phi^*}{\partial \sigma_i} < 0$$

A unit increase in  $\sigma_i$  is more likely to improve welfare than either of the treatment interventions in isolation ( $X_i < X_k < X_{pr}$ ).

An exogenous change in underlying health may also favor susceptible individuals. For example, if individuals infected with HIV have less access to a health innovation because of costs or limited mobility, then only  $\sigma_s$  increases. A change in  $\sigma_s$  will affect risk behavior, but has no epidemiological effect on steady state equilibrium.

The behavioral effect on HIV prevalence and the resulting change in equilibrium exposure rate from a change in  $\sigma_s$  are as follows:

$$\frac{\partial \alpha^*}{\partial \sigma_s} = \frac{-c(u - c)}{\beta(\sigma_s(u - c) - \sigma_i w)^2} = \frac{-c(u - c)}{\beta X^2} < 0$$

$$\frac{\partial \phi^*}{\partial \sigma_s} = \frac{\partial \phi^*}{\partial \alpha^*} \frac{\partial \alpha^*}{\partial \sigma_s} = \frac{1}{\beta(1 - \alpha^*)^2} \left( \frac{-c(u - c)}{\beta X^2} \right) < 0$$

If an improvement in underlying healthcare benefits susceptible individuals, then  $\sigma_s$  increases, the welfare gap between susceptible and infected states grows wider, and susceptible individuals choose protect at a lower HIV prevalence.

### Complementary effects

Treatment and prevention interventions as well as changes to underlying health may occur simultaneously. It is important to not only examine behavioral and epidemiological effects of each intervention independently, but to take a look at the ways in which one intervention may change the effectiveness of another. If one intervention increases the returns of another intervention it is said to have a complementary effect (as in Milgrom & Roberts, 1995). A prevention intervention will be more effective, or have higher returns, when there is a higher behavioral elasticity with respect to the cost of protection ( $\partial \alpha^* / \partial c$ ) because a unit change in the cost of protection leads to a larger drop in HIV prevalence. This elasticity depends on the expected welfare differential. Therefore, treatment and underlying health factors will in part determine the effectiveness of a prevention intervention.

As demonstrated below, the magnitude of a change in  $\alpha^*$  caused by a change in  $c$  increases with  $k$ ,  $p_r$ , and  $\sigma_i$ :

$$\frac{\partial^2 \alpha^*}{\partial k \partial c} = \frac{w(X + 2\sigma_s c)}{\beta X^3} \left( \frac{p_i(p_r - p_i)}{(1 - p_i - k(p_r - p_i))^2} \right) > 0$$

$$\frac{\partial^2 \alpha^*}{\partial p_r \partial c} = \frac{w(X + 2\sigma_s c)}{\beta X^3} \left( \frac{p_i k}{(1 - p_i - k(p_r - p_i))^2} \right) > 0$$

$$\frac{\partial^2 \alpha^*}{\partial \sigma_i \partial c} = \frac{w(X + 2\sigma_s c)}{\beta X^3} > 0$$

Improvement in the lifetime expected utility of infected individuals (through either treatment intervention or from an unrelated change in underlying health), increases the elasticity of HIV prevalence with respect to the cost of protection. This is because the risk behavior depends on the tradeoff between the cost of protection and the risk of infection. As the lifetime expected utility in the two health states converge, the marginal cost of choosing to protect depends more and more on the cost of protection rather than on the consequence if infected. Therefore, with higher treatment coverage or quality, HIV prevalence falls with the cost of protection at a faster rate. Intuitively, if the gap between welfare in the susceptible and infected state is smaller, then costs or barriers to protection play a larger role in determining risk behavior.

A change in  $\sigma_s$  has the opposite effect on the behavioral elasticity:

$$\frac{\partial^2 \alpha^*}{\partial \sigma_s \partial c} = \frac{uX^2 - 2X(u - c)(\sigma_s u - \sigma_i w)}{\beta X^4} \frac{\partial \alpha^*}{\partial \sigma_s \partial c}$$

$$\frac{\partial^2 \alpha^*}{\partial \sigma_s \partial c} = \frac{-u(\sigma_s u - \sigma_i w) - 2\sigma_i w}{\beta X^3} < 0$$

A rise in the survival odds of susceptible individuals relative to infected individuals increases the welfare differential. Then, a change in the cost of prevention will have a smaller behavioral effect when an improvement in underlying health disproportionately benefits susceptible individuals.

