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## Article

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Article

# Social Context and the Spread of HIV: An Evolutionary Game-Theoretic Investigation on the Impacts of Social Stigma on Epidemic Outcomes

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**Abstract:** We provide a theoretical foundation for analyzing how social stigma and adopted behavioral traits affect the transmission of HIV across a population. We combine an evolutionary game-theoretic model—based on a relationship signaling stage game—with the SIR (susceptible-infected-recovered) model of disease transmission. Our evolutionary model specifies how two types of social stigma—that which accompanies an HIV<sup>+</sup> condition and that which follows associating with an HIV<sup>+</sup> partner—influence behavioral propensities to honestly report one’s condition (or not) and to unconditionally accept relationships (or not). With respect to reporting an HIV<sup>+</sup> condition, we find that condition stigma impedes the fitness of honest reporting, whereas association stigma impedes the relative fitness of concealing an HIV<sup>+</sup> condition; and both propensities can coexist in a polymorphic equilibrium. By linking our model to the SIR model, we find that condition stigma unambiguously enhances disease transmission by discouraging both honest reporting and a society’s acceptance of AIDS education, whereas association stigma has an ambiguous impact: on one hand it can impede HIV transmission by discouraging concealing behavior and unconditional relationship acceptance, but it also compromises a society’s acceptance of AIDS education. Our relatively simple evolutionary/SIR model offers a foundation for numerous theoretical extensions—such as applications to social network theory—as well as foundation for many testable empirical hypotheses.

**Keywords:** evolutionary game theory; HIV transmission; polymorphic equilibrium; SIR model of disease transmission; social stigma

**JEL classifications:** C70; C73; D80; I18; Z13

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## 1. Introduction

The HIV epidemic poses a collective-action problem to society because social interactions among individuals—interactions that operate within and respond to prevailing social contexts—affect the transmission of the virus. In particular, the social stigma that often accompanies an individual's HIV<sup>+</sup> condition tends to enhance disease transmission by discouraging honest reporting and by reducing a society's acceptance to AIDS education.

This paper incorporates evolutionary game theory and the SIR (susceptible-infected-recovered) model of disease transmission to provide a theoretical foundation for examining how specific elements of social context—notably the social stigma that can accompany either an individual's HIV<sup>+</sup> condition or one's association with an HIV<sup>+</sup> partner, along with a society's provision and acceptance of AIDS education—affect the dynamics of HIV transmission within a population. More specifically, this paper develops an evolutionary game-theoretic model that illustrates how multiple relationship interactions within populations influence the prevalence of behavioral traits that affect the transmission of HIV. Using a two-person relationship signaling game as a stage game, our evolutionary model illustrates interactions between the disease status of members of a population (HIV<sup>+</sup> or HIV<sup>−</sup>) and two types of behavioral traits—a propensity to honestly report one's status (or not) and a propensity to unconditionally accept relationships (or not). We proceed to link specific variables and outcomes from our evolutionary model to the SIR model of disease transmission in order to gain insight on how social context influences the transmission of the HIV virus.

By itself, our evolutionary model allows for a polymorphic equilibrium in which the behavioral trait of honest reporting as well as that of concealment of an HIV<sup>+</sup> condition (two alternative strategies or phenotypes) can both survive with stable (or perhaps oscillating) population proportions. Moreover, the population proportion of the honest-reporting trait within such a mix decreases in the society's level of *condition stigma*—the social stigma that a person experiences from disclosing an HIV<sup>+</sup> condition; whereas prevalence of the concealing trait tends to decrease in *association stigma*—the social stigma that accompanies accepting a relationship with an HIV<sup>+</sup> partner who has disclosed her status. By contrast, the traits of unconditional acceptance of relationships as opposed to cautious acceptance can exhibit a positive-feedback dynamic—whereby an increase in the population proportion of one trait tends to enhance its own fitness, leading to an equilibrium predominance of one or the other. Here, association stigma tends to diminish the fitness of the unconditional-acceptance trait, rendering its predominance somewhat less likely. Applying these outcomes to the SIR model, we find that condition stigma unambiguously increases the rate of disease transmission by discouraging honest reporting and by also discouraging the provision and acceptance of AIDS education. Association stigma, however, has an ambiguous impact on HIV transmission: on one hand it impedes transmission by discouraging both concealment and unconditional relationship acceptance; yet—as a

component of overall social stigma associated with the disease—it also discourages a society’s provision and acceptance of AIDS education.

### *Related Literature*

Recent advances in game theory, information economics, and social preference theory facilitate rigorous modeling of fundamentally social phenomenon—in this case factors that affect the rate of HIV transmission within a society. For decades, game theory has enabled systematic modeling of strategic behavior among interdependent agents.<sup>1</sup> Because behavior within personal relationships involves strategic interactions—often related to revealing one’s attributes and to accepting personal relationships—and because both revelation and relationship acceptance affect rates of disease transmission, game-theoretic models offer fruitful terrain for systematically conceptualizing the dynamics of disease transmission. Furthermore, more recent developments in evolutionary game theory [2,3] facilitate modeling the relative propensities as well as the spread or decline of specific behavioral traits within or across populations of agents—and thus related processes of social learning [4]. Evolutionary game-theoretic models thus present fertile terrain for modeling the time trajectories of disease conditions across populations or societies, as well as the relative prevalence of related behavioral propensities to report conditions and accept relationships.

Information economics enhances such analysis by systematically addressing how asymmetrically available information creates opportunities for strategic manipulation—via concealing, selectively revealing, or distorting information related to environments, actions, or attributes (e.g., one’s own disease status) in various social interactions.<sup>2</sup> Social preference theory can then specify utility payoffs to social (as opposed to material) factors such as status, invidious comparison, reputation, social rewards (praise), or social punishments (scorn) [7–9]. These payoffs, in turn, affect both the incentives for HIV<sup>+</sup> agents to honestly reveal their condition and the incentives for potential partners to accept or reject relationships with them. Moreover, the social context in a given society affects the magnitude and influence of such social preferences, in addition to material considerations. Overall, because individual strategic decisions influence a society’s rate of HIV transmission, and because HIV transmission confers social costs, the epidemic represents a collective-action problem for affected societies.<sup>3</sup>

A more precise literature, both theoretical and empirical, focuses on rates of disease and/or HIV transmission. Funk *et al.* [12] establish conceptual foundations for the analysis of disease transmission. More specifically, they review approaches to modeling various impacts of human behavior on the spread of infectious diseases. Drawing from different portions of the literature, they offer seven basic assertions about disease transmission:

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<sup>1</sup> For an intuitive undergraduate introduction to applied game theory, see Dixit, Skeath and Reiley [1].

<sup>2</sup> Such manipulation fits Oliver Williamson’s [5] concept of opportunism: pursuit of self-interest with guile. For a discussion of the implications of information economics, see Stiglitz [6].

<sup>3</sup> On the political economy of collective-action problems, see Ferguson [10] and Sandler [11].

1. Most fundamentally, the SIR model [13] provides a conceptual foundation for modeling the spread of disease. This model divides the population into the categories of susceptible (S), infected (I), and recovered (R) and also specifies two parameters that determine rates of disease transmission: the mixing or transmission coefficient ( $\beta$ ) and per-infection rate of recovery ( $\sigma$ ).<sup>4</sup>
2. Sources of information—either global or local—affect rates of disease transmission.
3. The type of available information—either prevalence or belief based—influences rates of transmission, and belief-based information need not reflect actual disease prevalence.
4. Human behavior affects disease transmission by influencing the S, I, R,  $\beta$  and/or  $\sigma$  terms in the SIR model.
5. Analysts can fruitfully use game theory to model relevant decisions (e.g., whether or not to vaccinate).
6. Behavioral traits related to disease transmission can, themselves, be transferred among individuals; hence such traits and their prevalence evolve over time.
7. Local social network structure affects the transmission of information, behavioral traits, and, indeed, levels of infection across populations.

