The effects of prevention and treatment interventions in a microeconomic

model of HIV transmission

Allison Stashko

Professor Curtis R. Taylor, Faculty Advisor

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Abstract

A rational choice-based model for sexual transmission of HIV demonstrates the 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 is more effective when infected individuals are better off. Specifically, treatment interventions increase the elasticity of behavioral change with respect to the cost of protection. Complementary effects between different types of interventions are important for finding an optimal public health HIV strategy.

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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 policies must strike a balance between preventing

new infections and caring for those already infected. In order to determine if a health

intervention will be effective, it is important to understand the ways in which it will affect

risk-behaviors. A rational choice-based model for sexual transmission of HIV illustrates

the effects of prevention and treatment interventions on risk behaviors and can provide

insight into the optimal public health response to HIV.

Economic models of epidemics suggest that individual-level behaviors both

respond to and determine disease dynamics (Philipson, 2010). Models of epidemics that

incorporate rational behavior have different predictions from standard epidemiological

models. 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. Sexually transmitted diseases, such as HIV, are candidates for

economic modeling because exposure may be avoided through a protective behavior. The

elasticity of a risk behavior with respect to HIV prevalence, or the dynamic demand for

protection, has many important implications. For example, a permanent change in the

cost of protection may increase incentives for protective behavior and lower disease prevalence. Additionally, education or any change to informational aspects of HIV will only have an effect on the epidemic if individuals are able to make decisions (Philipson, 2000).

Some of the leading papers in economic epidemiology use a choice-based approach for models of HIV transmission (Philipson & Posner, 1993; Kremer, 1996; Geoffard & Philipson, 1996). However, several key 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 directly affect 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 regimen that combines several classes of ARVs, has transformed the wellbeing and mortality 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 changes incentives and affects 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, the preventative effects of treatment, or knowledge that ARV treatment lowers the likelihood of secondary infections, will change behavior and HIV outcomes.

Prevention interventions reduce the rate of risky exposure and HIV prevalence in 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 welfare depending on the weight of opposing behavioral and epidemiological effects. Additionally, any improvement in the welfare of infected individuals has a complementary effect on prevention interventions. The behavioral effects of an intervention and the complimentary effects between treatment and prevention 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 complimentary 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 for protection choice and sexual transmission of HIV; section IV analyzes the effects of exogenous health interventions in the model; 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 mortality and 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 trend in HIV mortality did not turn downward until 2005 in sub-Saharan Africa (UNAIDS, 2010).

In addition to improving the life expectancy and wellbeing of individuals living with HIV, the theory that an infected individual who receives treatment are less likely to transmit the virus has recently been supported in a randomized control trial (Cohen et al., 2011). Some promote the strategy of expanding ARV coverage as the most promising way to control the HIV epidemic (Montaner et al., 2006). Lima et al. (2008) simulate the scale-up of HAART and argue that large initial investments in ARV coverage would be offset by the number of avoided infections. Others express concern that with a large increase in treatment coverage, behavioral incentives against using protection might outweigh the biomedical prevention from ARVs and lead to a resurgent HIV epidemic (Bezemer, 2008; Dieffenbach & Fauci, 2009). The behavioral responses to treatment will

be of critical importance as more information accumulates on the preventative capabilities of ARV therapy.

Economic models of HIV epidemics

Incorporating rational choice into a model of infectious diseases 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).[1]

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 his model, the perception that a vaccine lowers HIV risk may increase the rate of partner change such that the new infections outweigh the infections avoided biologically from the vaccine. Kremer also compares the behaviors of high-risk and low-risk populations. At the time when the model was created, HIV epidemics were more commonly concentrated in high-risk populations, such as

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[1] 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.

commercial sex-workers, than they are today. Among the high-risk population, the marginal probability of infection from an additional partner is smaller or possibly negative as HIV infection increases. Because it is so likely that they are already infected, a high-risk individual can optimize utility by increasing the number of partners. The timing of interventions and the targeting high-risk groups are critical for effective 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 model shows that the demand for a protective behavior, such as condom use, increases 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 (1996), 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. 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 for protecting from HIV does not address 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 this model 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 representing both treatment coverage and quality, and by including three different survival rates for susceptible, infected, and treated individuals. 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 susceptible individuals. HIV prevalence is both an endogenous to sexual risk-behaviors and the result of decisions to use protection 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 status 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 α 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.[2] 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. 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

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[2] Male condoms are the most widely accessible and effective form of protection from sexual transmission. 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).

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 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 a time period t, a susceptible individual maximizes the value function V_s with the control variable a={protect, expose}, given the following exogenous parameters: the full cost of using a condom, c; utility per time period, u; HIV prevalence in the population, α; transmission rate, β; 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

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probability of transitioning to the infected state in the next period. The condition for optimality is clear when expressed as a Bellman equation below[3]:

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

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 probability β 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 that 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 overal 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

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[3] See Bertsekas (1995) for an overview of sequential decision making under uncertainty, or the original work of Bellman (1957) on dynamic programming.

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 state were delayed by a length of time, infected individuals may act as if they were susceptible and choose protect. Therefore, imperfect knowledge of HIV status would possibly decrease the rate of new infections. This counterintuitive results is dependent on the absence of any altruistic incentives.

Returning to the bellman equation for the infected 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, α^*, 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 α^* 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 is independent of HIV prevalence and is static. A constant proportion , of infected individuals receive ARV treatment. 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 $(1-k)$, they receive no treatment survive at a rate p_i :

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

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 $0 < p_i < p_r < p_s < 1$.

