Behavior-Disease Models with respect to HIV Dynamics

Aidan Grennell

Western Carolina University

Abstract

Expanding on the Susceptible-Infected-Recovered (SIR) epidemiological models, we review "Towards a Characterization of Behavior-Disease Models," which focuses on the impact of fear of a disease on behavior changes that impact the transmission of the disease within a society. The reviewed paper focuses on the impact of local and global prevalence-based fear, as well as local, belief-based fear and the respective impact on societal behavior as it affects the disease transmission process. This paper seeks to build on the ideas presented, interpreting the generalized results with respect to HIV epidemiological models.

Introduction

Epidemiological models have been an invaluable tool in understanding and describing disease processes. As epidemiological models have developed, they can be adapted to describe many types of infectious processes, from short-term, recoverable diseases to chronic and life-threatening diseases. While many models have been developed that describe behavioral changes that are externally imposed (for example, models that incorporate a period of quarantine), very few models exist that describe how self-imposed behavioral changes impact the disease processes.

In "Toward a Characterization of Behavior-Disease Models," by Nicola Perra, Duygu Balcan, Bruno Goncalves, and Alessandro Vespignani [7] present several models to describe two competing and interrelated disease processes: the biological disease spread coupled with the fear of the disease, where fear is a contagious process that impacts how the biological process manifests within a community. The paper addresses three distinct types of fear, adapting the basic Susceptible-Infected-Recovered (SIR) model to incorporate each of these fear contagions. The model is general, and is focused on application toward diseases with a short infectious period with minimal long-term effects on the individuals once they have recovered. Although the basic SIR model lends itself well to modeling diseases from which recovery is possible, with appropriate adaptations, SIR models can also describe the dynamics of chronic or terminal illnesses [1]. The behavioral adaptations presented by Perra et al. [7] are also applicable to different stages of a terminal disease process as it manifests within a community.

In this paper, we will explore how the behavior-disease model is formulated, as well as explore how the model might be applied to HIV models. We address model construction and some model analysis, as well as present a possible model for application to a behavior-disease dynamic for HIV.

Background Information

S-I-R Model

We begin by considering a basic SIR continuous-time, compartmental epidemiological model. In its most basic form, the model assumes a closed population, N, is partitioned into three separate sub-populations where members of the population transition through the different stages of a disease process, from susceptible to infected to recovered [1, 3, 7]. This can be seen schematically with the following diagram

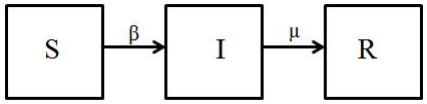

Figure 1: Schematic Representation of S-I-R Model

where β is the rate of transmission from susceptible to infected and μ is the rate of transmission from infected to recovered. We translate this diagram into the following nonlinear system of differential equations, given by

$$\begin{aligned} \frac{dS}{dt} &= -\beta S(t)\frac{I(t)}{N} \\ \frac{dI}{dt} &= \beta S(t)\frac{I(t)}{N} - \mu I(t) \\ \frac{dR}{dt} &= \mu I(t) \end{aligned}$$

We note in the model construction that a person transitions from susceptible to infected by interaction with a portion of the infected population, whereas recovery occurs with no interaction term. Furthermore, we note that

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$$

therefore imposing the constraint that the total population is constant.

Behavior-Disease Model

The focus of Perra et al. [7] was to couple two interacting models describing distinct contagious processes, a biological disease and the fear the disease. They focused on three distinct types of fear that lead to self-imposed behavioral changes that impact the transition parameters of the biological process. The first type of fear they consider is local prevalence-based fear, where susceptible individuals adopt behavior changes as a

result of interacting with an infected individual. The second type of fear considered is global prevalence-based fear, where individuals adopt behavioral changes as a result of information available to the general public, usually via television, newspapers, and the Internet. The final type of fear considered is local belief-based fear, which causes behavioral changes via contact with individuals who have already acquired a fear of the disease. Each type of fear is caused by different methods and therefore impact the biological disease process differently [7].

Main Results

Constructing the Behavior-Disease Model

In order to construct a coupled model, a fourth compartment is defined for the SIR model, which classifies the portion of the population who are still susceptible to the biological disease process, but have altered their behavior as a result of fear. This compartment, denoted by S^F, still requires an interaction with an infected individual in order to transition to the biologically infected class, but the rate of transmission is affected since those in the S^F compartment have incorporated behavioral changes to prevent biological illness. Thus we have that

$$S^F + I \xrightarrow{r_\beta \beta} 2I$$

where $0 \leq r_\beta < 1$ modulates the level of self-induced behavioral change. This is the premise of the introduction of a behavioral component to an epidemiological model; however, much like an SIR model, this modification can vary depending on how fear is introduced and spreads within a population [7].

Although the original paper considered three distinct types of fear, we will only consider the type that is most applicable to the current HIV epidemic. Local, prevalence-based fear is applicable to the early stages of the disease spread in the 1980's, where fear of HIV directly related to exposure to the virus [4, 5]. Local belief-based fear is more applicable to HIV in the 1990's and early 2000's, where fear was a self-perpetuating contagion, and interacting with someone who had fear of HIV was more likely to generate more fear [5, 6]. At the current state in the epidemic process, however, most people are familiar with the dangers of HIV via public information, so global prevalence-based fear is the most applicable to this disease process.