Focusing directly on HIV in an analysis that relates to assertions 2, 3, and 5, Schroeder and Rojas [14] use a game-theoretic signaling model to examine potential disclosure of HIV status and subsequent impacts on transmission of HIV, from the perspective of partners who decide whether or not to use protection and consent to sex. They find two possible signaling equilibria: a separating equilibrium, in which agent's actions related to offers of protected or risky sex clearly identify their HIV<sup>+</sup> or HIV<sup>−</sup> status and a pooling equilibrium in which agents cannot deduce another's status from their actions—enhancing prospects for disease transmission. In the pooling scenario, agents use average infection rates among the relevant population to infer a potential partner's HIV status, and such inference affects their willingness to use protection and/or engage in a relationship. The authors proceed to link their micro-level game results to macro-level population dynamics by incorporating population simulations. They find that rates of transmission can oscillate between high and low levels, depending on population averages among relevant subgroups: localized high rates induce protective measures that, in turn, reduce rates of transmission, and *vice versa*. As Schroeder and Rojas point out, a game theoretic approach has several advantages over previous approaches to analyzing HIV transmission. First, game-theoretic analysis specifically addresses strategic interactions: one partner's decision depends, in part, on the actions and information provided by others, who engage in similar strategic estimation. Second, the prevalence of asymmetric information regarding agents' HIV condition allows for strategic misrepresentation or selective revelation.

A related empirical literature examines social factors that affect the disclosure of disease status, and by extension, rates of disease transmission. In terms of Funk *et al.* [12], this literature most directly examines assertions 2, 3, and 4. In these papers, social stigma or related variables emerge as key influences on disclosure, and disclosure affects transmission. As a summary, Smith *et al.* [15] perform a meta-analysis of 21 studies that relate the inclinations of individuals to disclose their HIV<sup>+</sup> status to perceived social stigma (from such revelation) and perceived social support. They find a stable

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<sup>4</sup> As in Funk *et al.* [12], this model can be augmented with models of social network structure that specify how social connections influence the spread of infection. For a relatively simple application of the SIR model to network structure, see Chapter 11 of Ferguson [10].

negative relationship whereby social stigma discourages honest disclosure. Along similar lines, though far more specifically, King *et al.* [16] investigate disclosure among HIV<sup>+</sup> men and women in Eastern Uganda. They note that that blame, rejection, abandonment, and abuse (all elements or manifestations of social stigma) are the key barriers to disclosure. Furthermore, they find that disclosure increases care-seeking behavior and reduces risk of disease transmission. Another study, Kalichman *et al.* [17], uses interview data to find that perceived social support enhances disclosure to family members and that the stress associated with disclosure (again related to stigma) discourages both social support and honest disclosure. Similarly, Liu *et al.* [18] examine HIV disclosure rates among commercial blood donors in rural China to find that increased knowledge about HIV enhances prospects for honest disclosure because information decreases worry, while worry increases public stigma, public stigma enhances felt stigma, and felt stigma discourages disclosure. In all of these studies social stigma emerges as a key variable that interferes with honest reporting.

## 2. Models of Behavior and HIV Transmission

Our paper extends this literature. Our chief contribution involves combining an evolutionary game-theoretic approach with the SIR model. In so doing, we address Funk *et al.*'s assertions 1–6 and discuss assertion 7 in the conclusion. More specifically, we use evolutionary game theory to model the transmission of disease-influencing behavioral traits across populations (assertions 5 and 6), with attention to the sources and availability of information (assertions 2 and 3); we then combine our model with the SIR disease diffusion model (assertions 1 and 4). As in Schroeder and Rojas [14], we link micro behavior based on a game-theoretic signaling model to macro-level population outcomes, but our approach differs from theirs five important respects. First, we use an evolutionary game model (itself based on a classical signaling model) instead of a classical signaling model combined with a population simulation. Second, for a signal, we use a statement about one's HIV status, rather than an action of using or not using protection. In our model, such signaling affects relationship acceptance. Whereas in Schroeder and Rojas, the act of using protection (obviously) directly reduces HIV transmission, for us, a propensity to honestly signal an HIV<sup>+</sup> condition (a behavioral trait that evolves over time) reduces the transmission of infection, implicitly by enhancing the use of protection and also by enhancing tendencies to test one's condition and seek treatment. Third, following a key finding of the empirical literature, our model then relates social stigma to agents' propensities to honestly disclose their HIV condition. Fourth, we explicitly model two types of social stigma that are associated with HIV, condition stigma and association stigma, to find distinct and somewhat contrasting impacts on honest reporting, relationship acceptance, and disease transmission. Fifth, we link our model to the SIR disease transmission model, establishing a link from our evolutionary game-theoretic approach to a generally accepted approach to modeling disease transmission.

### 2.1. An Evolutionary Game-Theoretic Perspective

Evolutionary game theory (EGT) facilitates modeling socially transmitted behaviors and associated processes of adaptive learning within or across populations; certain practices gain acceptance over time, while others decline or perish. Whereas in classical game theory, players choose strategies via estimated best responses to potential strategies of others, in EGTs, players "inherit" strategies. For the

social sciences, such inheritance emerges from education and other forms of cultural (rather than genetic) transmission.<sup>5</sup> Individuals, and by extension populations, inherit strategies (established practices) in various combinations. For example, some individuals or groups may instinctively bargain hard; others may tend to conciliate; some speak Chinese, others English. EGT strategies operate in a manner like that of biological phenotypes: an agent plays an assigned strategy in all relevant encounters as long as the player bears the relevant phenotype. Strategies (not players!) are then subject to evolutionary selection via fitness criteria—their relative success across multiple experienced encounters—that, in turn, affect their transmission (or acceptance) across populations.<sup>6</sup>

More precisely, in EGT models, randomly matched pairs of agents, as bearers of specific inherited strategies, interact. As in classical game theory, specific strategy combinations generate *payoffs*, but in EGT models payoffs represent a strategy's reproductive potential—its evolutionary fitness—rather than a player's utility. Strategies that earn relatively high payoffs across multiple encounters reproduce more rapidly than alternative strategies and thus spread across populations, as agents tend to adopt apparently successful strategies that they repeatedly hear about.<sup>7</sup> Likewise, agents tend to abandon strategies with relatively low fitness payoffs. From a social scientific perspective such evolutionary selection processes represent gradual adaptive social learning among populations of agents.

In the present context, agents—and by extension populations—may gradually learn that a strategy of either honest signaling or of concealing an HIV<sup>+</sup> condition offers greater or lesser rewards than the alternative. Likewise, they may learn that either accepting or rejecting a relationship after either receiving or not receiving a signal offers relative benefits. An evolutionary model can then generate predictions concerning the signaling and acceptance practices that develop among certain populations—with attendant implications on disease transmission.<sup>8</sup> We adopt this approach.

We now present an evolutionary model of HIV transmission among randomly matched pairs of potential relationship partners. The associated stage game unfolds in four steps:

1. Nature decides whether each player is an HIV carrier (HIV<sup>+</sup>), an event that occurs with probability  $p$ .
2. Each player privately observes nature's move and then either signals to their potential partner that he or she is HIV<sup>+</sup> ( $S$ ) or does not signal ( $NS$ ). The latter choice could imply, honestly or not, an HIV<sup>-</sup> status.
3. Neither player observes the other's type, but each observes either  $S$  or  $NS$ . Each then either accepts a relationship with the other ( $A$ ) or rejects the relationship ( $R$ ).
4. If and only if both choose  $A$ , a relationship ensues.
5. Payoffs follow.

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<sup>5</sup> Evolutionary biologists argue that evolutionary selection can apply to cultural processes and that cultural evolution unfolds much more rapidly than its biological counterpart. See for example, Wilson and Wilson [19] or Dawkins [20].