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.

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, and the rate of exposure among susceptible individuals, ϕ. A new infection occurs if

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

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 ϕ^* such 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, ϕ^*, only occurs when

α>α*satisfies the susceptible individual's indifference condition. The next step is to solve for α* in terms of the parameters of susceptible and infected state value functions.

Behavioral Equilibrium

Behavioral equilibrium occurs at α* 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 α* into the condition for epidemiological equilibrium yields an

expression for φ* 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 (α*) 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 (β) and the lifetime expected loss in welfare (X).

Exposure rate in steady state equilibrium (φ*) 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 (β(1-α*)).

To summarize, there are two conditions for a steady-state equilibrium (α*, φ*):

1) $$\alpha^* = \frac{c}{\beta X}$$

2) $$\phi^* = \frac{1}{\sigma_i \beta(1 - \alpha^*)}$$

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 ϕ^* indicates that fewer new infections are needed to replace HIV-related deaths. In other words, for any given level of HIV prevalence, as ϕ^* 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 α^* (HIV prevalence) and ϕ^* (HIV incidence, and death rate) will improve social welfare, an intervention that raises α^* and ϕ^* will worsen social welfare, and the impact of an intervention that has opposing effects on α^* and ϕ^* will depend on the welfare function W:

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

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 σ_s or σ_i represent changes to underlying health that affect susceptible and infected populations differently.

Parameters that affect α^* are said to have a behavioral effect, and parameters that affect ϕ^* independent of α, are said to have an epidemiological effect on steady-state equilibrium. Since ϕ^* is a function of α^*, the behavioral and epidemiological effects together determine the net effect of an intervention on ϕ^*. Table 1 Summarizes the

existence and direction of behavioral and epidemiological effects as well as the total

effects on α* and ϕ* for each type of intervention.

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

Parameter (x)	Behavioral Effect		Epidemiological Effect		$\partial\alpha^*/\partial(x)$	$\partial\phi^*/\partial(x)$
c	yes	+	no		+	+
k	yes	+	yes	-	+	+/-
p_r	yes	+	yes	-	+	+/-
σ_i	yes	+	yes	-	+	+/-
σ_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 α*:

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

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 α* 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 ϕ^*. Since

$\partial \phi^* / \partial \alpha^* = 1/(\sigma_i \beta (1 - \alpha^*)^2) > 0$, both α^* and ϕ^* will change in the same direction:

$$\frac{\partial \phi^*}{\partial c} = \frac{\partial \phi^*}{\partial \alpha_j^*} \frac{\partial \alpha_j^*}{\partial c} = \frac{\sigma_s u - \sigma_i w}{\sigma_i \beta^2 (1 - \alpha^*)^2 (\sigma_s (u - c) - \sigma_i w)^2}$$

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

A prevention intervention that lowers the costs of protection will improve social

welfare because both α^* and ϕ^* 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,

$\partial \phi^* / \partial \alpha^* > 0$. 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 φ*. To

analyze the total effect on the rate of exposure, it is useful to break φ* 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 φ* 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 $\partial \phi^*/\partial k < 0$:

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

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

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 φ* 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 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 ϕ^* outweighs the effect of an increase in α^* 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 α^* 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 α^* 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 ϕ^* and a countering negative effect due to the higher survival rate of infected individuals on treatment. The same method of breaking ϕ^* into behavioral and epidemiological effects is used to solve the net of the behavioral and epidemiological effects:

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

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 ϕ^* 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 ϕ^* may be different for treatment coverage and treatment effectiveness interventions. For treatment

coverage, the minimum welfare differential X_k satisfies $cw\sigma_i < (\beta X)^2 - \beta c X$, 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:

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

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 (σ_s-σ_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* ϕ*, while a treatment *coverage* intervention would *decrease*

ϕ*. 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 σ_s and σ_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 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 σ_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 σ_i. If the following condition is satisfied then there will be a positive net effect on ϕ^*:

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

A unit increase in σ_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 σ_s increases. A change in σ_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 σ_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 σ_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 complimentary 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 α* caused by a change in c increases with k, p_r, and σ_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, HIV prevalence falls with the price 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 σ_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. This may be modeled with a relationship between treatment coverage and the transmission rate, $\beta = g(k)$. Then, in addition to changing the expectations of welfare in the infected state, treatment coverage will affect risk behavior through the likelihood of becoming infected.

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

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

Before, β 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 α* and the total effect on φ* are the same, but with (1-k) replacing β:

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

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

However, the complimentary 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 β=(1-k), a rise in k has an additional complimentary 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 ϕ^*; an increase in k causes more infected people 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 ϕ^*; an increase in k causes a decrease in transmission rate and requires a higher exposure rate to reach the same incidence of new HIV 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 when treatment prevents infections:

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

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

This extreme case where no infected 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 death 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 with HIV. This is likely to occur under a wider set of circumstances if treatment coverage increases than if treatment quality 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, increase in treatment quality, or increase in health for infected individuals. Oster (2007) finds that low life expectancy suppresses behavioral change. This model suggests that in addition to higher life-expecancy, care for infected individuals including palliative care and mental health support, may cause behaviors change of susceptible individuals to be more sensitive health interventions. The existence of complimentary 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 complimentary 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 complimentary 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 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 combined with sociological evidence suggests that women are less likely to be able to negotiate safe sex (UNAID, 2010). Given that women are eight to ten times more likely to be infected with HIV in sub-Saharan Africa, 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 altruistic incentives toward better understanding the behavioral implications of public health interventions.

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