## V. Treatment as Prevention

Clinical research indicates that individuals who receive treatment are less likely to transmit the virus because of a suppressed viral load (Cohen et al., 2011). This may be modeled with a relationship between treatment coverage and the transmission rate,  $\beta=g(k)$ . Then, treatment coverage affects both dimensions of risk: the likelihood of becoming infected and the expected welfare in the infected state.

As more individuals receive treatment, the transmission rate decreases,  $g'(k)<0$ . If a new infection occurs with probability  $\rho$  when a susceptible individual chooses expose and matches with an infected individual, then  $\beta=E(\rho)$ . For illustrative purposes, assume that  $\rho$  has a binary relationship with treatment:

$$\rho = \begin{cases} 0 & \text{if treatment} \\ 1 & \text{if no treatment} \end{cases}$$

At the population level, the transmission rate will equal the proportion of infected people not on treatment,  $\beta=1-k$ :

The new steady-state equilibrium conditions with preventative treatment effects are as follows:

$$\alpha^* = \frac{c}{(1-k)X} \quad \phi^* = \frac{1}{\sigma_i(1-k)(1-\alpha^*)}$$

Before,  $\beta$  was an arbitrary value representing the natural transmission rate. With this modification, a susceptible individual takes into account the fact that ARV treatment leads to a lower transmission rate. Behavioral and epidemiological effects of treatment coverage, and complementary effects of treatment coverage must be reconsidered.

## Treatment as prevention in health interventions

In the case of a prevention intervention, the behavioral effect on  $\alpha^*$  and the total effect on  $\phi^*$  are the same, but with  $(1-k)$  replacing  $\beta$ :

$$\frac{\partial \alpha^*}{\partial c} = \frac{\sigma_s u - \sigma_i w}{(1-k)(\sigma_s(u-c) - \sigma_i w)^2} > 0$$

$$\frac{\partial \phi^*}{\partial c} = \frac{X + \sigma_s c}{\sigma_i(1-k)X - \sigma_i c} > 0$$

However, the complementary effect of treatment coverage on prevention interventions is different because  $k$  now affects the risk of new infections. Originally, as  $k$  increased and expected welfare in the infected state improved, the elasticity of HIV prevalence with respect to  $c$  increased. When  $\beta=(1-k)$ , a rise in  $k$  has an additional complementary effect:

$$\frac{\partial^2 \alpha^*}{\partial k \partial c} = \frac{\partial}{\partial \sigma_i} \left( \frac{\sigma_s u - \sigma_i w}{(\sigma_s(u-c) - \sigma_i w)^2} \right) \frac{\partial \sigma_i}{\partial k} \left( \frac{1}{1-k} \right) + \frac{\sigma_s u - \sigma_i w}{(\sigma_s(u-c) - \sigma_i w)^2} \left( \frac{1}{1-k} \right)^2$$

$$\frac{\partial^2 \alpha^*}{\partial k \partial c} = \frac{w(X + 2\sigma_s c)}{X^3} \left( \frac{p_i(p_r - p_i)}{(1 - p_i - k(p_r - p_i))^2} \right) \left( \frac{1}{1-k} \right) + \frac{X + \sigma_s c}{X^2} \left( \frac{1}{1-k} \right)^2$$

The change in the behavioral elasticity is amplified by the second term, the effect of the transmission rate. When the transmission rate declines because of higher treatment coverage, susceptible individuals face a smaller risk of becoming infected, and the behavioral response to  $c$  is more elastic.

When treatment coverage increases, susceptible individuals choose expose for a higher level of HIV prevalence because both dimensions of risk, the likelihood and



consequence of HIV infection, decrease. The total behavioral effect of  $k$  includes a second positive term that reflects the preventative effect of treatment:

$$\frac{\partial \alpha^*}{\partial k} = \frac{cw}{(1-k)X^2} \left( \frac{p_i(p_r - p_i)}{(1 - p_i - k(p_r - p_i))^2} \right) + \frac{c}{X(1-k)^2}$$

Without the preventative effect of treatment, there is a purely negative epidemiological effect on  $\phi^*$ ; an increase in  $k$  causes more infected individuals to survive at a rate  $p_r$ , so the exposure rate in equilibrium decreases. With the preventative effect of treatment, there is also a positive epidemiological effect on  $\phi^*$ ; an increase in  $k$  causes a decrease in transmission rate and requires a higher exposure rate to reach the same number of new infections. The total epidemiological effect of  $k$  is positive when  $\phi^*_E = 1/((1-k)\sigma_i)$ :

$$\frac{\partial \phi^*_E}{\partial k} = \frac{1 - p_r}{p_i(1 - k)^2}$$

In contrast to the case when treatment only affects health, the elasticity of the exposure rate to a change in treatment coverage is always positive:

$$\frac{\partial \phi^*}{\partial k} = \frac{1 - p_r}{p_i(1 - k)^2(1 - \alpha^*)} + \frac{1}{\sigma_i(1 - k)(1 - \alpha^*)^2} \frac{\partial \alpha^*}{\partial k} > 0$$