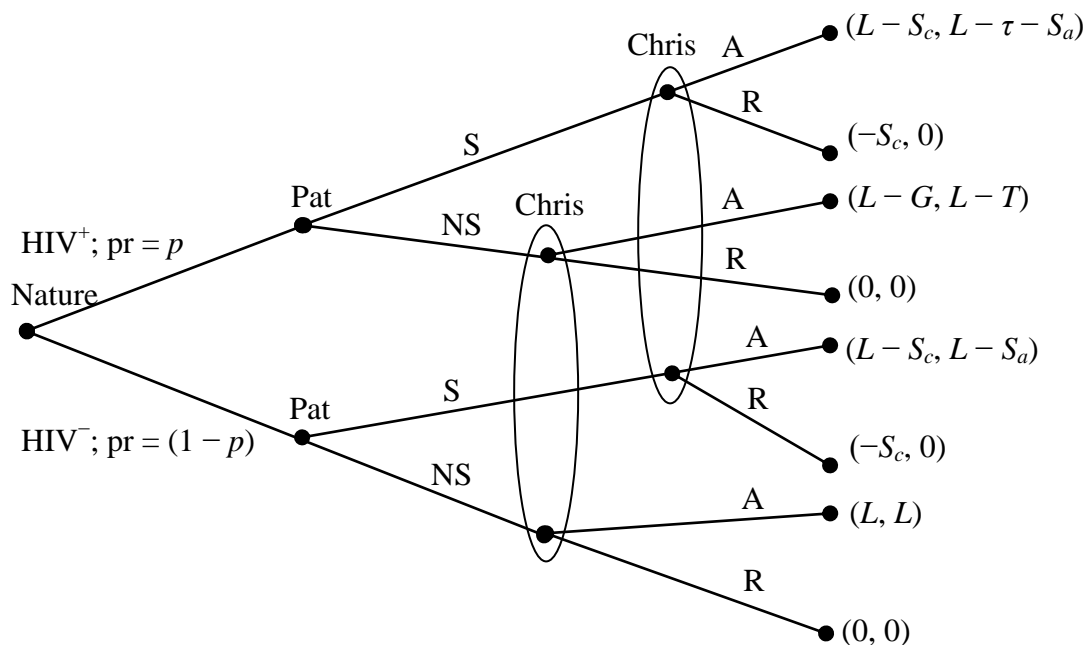
<sup>6</sup> For an introduction to evolutionary game theory, see Chapter 13 of Dixit, Skeath and Reiley [1]; for a thorough discussion, see Gintis [3].

<sup>7</sup> For Bowles, strategies (not individuals) are the “*personae dramatis*” of evolutionary game theory [8], p. 60.

<sup>8</sup> The dynamic emergence properties of evolutionary models present a new terrain for economic and social scientific analysis; see Epstein [21].

To proceed, we use a simplified version of this stage game to illustrate the sequence of interaction and the basic payoffs. Figure 1 represents a version of the stage game taken from the point of view of a player (Pat) who is the type of person who would always accept a relationship regardless of signal from another (later called an A-type) and whose potential partner (Chris) is HIV<sup>−</sup>.<sup>9</sup> More specifically, in step 1, with probability  $p$ , nature decides whether Pat is HIV<sup>+</sup>. Pat observes Nature's move, but Chris does not. In step 2, Pat chooses whether to signal (S) an HIV<sup>+</sup> status to Chris—or to not signal (NS).<sup>10</sup> In step 3, Chris decides whether to accept a relationship with Pat (A) or reject (R). Payoffs ensue.

**Figure 1.** Stage Game from the perspective of an accepting (A)-type agent (Pat) interacting with an HIV<sup>−</sup> agent (Chris).



**Legend:** An A-type agent always accepts a relationship, regardless of signal;  $L$ : relationship benefit to either player;  $G$ : guilt of an HIV<sup>+</sup> agent from not signaling to any agent who is not known to be HIV<sup>+</sup>;  $S_c$ : condition stigma;  $S_a$ : association stigma;  $T$ : risk to an HIV<sup>−</sup> agent of contracting HIV in an uninformed relationship;  $\tau$ : risk to an HIV<sup>−</sup> agent of contracting HIV in an informed relationship. The circles represent information sets, signifying that Chris does not know which node within the circle she is at.

The payoffs are as follows. Assume that, independent of the presence of HIV, Pat and Chris would benefit equally from having a relationship: each would receive positive payoff  $L$ . If Pat is HIV<sup>+</sup> and chooses to signal his status to Chris, he experiences *condition stigma*—the social stigma that society

<sup>9</sup> The full stage game includes all such interactions between all possible pairs, where each agent in a pair may belong to one of four distinct combinations of phenotype, with associated probabilities. In Figure 1, Pat does not know Chris's HIV<sup>−</sup> status and Chris will not signal (nor would an HIV<sup>−</sup> Pat; see payoffs); Pat will always accept a relationship if Chris agrees (Pat is an A-type; defined on page 178).

<sup>10</sup> Here S means signal HIV<sup>+</sup> and NS can imply HIV<sup>−</sup>. By contrast, in many games, a better-informed party signals (honestly or not) the presence of some advantageous characteristic (e.g., high qualifications for a job). While the S and NS names could be reversed without affecting outcomes, we choose this terminology because it more accurately reflects stylized relationship dynamics.



ascribes to an HIV<sup>+</sup> condition—which here constitutes the cost of signaling  $Sc$ . Here we assume that by signaling to Chris, Pat also discloses his type to at least some other members of society (*i.e.*, the knowledge will, at least eventually, leak out). In this sense, all signals are public. If an HIV<sup>+</sup> Pat fails to reveal his status to Chris, and if she accepts the relationship, Pat experiences internal guilt cost  $G$ . Should Chris enter a relationship with Pat when he is HIV<sup>+</sup>, she bears some *transmission risk*: the risk that he transmits HIV to her—a cost of entering a such a relationship. If Chris is uninformed about Pat's condition, she faces *transmission risk* is  $T$  ( $\tau$  if informed). Because an uninformed agent cannot take preventative measures,  $T > \tau$ . Note that both  $T$  and  $\tau$  depend (negatively) on the relevant society's medical infrastructure ( $M$ ) and that  $\tau$  also depends (again negatively) on a society's provision of HIV education and associated access to protection ( $E$ ).<sup>11</sup> Finally, should Chris enter a relationship with an HIV<sup>+</sup> Pat who has signaled his status, she experiences association stigma  $S_a$ —another potential cost of entering such a relationship. We now construct an evolutionary game that illustrates how social stigma affects the propensities of individuals to honestly signal or not and to subsequently accept or reject relationships.

For each of the three steps in the stage game, our evolutionary model establishes two possible phenotypes that agents may exhibit. While there are eight possible combinations of phenotypes, only four such combinations actually affect behavior. Nature's move in step 1 divides the population into two basic disease-condition phenotypes: HIV<sup>+</sup> with probability  $p$ , and HIV<sup>−</sup> with probability  $1 - p$ . For step two, there are two behavioral phenotypes: responsible types (REs) who would always signal if HIV<sup>+</sup> and always not signal if HIV<sup>−</sup>, and irresponsible types (IRs) who would never signal, regardless of their condition. The proportions of RE and IR players are, respectively,  $q$  and  $1 - q$ . Note that the RE-IR distinction does not affect the behavior of HIV<sup>−</sup> agents, since they would never signal (never pretend to be HIV<sup>+</sup>); accordingly, the model does not address this distinction. In contrast, among HIV<sup>+</sup> agents, the RE-IR distinction determines their response in stage 2. Stage three presents two additional behavioral phenotypes that pertain only to HIV<sup>−</sup> agents: each may be either accepting (A; an A-type), meaning he will accept a relationship regardless of the presence or absence of a signal; or cautious (C; a C-type), meaning she will always reject a relationship if she receives a signal, but always accept with no signal.<sup>12</sup> Among HIV<sup>−</sup> agents, these population proportions are respectively  $z$  and  $1 - z$ . For simplicity, we assume that all HIV<sup>+</sup> agents always accept relationships.<sup>13</sup>

Thus, there are four phenotype combinations (two sets of two) that actually affect agents' behavior:

- HIV<sup>+</sup> agents either signal or not, depending on whether they are RE or IR, but they always accept relationships.
- HIV<sup>−</sup> agents never signal, but depending on whether they are A- or C-types, they either always accept a relationship or do so only if they have not received an HIV<sup>+</sup> signal.

<sup>11</sup> For simplicity, we combine AIDs education and access to protection into the variable  $E$  and hereafter refer to it as AIDs education.

<sup>12</sup> In a more complicated model, a C-type agent could base his probability of acceptance on the population infection rate  $p$ . Another variation could introduce a third behavioral phenotype at this stage: conformists, who do what they expect others to do.

<sup>13</sup> In terms of infection, they have nothing to lose. Implicitly we assume that for IR agents,  $L > S_a$ .

In this evolutionary game, agents match randomly in pairs. Using the described payoffs, from Figure 1, we assign fitness payoffs to all possible matches of the four phenotype-based combinations, as shown in Table 1.

In this table, the cells that represent interactions between two  $HIV^+$  agents do not follow from Figure 1's limited case, and so merit further explanation. When two RE  $HIV^+$  agents interact, they accept relationships but also experience condition stigma because they have both signaled their condition. When an RE interacts with IR agent, they again accept the relationship. The former loses  $S_c$  from signaling. The latter does not experience  $S_c$ , but does face association stigma,  $S_a$ . Note that a quick examination of the cells in Table 1 suggests that the relative fitness of the RE and IR phenotypes depends critically on the relative magnitudes of social context variables  $S_c$ ,  $S_a$ , and  $G$ , as well as  $L$ ,  $\tau$ , and  $T$ .

**Table 1.** Fitness payoffs from interactions among different phenotypes.

Phenotype (proportion)	$HIV^+$ & RE $pq$	$HIV^+$ & IR $p(1-q)$	$HIV^-$ & A $(1-p)z$	$HIV^-$ & C $(1-p)(1-z)$
$HIV^+$ & RE $pq$	$L - S_c$ $L - S_c$	$L - S_c, L - S_a$	$L - S_c$ $L - \tau - S_a$	$-S_c, 0^*$
$HIV^+$ & IR $p(1-q)$	$L - S_a$ $L - S_c$	$L - G, L - G$	$L - G, L - T$	$L - G, L - T$
$HIV^-$ & A $(1-p)z$	$L - \tau - S_a$ $L - S_c$	$L - T, L - G$	$L, L$	$L, L$
$HIV^-$ & C $(1-p)(1-z)$	$0, -S_c^*$	$L - T, L - G$	$L, L$	$L, L$

**Legend:** \* Indicates no relationship formed.  $p$  signifies the  $HIV^+$  proportion of the population (from step 1);  $q$  signifies the portion of  $HIV^+$  agents who are responsible (RE) agents (in step 2); and  $z$  signifies portion of accepting (A)-types among the  $HIV^-$  agents (in stage 3). IR = irresponsible types.

To proceed and more fully integrate social context into our analysis, we assume that Table 1's social context variables,  $G$ ,  $S_c$ , and  $S_a$ , all respond to (are functions of) predominant characteristics within the population: these variables exhibit a type of conformity effect. Specifically, we assume that both  $G$  and  $S_c$  respond to  $q$  (the population proportion of RE types)—albeit in opposite fashions:  $G = G(q)$ ,  $\partial G / \partial q > 0$ ; and  $S_c = S_c(q)$ ,  $\partial S_c / \partial q < 0$ . The former relation could emerge from a social norm of honest reporting within a population whose degree of norm internalization (the extent to which individuals will follow a norm from an internal sense of obligation) depends at least to some degree on observed adherence by others.<sup>14</sup> Over longer time horizons, the emergence and strength of such a norm could reflect historical patterns of adherence. Similarly, widespread honest reporting of an  $HIV^+$  condition may diminish condition stigma. For association stigma  $S_a$ , we (analogously) assume that a greater population propensity to accept relationships with RE agents diminishes association stigma:  $S_a = S_a(z)$ ,  $\partial S_a / \partial z < 0$ .

<sup>14</sup> Norm internalization follows from ethical content Ferguson [10]. On the ethical content of norms see Bowles [8], Dequech [22], Elster [23,24], and Mengel [25].

We now specify the expected fitness payoff ( $W$ ), for each phenotype, based on their population proportions and the associated payoffs from the possible matches shown in Table 1. The following equations represent the respective fitness payoffs:

$$W(\text{RE}) = [p + (1 - p)z]L - S_c(q) \quad (1)$$

$$W(\text{IR}) = L - (1 - pq)G(q) - pqS_a(z) \quad (2)$$

$$W(\text{A}) = L - pq(\tau + S_a(z)) - p(1 - q)T \quad (3)$$

$$W(\text{C}) = (1 - pq)L - p(1 - q)T \quad (4)$$

Since the most important question with respect to HIV transmission concerns the propensity of HIV<sup>+</sup> agents to report, we first compare the relative fitness of strategies RE and IR within the HIV<sup>+</sup> population. Equation (1) states that the fitness of REs equals their  $L$  payoff, received from relationships with either HIV<sup>+</sup> or A-type HIV<sup>−</sup> agents, minus payoff  $S_c$ , received in all cases. Equation (2) states that the fitness of IRs equals their  $L$  payoff, received from all encounters, minus the guilt ( $G$ ) they experience from relationships with IRs as well as from all relationships with HIV<sup>−</sup> agents, minus the association stigma ( $S_a$ ) they receive from relationships with REs. REs then are fitter if  $W(\text{RE}) > W(\text{IR})$ ; that is, if

$$\Phi = G(q) + pq[S_a(z) - G(q)] - (1 - p)(1 - z)L - S_c(q) > 0 \quad (5)$$

Equation (5) states that REs exhibit greater fitness than IRs when the guilt that IRs experience from their fear of infecting other IRs and all HIV<sup>−</sup> agents plus the association stigma they receive from relationships with REs exceeds the their relative gains with respect to attaining relationships with HIV<sup>−</sup> C-types plus their avoidance (in all cases) of signaling cost  $S_c$ , accounting for the population proportions  $p$ ,  $q$  and  $z$ . Note the contrasting influences of  $S_c$  and  $S_a$  on  $\Phi$ .

Turning now to HIV<sup>−</sup> agents, we compare the fitness of A-types with that of C-types. Equation (3) states that A-type fitness equals payoff  $L$  (from all relationships) minus the sum of risk  $\tau$  plus  $S_a$  (both from REs), minus risk  $T$  (from IRs). In Equation (4), C-type fitness equals  $L$  (from all who are not RE) minus  $T$  (from all IRs). Hence  $W(\text{C}) > W(\text{A})$  if  $pqL + pq(\tau + S_a) > 0$ ; or if:

$$\Gamma = \tau + S_a(z) - L > 0 \quad (6)$$

C-types exhibit greater relative fitness if the disadvantage to A-types from risk  $\tau$  plus association stigma  $S_a$  exceeds their relative gain from always receiving  $L$ . The overall statement of (6) may be surprising because it suggests that, among HIV<sup>−</sup> agents, the relative fitness of C-types does not directly depend on population proportions  $p$ ,  $q$ , or  $z$ . In terms of Funk *et al.* [12],  $\Gamma$  does not depend on actual disease prevalence ( $p$ ) but rather on the joint influence of a type of belief-based information reflected in  $S_a$  (which does respond to  $z$ ) along with the actual probability of informed transmission ( $\tau$ ), both weighed against potential benefits from experiencing relationships ( $L$ ). Here, association stigma ( $S_a$ ) enhances the relative fitness of C-types, an outcome that would, *ceteris paribus*, reduce disease transmission.

We now consider possible evolutionary equilibria that can emerge from this model. Table 2 shows the existence and stability conditions for possible evolutionary equilibria for the RE and IR phenotypes (from Equation (5)) and the A and C phenotypes (from Equation (6)).