Under the social welfare framework discussed earlier, this implies that an increase in  $k$ , with full preventative effects of treatment, causes a decrease in social welfare. The number of new infections grows at a faster rate because of amplified behavioral effects. With  $\beta = 1 - k$ , the epidemiological effects also contribute to a higher  $\phi^*$ . The rise in  $\phi^*$  indicates that in addition to a higher amount of infected individuals, a higher proportion of them are new infections.

This extreme case where no treated individuals transmit the virus exemplifies the concern that expanding drug coverage will worsen the epidemic and social welfare. However, it also illustrates that the preventative effects of treatment amplify any complementarities between treatment and prevention. This suggests that the negative behavioral impact of treatment as prevention may be mediated by an increased behavioral elasticity with respect to other prevention interventions.

## **VI. Discussion**

This model illustrates the importance of individual decision-making in determining the effectiveness of HIV prevention and treatment interventions. Treatment coverage, treatment quality, the cost of protection, and the underlying health differential between susceptible and infected individuals affect the behavioral outcomes of the model. In certain cases, they also affect the epidemiological nature of the system, by either changing the average survival rate of infected individuals or the rate of new infections.

While prevention interventions reduce the rate of risk-taking behaviors, treatment interventions increase risky behaviors of forward-looking susceptible individuals. However, treatment interventions also have epidemiological effects, which are important for social welfare. Treatment interventions cause infected individuals to survive longer, so that the composition of the infected population may shift toward a smaller proportion of new infections and a larger proportion of surviving individuals. This is likely to occur under a wider set of circumstances if treatment coverage increases than if treatment quality alone increases.

A prevention intervention will have greater effectiveness at changing risk behavior and lowering HIV prevalence if there is a concurrent increase in treatment

coverage, treatment quality, or health for infected individuals. Oster (2005) finds that low life expectancy suppresses preventative behavioral change. This model suggests that in addition to extending life-expectancy, other types of care including palliative care and mental health support for infected individuals may improve the effectiveness of prevention interventions. The existence of complementary effects between treatment and prevention interventions implies that the optimal public health strategy can only be achieved through coordination (Milgrom & Roberts, 1995). If behavioral and epidemiological effects of treatment and prevention interventions are considered in isolation, then complementary effects will be overlooked and the benefits of improving care for infected individuals may be underestimated.

Previous economic models of HIV demonstrate that the introduction of a hypothetical imperfect vaccine could lead to higher HIV prevalence or disease cycles (Kremer, 1996; Geoffard & Philipson, 1996). Evidence that that ARV treatment prevents new infections suggests that we may have had an imperfect vaccine without initially realizing it. Analogous to the results in Kremer's model, the knowledge that ARV treatment acts as prevention adds further incentive for risk behaviors, and HIV prevalence increases more than in the case where transmission is unrelated to treatment. However, the complementary effect on a prevention intervention is also amplified given that treatment affects both dimensions of risk. In summary, prevention interventions are more effective when ARVs are widely accessible, effective, and known to decrease infectiousness.

The simplifications in this model are meant to provide intuitive and interpretable results. However, these simplifications can also be launching points for future research.

Take, for example, the assumption that all individuals have the ability to choose protection and face the same costs. The empirical finding that women are less likely to use condoms despite facing a higher biological risk of contracting HIV through heterosexual sex suggests, in conjunction with sociological research, that women are less likely to be able to negotiate safe sex (UNAID, 2010). Given that, in sub-Saharan Africa, women are eight to ten times more likely to be infected with HIV than men, a model that incorporates bargaining and differentiates between male and female initiated forms of protection may have important implications. Further extensions of this model may also consider the effects of non-random matching, imperfect health status information, or altruism toward better understanding the behavioral implications of public health interventions.