**Table 2.** Evolutionary existence and stability conditions.

ESS Outcome	Existence	Stability
RE is an ESS	(i) $(1 - p)G(q) + pS_a(z) > (1 - p)(1 - z)L + S_c(q)$ , as $q \rightarrow 1$	a. (i) holds for all $q$ or b. $\partial\Phi/\partial q > 0$ : $G' > S_c'$
IR is an ESS	(ii) $S_c(q) + (1 - p)(1 - z)L > G(q)$ , as $q \rightarrow 0$	c. (ii) holds for all $q$ or b. $\partial\Phi/\partial q > 0$ : $G' > S_c'$
Polymorphic $q^*$	(iii) $(1 - pq^*)G(q) + pq^*S_a(z) = (1 - p)(1 - z)L + S_c(q^*)$ , for any $q < 1$	d. $\partial\Phi/\partial q < 0$ : $G' < S_c'$
A is an ESS	(iv) $L > \tau + S_a(z)$ , as $z \rightarrow 1$	e. (iii) holds for all $z$ or f. $\partial\Gamma/\partial z < 0$ : $S_a' < 0$
C is an ESS	(v) $\tau + S_a(z) > L$ , as $z \rightarrow 0$	g. (iii) holds for all $z$ or f. $\partial\Gamma/\partial z < 0$ : $S_a' < 0$
Polymorphic $z^*$	(vi) $\tau + S_a(z^*) = L$ , for any $z \in [0, 1]$	h. $\partial\Gamma/\partial z > 0$ : $S_a' > 0$

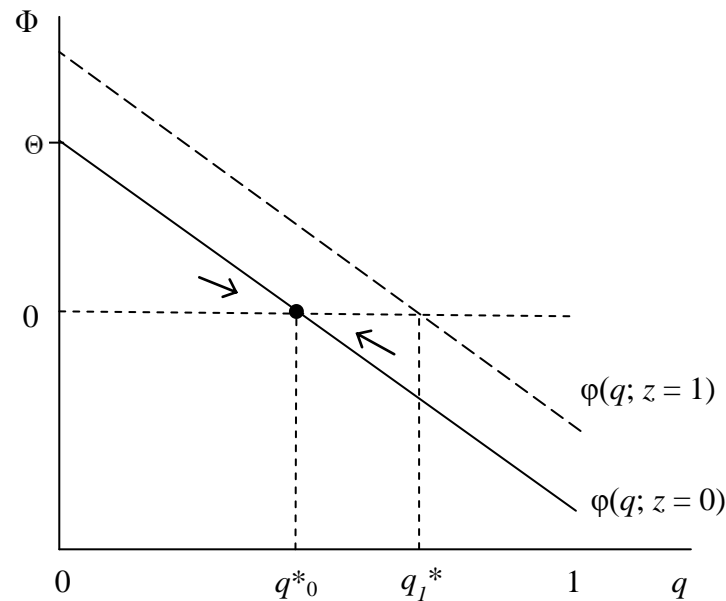
In principle, there are nine possible equilibrium phenotype combinations. There are four possible pairings of evolutionary stable phenotypes (corner solutions): (RE, A); (RE, C); (IR, A); and (IR, C).<sup>15</sup> And there are five possible evolutionary outcomes that involve polymorphic mixes of proportions  $q$  and  $z$  at specific equilibrium levels  $q^*$  and  $z^*$ : (RE,  $z^*$ ), (IR,  $z^*$ ), ( $q^*$ , A), ( $q^*$ , C), and ( $q^*$ ,  $z^*$ ).

Corner solutions are of interest only if they are stable. Accordingly, we first examine the stability conditions phenotypes RE and IR. Given the previously discussed derivatives of the  $G(q)$  and  $S_c(q)$  functions, stability condition (b) does not hold. Thus, RE can be an ESS only if (a) holds and IR can be an ESS only if (c) holds. While these two outcomes are mathematically possible, we do not expect to observe all RE or all IR behavior in any society.<sup>16</sup> For this reason, we focus on the polymorphic  $q^*$  outcome (iii).

The  $q^*$  component of either equilibrium outcome represents a stable (or possibly oscillating) mix of the RE and IR phenotypes. Arguably such mixes (possibly oscillating) obtain in virtually all societies. Figure 2 shows a partial equilibrium representation, from a simplified linear evolutionary RE-IR game, with  $z$  held constant, that illustrates the determination of  $q^*$ . In this partial equilibrium analysis, interactions between RE and IR agents resemble an evolutionary game of chicken.

<sup>15</sup> Each member of these pairs is an evolutionary stable strategy (an ESS). A strategy is an ESS if, once it has attained significant predominance in a population (as in 99%), it cannot be successfully invaded by a new “mutant” strategy—because (in that region) the mutant receives lower fitness payoffs. Note that the ESS concept does not specify what would happen in cases with a substantial proportion of mutants (e.g., 30%).

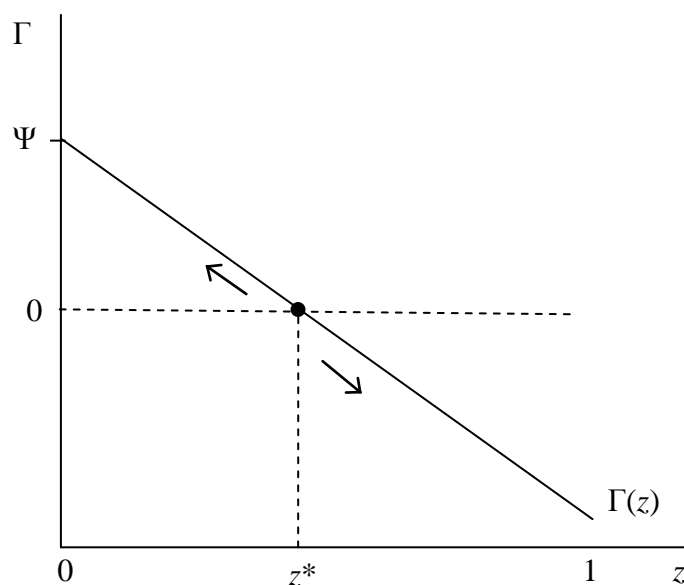
<sup>16</sup> Our brief review of relevant empirical literature clearly suggests a mix of honest reporting and concealment.

**Figure 2.** Relative fitness of RE and IR phenotypes.

**Legend:**  $\Phi$  relative fitness of phenotype RE;  $q$  proportion of RE in the population;  $\Theta = G_0 + p_0 q(S_a - G_0) - (1 - p)(1 - z)L + S_{c0}$ , where  $G_0$ ,  $S_{a0}$  and  $p_0$  signify the relevant values at  $q = 0$ ; symbol  $\rightarrow$  shows the path of adjustment. The negative slope of the function arises from conformity effects  $\partial G/\partial q > 0$  and  $\partial S_c/\partial q < 0$ .

Turning to stability conditions for partial equilibria that include the A and C phenotypes, we see that condition (f) is consistent with our specification of the  $S_a$  function. Hence we focus on equilibria that include all C and all A. While arguably this partial-equilibrium result oversimplifies real-world interactions, where we expect to observe both types, it nonetheless offers several interesting implications, discussed below.<sup>17</sup> Accordingly, we interpret the A and C equilibria as predominantly A or predominantly C. Concerning existence conditions for A and C equilibria, (iii) and (iv) are both possible, depending on the specified relationships. Moreover, an unstable internal equilibrium at  $z^*$  attains whenever (vi) holds. In this (quite interesting) case, a partial equilibrium game between phenotypes A and C exhibits the properties of evolutionary assurance. The internal equilibrium  $z^*$  signifies a critical-mass tipping point. For any initial population ratio  $z < z^*$  ( $z > z^*$ ), a positive-feedback adaptation dynamic will push the equilibrium to an all A (all C) equilibrium. Figure 3 illustrates this partial-equilibrium configuration.

<sup>17</sup> A more complete (and far more complicated) model would include full specification of differential replicator equations for all four phenotypes (specified over time) and would consider long-term implications of changes in population ratios  $z$ ,  $q$ , and  $p$  on all four phenotypes. The conclusion mentions this possible extension.

**Figure 3.** Relative fitness of A and C phenotypes.

**Legend:**  $\Gamma$  relative fitness of phenotype C;  $z$  population proportion of the A phenotype;  $\Psi = \tau + S_a(0) - L$ , where  $S_a(0)$  signifies  $S_a(q)$  at  $q = 0$ ; symbol  $\rightarrow$  shows the path of adjustment. The negative slope of  $\Gamma(z)$  arises from conformity effects  $\partial S_a / \partial z < 0$ .

We thus end up with two possible stable combined evolutionary equilibria among our four phenotypes,  $(q^*, A)$  and  $(q^*, C)$ , with critical-mass tipping point (unstable internal partial equilibrium  $z^*$ ) demarcating two regions that exhibit reinforcing tendencies toward one equilibrium combination or the other.

We now address comparative statics from this model, first examining influences relative fitness of RE vs. IRs and then A-types vs. C-types. Table 3 shows the relevant partial derivatives.

**Table 3.** Comparative statics.

A. RE vs. IR Fitness	
Partial Derivative	Statement, $\Phi$ and $q^*$ decrease (d) or increase (i) in:
(i) $\partial \Phi / \partial S_c = -1$	Condition stigma (d)
(ii) $\partial \Phi / \partial S_a = pq$	Association stigma (i)
(iii) $\partial \Phi / \partial L = -(1-p)(1-z)$	Relationship utility (d)
(iv) $\partial \Phi / \partial G = 1 - pq$	Guilt (i)
(v) $\partial \Phi / \partial z = (1-p)L$	The population proportion of A-types (i)
(vi) $\partial \Phi / \partial p = (1-z)L + qS_a - qG$	The population proportion of HIV <sup>+</sup> agents (i) or (d); if $(1-z)L + qS_a > qG$ , then (i).
B. A vs. C Fitness	
Partial Derivative	Statement, $\Gamma$ and $\Psi$ decrease (d) or increase (i) in:
(vii) $\partial \Gamma / \partial \tau = 1$	Informed transmission (i)
(viii) $\partial \Gamma / \partial S_a = 1$	Association stigma (i)
(ix) $\partial \Gamma / \partial L = 1$	Relationship value (d)

First note that results (i) and (ii) imply that condition and association stigma have opposite effects on  $\Phi$ , since the former applies to REs and the latter to IRs. In (iii),  $\Phi$  decreases in  $L$  because IRs can

successfully enter relationships with C-types (who exist in proportion  $(1 - p)(1 - z)$ ) whereas REs cannot. By contrast, from (iv),  $\Phi$  increases in  $G$  because only IRs experience guilt for not informing (all non-RE) partners about their HIV<sup>+</sup> condition. These four results apply to both stable equilibrium combinations  $(q^*, A)$  and  $(q^*, C)$ . Note that all of these four effects shift Figure 2's intercept  $\Theta$  in the appropriate direction; all negative influences on  $\Phi$  reduce the equilibrium  $q^*$  and *vice versa*.

The two population results (v and vi.) are somewhat more complicated. In (v),  $\Phi$  increases in  $z$  because A-types, unlike C-types, accept relationships with REs. This result implies that  $(q^*, A)$  equilibrium will occur at a higher  $q^*$  than a  $(q^*, C)$  equilibrium, as shown by the difference between  $q_1^*$  and  $q_0^*$  in Figure 2. Under the stated condition in (vi),  $\Phi$  increases in  $p$  because an RE's disadvantageous access to  $L$  decreases in the population proportion of HIV<sup>+</sup> ( $p$ ) and also the negative effects of  $S_a$  on IRs increase in  $p$ . This final outcome implies at least some negative feedback in transmission of the disease—at least at high levels of  $p$ .

The A vs. C results shown in section B are straightforward. Increases in  $\tau$  or  $S_a$  increase Figure 3's intercept  $\Psi$  upward, pushing the tipping point  $z^*$  closer to 1, rendering an all C equilibrium (at  $z = 0$ ) more difficult to reverse.<sup>18</sup> An increase in  $L$  has the opposite effects. Note, however, that the influence of  $S_a$  on  $z$  to some degree counteracts result (ii).<sup>19</sup>

On this foundation, we now apply our evolutionary model to the SIR model of infection transmission.

## 2.2. The SIR Model of Infection

One may use the SIR model to depict the time trend in HIV infection over time. Our evolutionary model informs this analysis in two fashions: first, we may incorporate specific variables and functions from our model into the SIR model to derive a number of hypotheses concerning the spread of HIV; in particular, our variables influence the level of the SIR mixing coefficient ( $\beta$ ). Second, both the  $(q^*, A)$  and  $(q^*, C)$  equilibria suggest a certain amount of stability for the mixing coefficient  $\beta$ . Third, while the distinction between these two evolutionary equilibria likely affects the level of  $\beta$ , counteracting effects of ratio  $z$  on determinates of  $\beta$  render the direction of influence unclear. We begin with a brief explanation of the SIR model before relating it to our evolutionary variables, functions, and equilibria.

Here is a summary of the original SIR model. Consider a specific strain of virus, such as the Hong Kong flu, that spreads via direct contact, and for which some agents develop immunity after being infected. There are three types of agents: susceptible (S), infected (I), and recovered or immune (R). At time period  $t = 0$ , one agent in the fixed population (of size  $N$ ) is infected. Let  $s(t)$ ,  $i(t)$ , and  $r(t)$  designate the proportions of  $N$  that, at time  $t \geq 0$ , are (respectively) S, I and R. Note that for all  $t$ ,  $s(t) + i(t) + r(t) = 1$ . Infected agents transmit the virus to others at a constant per-period rate  $\beta$  (sometimes called the mixing rate).<sup>20</sup> During each period  $t$ , a fraction  $\sigma$  of the infected agents recover. Note that only susceptible agents can become infected.

<sup>18</sup> The basin of attraction around an all C-type equilibrium increases.

<sup>19</sup> We assume that this second-order effect does not outweigh the first-order effect shown in Table 3, (ii).

<sup>20</sup> We assume homogeneous mixing among agents (effectively no social network), hence parameter  $\beta$  characterizes all social interaction that affects disease transmission.

The following equations describe the time path of the three types of agents:

$$ds/dt = -\beta s(t)i(t) \quad (7)$$

$$dr/dt = \sigma i(t) \quad (8)$$

$$di/dt = \beta s(t)i(t) - \sigma i(t) \quad (9).$$

Equation (7) states that the pool of susceptible agents declines steadily as more become infected (no longer susceptible). Mathematically, Equation (7) follows from the definitions of  $\beta$ ,  $s(t)$ , and  $i(t)$ . Equation (8) states that the rate of recovery equals the per-period recovery fraction ( $\sigma$ ) multiplied by the portion infected. In Equation (9), the time trend for infection is negative that of susceptibility (hence a positive entry) minus the rate of recovery.<sup>21</sup> Note that the SIR model's dynamics and outcomes depend entirely on the relative sizes of its two parameters—the mixing coefficient  $\beta$  and the recovery rate  $\sigma$ . For example, if  $\beta < \sigma$ , the disease eventually dies out. Policy implications are simple but general: to slow the spread of disease, reduce mixing and increase the recovery fraction via treatment.<sup>22</sup>

Following the assertion of Funk *et al.* [12] that all disease modeling can be expressed in terms of the SIR model, we now relate our evolutionary model to SIR terms  $s$ ,  $i$ ,  $r$ , and their respective time trends, to  $\sigma$ , and especially to  $\beta$ . Note first that because there is no immunity to HIV among non-infected humans, HIV<sup>+</sup> agents can only “recover” (*i.e.*, become biologically incapable of transmitting the disease) by dying. Hence,  $dr/dt$  in Equation (8) is the population mortality rate, and its parameter  $\sigma$  is the per-infection mortality rate. On this basis, the terms  $s(t)$  and  $i(t)$  must respectively be time-delineated variables  $(1 - p)$  and  $p$ : all HIV<sup>−</sup> agents are susceptible, and all HIV<sup>+</sup> agents are (by definition) infected:  $s(t) = (1 - p_t)$  and  $i(t) = p_t$ . SIR Equations (7) and (9), then, describe the time path of the two disease-status phenotypes from our evolutionary model.

We now use variables from our evolutionary model to specify factors that influence the time trends in Equations (8) and (9).<sup>23</sup> Concerning the mortality rate, we rewrite Equation (8) as  $dr/dt = \sigma i(t) = \sigma p_t$ . To relate this equation to social context, we now stipulate that the per-infection mortality rate  $\sigma$  depends on the state of HIV education ( $E$ ) for the society in question and on its medical system ( $M$ ), representing its medical technology, medical infrastructure, and availability of treatment (hereafter medical infrastructure). We take  $M$  to be exogenous, whereas  $E$  includes exogenous provision of AIDS education ( $E_o$ ; *e.g.*, relevant infrastructure and technology) as well as an endogenous element that responds to the total (HIV-related) social stigma in the society  $S_s$ , itself a weighted average of  $S_c$  and  $S_a$ :  $S_s = \lambda S_c + (1 - \lambda)S_a$ ;  $\lambda \in [0,1]$ .  $S_s$  impedes a society's provision and absorption of AIDS education. Accordingly, we rewrite Equation (8) to include a mortality rate function:

$$dr/dt = \sigma i(t) = \sigma p_t = \sigma(M, E(E_o, S_s))p_t \quad (10)$$

where  $\partial\sigma/\partial M < 0$  and  $\partial\sigma/\partial E < 0$ . Because  $\partial E/\partial S_s < 0$ ,  $\partial\sigma/\partial S_s > 0$ . Both types of social stigma, unfortunately, enhance a society's HIV mortality rate, *ceteris paribus*.