## References

- Anderson, R. (1991) *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press: Oxford.
- Auld, M.C. (2003). Choices, beliefs, and infectious disease dynamics. *Journal of Health Economics*. 22: 361-377.
- Bauch, C.T. & Earn, J.D. (2004). Vaccination and the theory of games. *Proceedings of the National Academy of Sciences*. 101: 13391-13394.
- Bellman, R. (1956). *Dynamic Programming*. Princeton University Press: Princeton, NJ.
- Bertsekas, D. (1995). *Dynamic Programming and Optimal Control*. Athena Scientific: New York, NY.
- Bezemer, D., de Wolf, F., Boerlijst, M.C., van Sighem, A., Hollingsworth, T.D., Prins, M.,..., Fraser, C. (2008) A resurgent HIV-1 epidemic among men who have sex with men in the era of potent antiretroviral therapy. *AIDS*. 22: 1071-1077.
- Chen, F.H. (2009). Modeling the effect of information quality on risk behavior change and the transmission of infectious diseases. *Mathematical Biosciences*. 217: 125-133.
- Cohen, M.S., Chen, Y.Q., McCauley, M., Gamble, T., Hosseinipour, M. C, Kumarasamy, N...& Fleming, T.R. (2011) Prevention of HIV-1 infection with early antiretroviral therapy. *The New England Journal of Medicine*. 365: 493-505.
- Dieffenbach, C.W., & Fauci, A.S. (2009) Universal voluntary testing and treatment for prevention of HIV transmission. *The Journal of the American Medical Association*. 301: 2380-2382.

- Dow, W.H., Philipson, T., & Sala-i-Martin, X. (1999). Longevity complementarities under competing risks. *The American Economic Review*. 89: 1358-1371.
- Funk, S., Salathé, M., & Jansen, V.A. (2012). Modelling the influence of human behaviour on the spread of infectious diseases: A review. *Journal of the Royal Society Interface*. 7: 1247-1256.
- Geoffard, P. & Philipson, T. (1996). Rational epidemics and their public control. *International Economic Review*. 37: 603-624.
- Gray, R.H., Wawer, M.J., Bookmeyer, R., Sewankambo, N.K., Serwadda, D., Wabwire-Mangen, F., ..., Quinn, T.C. (2001). Probability of HIV-1 transmission per coital act in monogamous, heterosexual, HIV-1 discordant couples in Rakai, Uganda. *The Lancet*. 357: 1149-1153.
- Hearst, N. & Chen, S. (2004). Condom promotion for AIDS prevention in the developing world: Is it working? *Studies in Family Planning*. 35: 39-47.
- Kremer, M. (1996). Integrating behavioral choice into epidemiological models of AIDS. *The Quarterly Journal of Economics*. 111: 549-573.
- Lima, V., Johnston, K., Hogg, R.S., Levy, A.R., Harrigan, R., Anema, A., & Montaner, J.S.G. (2008) Expanded access to Highly Active Antiretroviral Therapy: A potentially powerful strategy to curb the growth of the HIV epidemic. *The Journal of Infectious Diseases*. 198: 59-67.
- Mannberg, A. (2011). Risky sex in a risky World: Sexual behavior in an HIV/AIDS environment. *Scandinavian Journal of Economics*. (upcoming).

- Milgrom, P. & Roberts, J. (1995). Complementarities and fit strategy, structure, and organizational change in manufacturing. *Journal of Accounting and Economics*. 19: 179-208.
- Montaner, J., Hogg, R., Wood, E., Kerr, T., Tyndall, M., Levy, A., & Harrigan, P.R. (2006). The case for expanding access to highly active antiretroviral therapy to curb the growth of the HIV epidemic. *The Lancet*. 368: 531-536.
- Oster, E. (2005). Sexually transmitted infections, sexual behavior, and the HIV/AIDS epidemic. *Quarterly Journal of Economics*. 120: 467-515.
- Philipson, T. (2000). Economic epidemiology and infectious diseases. In Culyer, A.J. & Newhouse, J.P. (Eds.), *Handbook of Health Economics, Volume 1* (pp. 1761-1799). Elsevier.
- Philipson, T., & Posner, R. (1993). *Private Choices and Public Health: The AIDS epidemic in an economic perspective*. London, UK: Harvard University Press.
- Schroeder, K. D. & Fabio, R. G. (2002). A game theoretical analysis of sexually transmitted disease epidemics. *Rationality and Society*. 14: 353-383.
- UNAIDS. (2010). *UNAIDS Report on the Global AIDS Epidemic*. Geneva: UNAIDS, WHO.
- Weller, S.C. & Davis-Beaty, K. (2007). Condom effectiveness in reducing heterosexual HIV transmission (Review). *The Chocrane Library*. John Wiley & Sons, Ltd.