More fundamentally, our evolutionary model can illustrate how social context influences HIV transmission via mixing coefficient  $\beta$ . We simplify the remaining analysis by abstracting from

<sup>21</sup> This relation follows from the requirement that proportions  $s(t)$ ,  $i(t)$ , and  $r(t)$  must always sum to 1; hence, the three time trends sum to 0. An intuitive explanation (with slightly different notation) appears at [26].

<sup>22</sup> A more complicated model could add immunization.

<sup>23</sup> It is easy to deduce influences on Equation (7) from our discussion of Equation (9).



mortality rate  $\sigma$  and also assuming a zero birth rate, so that the total population remains fixed at size  $N$ . We begin by expressing time trend for  $p(t)$ . The proportion  $p$  of HIV<sup>+</sup> agents at time  $t$  is:

$$p_t = p_0 + \int_{t=0}^t \frac{dp}{dt} dt \quad (11)$$

where  $p_0$  signifies the level of infection when HIV was first introduced to the society. To simplify notation, let  $dp_t$  signify  $(dp/dt)dt$ , the rate of change in  $p$  (the rate of new infection) at time  $t$ . For a given period  $t$ ,  $dp_t$  equals the probability that an HIV<sup>+</sup> agent encounters an HIV<sup>−</sup> agent multiplied by the associated rate of HIV transmission. Noting the definitional equivalence of  $i(t)$  and  $p_t$ , and abstracting from both birth and mortality rates, we have:

$$di/dt \equiv dp_t = \beta s(t)i(t) = p_t(1 - p_t)N[qz\tau + (1 - q)T] \quad (12)$$

Equation (12) states that the aggregate rate of HIV transmission for a population of size  $N$  equals the probability of an HIV<sup>+</sup>/HIV<sup>−</sup> encounter multiplied by the probability that an RE type transmits the disease (only to an A-type, an informed transmission) plus the probability that an IR type transmits the disease (to either an A-type or a C-type, an uninformed transmission).<sup>24</sup> The rate of informed transmission, in turn, depends on a society's medical infrastructure and HIV education, so that we can write  $\tau = \tau(M, E(E_o, S_s))$ . Using this function, Equation (12) implies that the (not necessarily constant) SIR mixing coefficient for HIV transmission is:

$$\beta = N[q(\Phi)z(\Gamma)\tau(M, E(S_c, S_a)) + (1 - q)T(M)] \quad (13)$$

We immediately see that  $\partial\beta/\partial q < 0$  and  $\partial\beta/\partial z > 0$ : mixing decreases in the proportion of REs and increases in the proportion of A-types. Prior analysis has shown that equilibrium levels  $q^*$  and  $z^*$  depend on their respective evolutionary fitness  $\Phi$  and  $\Gamma$ , which in turn depend on social context variables  $G$ ,  $S_c$ , and  $S_a$ , as well as on  $L$ . On this basis, Table 4 translates outcomes from Table 3 into social influences on the SIR mixing coefficient  $\beta$ .

**Table 4.** Social influences on the SIR (susceptible-infected-recovered) model mixing coefficient  $\beta$  for HIV.

Partial derivatives for $\Phi$ and $\Gamma$ from Table 3	Impacts on $\tau$ (via $E$ )	Partial derivatives with respect to $\beta$
$\partial\Phi/\partial S_c < 0$	$\partial\tau/\partial S_c > 0$	(x) $\partial\beta/\partial S_c > 0$
$\partial\Phi/\partial L < 0$ and $\partial\Gamma/\partial L < 0$	--	(xi) $\partial\beta/\partial L > 0$
$\partial\Phi/\partial G > 0$	--	(xii) $\partial\beta/\partial G < 0$
$\partial\Phi/\partial S_a > 0$ and $\partial\Gamma/\partial S_a > 0$	$\partial\tau/\partial S_a > 0$	(xiii) $\partial\beta/\partial S_a$ indeterminate (see below)
$\partial\Phi/\partial p > 0$ (usually)	--	(xiv) $\partial\beta/\partial p = (\partial q/\partial p)(\partial\beta/\partial q) < 0$

<sup>24</sup> The probability of a HIV<sup>+</sup> HIV<sup>−</sup> match between two partners is  $2p(1 - p)$ . There are  $N/2$  possible matches in population of size  $N$ . Note that Equation (12) accounts for the probability of a rejected relationship in an RE/C-type match.

Overall, the HIV mixing coefficient  $\beta$  increases in relationship value  $L$  and condition stigma  $S_c$  and decreases in guilt  $G$ . Association stigma is more complicated, as shown in Equation (14).

$$\partial\beta/\partial S_a = (\partial\beta/\partial q)(\partial q/\partial S_a)z\tau + (\partial\beta/\partial z)(\partial z/\partial S_a)q\tau + (\partial\tau/\partial z)(\partial\tau/\partial S_a)qz \quad (14)$$

The first two products in Equation (14) are negative, but the third is positive. Hence the overall influence of  $S_a$  on  $\beta$  is negative if and only if the absolute values of the first two terms exceed that of the third term.<sup>25</sup> If so, condition stigma and association stigma have opposite influences on  $\beta$ . From (xiv), coefficient  $\beta$  (usually) decreases in HIV<sup>+</sup> proportion  $p$ . By itself, (xv) suggests a type of negative feedback that could limit the extent of infection. Even so, as long as  $\beta > 0$ , some transmission will occur.<sup>26</sup> Moreover, the overall effect of  $p$  on disease transmission also depends on the level  $p_i$  in Equation (12). Here we see that  $\partial dp_i/\partial p > 0$ . Hence the overall impact of level  $p_i$  on HIV transmission in Equation (12) is indeterminate.

Concerning evolutionary equilibria, our model makes two basic statements. First, at either equilibrium, the polymorphic value  $q^*$  (or an oscillating range), lends some stability to the mixing coefficient  $\beta$ , as does a stable outcome at either all A or all C. Second, the distinction between the  $(q^*, A)$  and  $(q^*, C)$  equilibria affects the level of coefficient  $\beta$  in two counteracting fashions. On one hand, from Equation (12), we see that  $d\beta/dz > 0$ , implying that the  $(q^*, A)$  equilibrium generates a higher coefficient  $\beta$ , *ceteris paribus*. On the other hand, result (v) from Table 3 (and the drawing in Figure 2) show that the level  $q^*$  in  $(q^*, A)$  exceeds that in  $(q^*, C)$ , implying (from Equation (13)) a lower value of  $\beta$ . Without additional specification, we cannot determine which of these effects has a larger magnitude.

Finally, we consider the overall impact of total social stigma  $S_s$  on the mixing coefficient and on disease transmission. This impact is negative if and only if  $\partial\beta/\partial S_a < 0$  and if  $(1 - \lambda)|(\partial\beta/\partial S_a)| > \lambda(\partial\beta/\partial S_c)$ . Rearranging, the latter condition, we have:

$$\lambda < \frac{|\partial\beta/\partial S_a|}{|\partial\beta/\partial S_a| + \partial\beta/\partial S_c} \quad (15)$$

In circumstances when  $\partial\beta/\partial S_a < 0$ , Equation (15) implies that aggregate social stigma reduces the rate of HIV transmission  $di/dt$  if and only if the relative weight of condition stigma within total stigma is less than the association stigma's share of the total effect (in terms of absolute value) that social stigma exerts on HIV transmission. Hence, in addition to the opposing influences of the two types of social stigma on the fitness of the (honest) RE phenotype, the relative weights of the two types of stigma can affect a society's rate of HIV transmission such that the greater the weight of condition stigma, the more likely it is that overall social stigma enhances the transmission of HIV.

### 3. Conclusions

This paper has combined an evolutionary game-theoretic model of HIV transmission within a society with the SIR model of disease transmission, noting the related influences of social stigma, and AIDS education on the propensities of individuals to honestly report their HIV status, the propensities

<sup>25</sup>  $\partial\beta/\partial S_a < 0$  if and only if  $|(\partial\beta/\partial q)(\partial q/\partial S_a)z\tau| + |(\partial\beta/\partial z)(\partial z/\partial S_a)q\tau| > |(\partial\tau/\partial z)(\partial\tau/\partial S_a)qz|$ .

<sup>26</sup> Whether society settles on an equilibrium  $p$  will depend (*ceteris paribus*) on the relationship between birth and death rates—a topic beyond the scope of this paper.

to accept or reject relationships on the basis of such reports, the associated within-relationship risks of transmission, and the ensuing population rate of transmission of the virus.

We now list our key findings, each of which can generate one or more testable hypotheses, in three basic categories:

A. Our discussion of evolutionary equilibria implies the following:

1. Among the  $HIV^+$  population, the behavioral traits of honest and dishonest reporting of an  $HIV^+$  status (the RE and IR phenotypes) can both survive. More precisely, both phenotypes coexist in a polymorphic evolutionary equilibrium, as signified by a stable (or possibly oscillating) population ratio of REs ( $q^*$ ). Arguably, such mixes exist in most societies.
2. Among the  $HIV^-$  population, the behavioral traits of unconditional and conditional acceptance of relationships (the A and C phenotypes) can each be evolutionary stable strategies. Moreover, the accompanying evolutionary dynamics likely exhibit a positive-feedback dynamic whereby increased prevalence of phenotype A (C) above (below) critical mass tipping point (unstable internal equilibrium  $z^*$ ) enhances its own fitness. We interpret this result to imply two that predominance (rather than 100%) of either A- or C-types will accompany a  $q^*$  equilibrium.
3. The evolutionary model thus generates two stable evolutionary equilibrium combinations of phenotypes: a polymorphic mix  $q^*$  of the RE and IR phenotypes among the  $HIV^+$  population, combined with either a predominantly A or predominantly C phenotype among the  $HIV^-$  population.

B. Concerning key influences on the nature of the evolutionary equilibria, we find:

4. The two types of social stigma—condition and association stigma—exert opposing direct influences on the fitness  $\Phi$  of the RE phenotype. Whereas condition stigma compromises RE fitness, reducing equilibrium proportion  $q^*$ , association stigma ( $S_a$ ) enhances  $\Phi$  and hence increases  $q^*$ , *ceteris paribus*.
5. Fitness  $\Phi$  and proportion  $q^*$  also increase in guilt  $G$  and decrease in relationship value  $L$ .
6.  $\Phi$  and  $q^*$  increase in society's proportion  $z$  of A-types and, under specified conditions, also increase in disease prevalence  $p$ , potentially generating some negative-feedback with respect to disease prevalence.
7. C-type fitness  $\Gamma$  increases in  $S_a$  and decreases in  $L$ .

C. Combining these findings with the SIR model, we find:

8. Condition stigma  $S_c$  unambiguously enhances disease transmission as represented by SIR mixing coefficient  $\beta$  and the rate  $di/dt$ . In this regard,  $S_c$  both reduces  $q^*$  (from finding 4) and reduces acceptance of AIDS education ( $E$ ), ultimately enhancing (informed) transmission rate  $\tau$ .
9. Association stigma  $S_a$ , by contrast, can exert a negative impact on HIV transmission: whereas  $S_a$  enhances transmission by reducing  $E$ ,  $S_a$  also increases  $q^*$  and enhances  $\Gamma$  (as in findings 4 and 7).

10. Either evolutionary equilibrium  $(q^*, A)$  or  $(q^*, C)$  lends some stability to the SIR mixing parameter  $\beta$ .
11. The distinction between the  $(q^*, A)$  and  $(q^*, C)$  equilibria, has an ambiguous impact on  $\beta$ : On one hand, an all or predominantly A-type equilibrium leads to relatively high  $q^*$ , tending to decrease  $\beta$ . On the other hand, as shown in Equation (13),  $\beta$  increases in  $z$ .
12. Finding 9 notwithstanding, the greater the share of condition stigma within a society's overall social stigma, the greater the likelihood that overall social stigma enhances the SIR rate of HIV transmission.

### Extensions

This paper offers a preliminary examination of the evolutionary dynamics of HIV transmission. Our approach can be extended in four basic fashions that relate to each of the following: the specification of phenotypes; fuller specification of the entire model; alternative evolutionary approaches; and extensions to network analysis.

Concerning phenotypes, the C-type could include a more precise criterion for acceptance: it could stipulate that cautious players reject relationships in the presence of a signal (as in the present model) but only accept relationships with a probability equal to HIV<sup>+</sup> prevalence  $(1 - p)$  in the relevant society. In addition, the model could incorporate a third, and more extreme acceptance phenotype: a fearful agent (F-type) who would reject all relationships.<sup>27</sup> Another possible phenotype (as an addition or substitute) could be a conforming type for whom relationship acceptance depends on the average population rate of acceptance. Such a modification would likely enhance the positive-feedback A vs. C outcome in the present model and could generate additional avenues for positive-feedback effects, possibly suggesting additional critical-mass tipping points.

Concerning the full model, a thorough description of the present evolutionary mechanism would involve stipulating and analyzing its dynamic replicator equations, a set of differential equations that would specify time trends for each phenotype. Although quite complicated, such specification would permit a thorough description of any polymorphic IR-RE equilibrium (e.g., the precise nature of oscillation) and would likely condition our result that allows both C- and A-types to be evolutionary stable strategies. An alternative, though related, modeling approach for the same set of specifications would be to incorporate the basic dynamics of this model into an agent-based model. A systematic series of simulations could then illustrate multiple evolutionary developments.<sup>28</sup>

As another variant on evolutionary approaches, one could replace our (direct) evolutionary model with an indirect evolutionary approach [27,28] whereby behavior in the stage-game reflects both social and material preferences, but long-run fitness depends on relative material payoffs.

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<sup>27</sup> Preliminary analysis suggests that the more complex C-type would not alter key results of a polymorphic IR-RE equilibrium and positive feedback dynamics for A- and C-types. Adding an F-type as a fifth behavioral phenotype would complicate the model considerably, but a prior analysis suggested this addition did not significantly affect outcomes; but its inclusion could generate a negative-feedback dynamic for relationship acceptance, operating above a tipping-point level of  $p$ , where the A-type would lose relative fitness.

<sup>28</sup> For a discussion of agent-based models, see Epstein [21].

Finally, this model lends itself to a social network analysis of its SIR component (as in the seventh assertion of Funk *et al.* [12]). Because an agent's degree of social connectedness can significantly influence disease transmission via social mixing, social network analysis can specify how social connections (within, say, a network of friends) affect a more extensive SIR model's rates of transmission at both a local level (as in a  $\beta_{Li}$  for each subgroup  $i$ ) and population level ( $\beta_p$ ).<sup>29</sup>

In all cases, this paper's combination of an evolutionary game-theoretic model of HIV transmission with the SIR disease transmission model offers a foundation for subsequent empirical and theoretical examination of society's collective-action problem with respect to HIV transmission. Moreover, our models can also serve as a basis for more general analysis of disease transmission in society.

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## Authors Contribution

Both authors contributed to this paper. The initial idea for the paper including the initial version of the signaling game and basic evolutionary model appeared in a draft written by TKN several years ago. WDF revised and added substantially to this draft several times before and after its initial submission to *Economics*, adding the review of the literature, more fully specifying some of the initial equations, specifying the signaling game as a stage game for the evolutionary game, specifying the evolutionary equilibrium conditions with associated figures and tables, and adding the discussion of the SIR model of disease transmission and its relation to the evolutionary model.

## Conflicts of Interest

The authors declare no conflict of interest.

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<sup>29</sup> For a dramatic example of how connectedness affected HIV transmission in its early history, see Shilts [29]. For an introduction to social network theory with an application to the SIR model, see Ferguson [10]; for detailed treatments of social network analysis see Jackson [30] and Vega-Redondo [31].

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