If we denote the rate of transmission of fear as β_F, then Perra et al. [7] defines the contagion term as

$$\beta_F(1 - e^{-\delta I(t)})$$

for $0 < \delta \leq 1$. Since global prevalence-based fear is not dependent on interaction with an infected individual, but does consider the absolute number of infected individuals, the fear contagion process acts on the entire population but is still dependent on the number of infected individuals. Thus we have the following system

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S(t)\frac{I(t)}{N} - \beta_F S(t)[1 - e^{-\delta I(t)}] + \mu_F S^F(t)\left[\frac{S(t) + R(t)}{N}\right] \\
\frac{dS^F}{dt} &= -r_\beta \beta S^F(t)\frac{I(t)}{N} + \beta_F S(t)[1 - e^{-\delta I(t)} - \mu_F S^F(t)\left[\frac{S(t) + R(t)}{N}\right] \\
\frac{dI}{dt} &= -\mu I(t) + \beta S(t)\frac{I(t)}{N} + r_\beta \beta S^F(t)\frac{I(t)}{N} \\
\frac{dR}{dt} &= \mu I(t)
\end{aligned}$$

noting that

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dS^F}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$$

which gives a constant population. The parameters are as follows:

Parameter	**Interpretation**
β	Average rate of new infections of disease
β_F	Average rate of new infections of fear
μ_F	Rate at which a susceptible individual modifies behavior
r_β	Rate of behavior change
μ	Rate of recover

This is the model that is presented as a general model by Perra et al. [7] to describe the coupling of a global prevalence-based fear contagion with a disease process [7].

Results of the Behavior-Disease Model

A key component of epidemiological models is the concept of a basic reproduction number, denoted R_0. The basic reproduction number is a method of quantifying the dynamics of a disease by consider the average number of new infections that are generated from a single infected person in a population of susceptible individuals [1]. If $R_0 < 1$, then the model tends toward the disease-free equilibrium. However, if $R_0 > 1$, then the disease spreads and becomes an epidemic.

In considering the behavior-disease model, if the disease spreads faster than the fear of the disease, then the basic reproduction number is defined as $R_0 = \frac{\beta}{\mu}$, which is standard for SIR models [1]. Biologically, we interpret R_0 to be a ratio of the rate of new infections to the rate of recovery, and it is clear that, when $\beta > \mu$, then the disease spreads, while $\beta < \mu$ implies that the disease dies out. Furthermore, since we have a coupled model that considers two interacting disease processes, we have a basic reproduction number for the

fear processes as well, defined as $R_0^F = r_\beta R_0$. That is, the reproductive number for the spread of fear differs from the reproductive number for the disease process by a magnitude of the same factor that regulates the self-induced change [7].

Furthermore, for small values of δ, local, prevalence-based fear is nearly indistinguishable from the global prevalence-based fear. Although local prevalence-based fear is not covered in this paper, it is noteworthy that the spread of fear in both models is, in some way, dependent on interactions with an infected individual, thus showing a connection between the result of two different social processes [7].

Constructing the HIV model

Since HIV can be spread through several different methods, we first note that we are considering transmission of HIV via sexual interaction, rather than needle-sharing behavior of intravenous drug users or mother-to-child transmission. There have been several robust HIV models that have been presented to describe the dynamics of sexual transmission of HIV [2, 3, 4, 6]. For simplicity's sake, we have chosen Blower et al.'s model presented in [3]

$$\begin{aligned} \frac{dS}{dt} &= S_0 - S(t)[\beta\lambda + \mu_S] \\ \frac{dH}{dt} &= S(t)\beta\lambda - H(t)[\nu + \mu_H] \\ \frac{dA}{dt} &= H(t)\nu - A(t)[\mu_A + \delta_A] \end{aligned}$$

where S is the portion of the population susceptible to HIV, H is the portion of the population infected with HIV, and A is the portion of the population that has progressed to AIDS. The model parameters are as follows:

Parameter	**Interpretation**
S_0	Rate at which new susceptibles join the population
β	Average rate of aquiring new partners
λ	Probability that partner is infected with HIV
μ_S	Proportion of the susceptible population that becomes sexually inactive
ν	Proportion of the HIV+ population that progresses to AIDS
μ_H	Proportion of the HIV+ population that becomes sexually inactive
μ_A	Proportion of the AIDS population that becomes sexually inactive
δ_A	Proportion of the AIDS population that dies

We note that this model has several distinct features. First, new infections are characterized by two constant rate terms, β and λ, rather than a dependency on interaction between the susceptible population and a subpopulation of infected individuals, usually shown by $\beta S(t)\frac{I(t)}{N}$. We also note that this model

assumes a total population of $N = 1$, and to guarantee no net flux within the population, we assume that $S_0 = S(t)\mu_S + H(t)\mu_H + A(t)[\mu_A + \delta_A]$.

Analysis of the HIV model

We note that the basic reproductive number, R_0, is given by

$$R_0 = \frac{\beta}{\gamma}$$

where

$$\gamma = \mu_S + \mu_H + \mu_A + \delta_A$$

Biologically, we can interpret R_0 to be a ratio of new infection rates to infected individuals removing themselves from the population via sexual abstinence or death. We note that, if the rate of new infections is greater than the sum of the rates of individuals leaving the population, then $R_0 > 1$ and the endemic equilibrium is reached. However, if individuals leave the population faster than the rate of new infections, then $R_0 < 1$ and the population achieves a disease-free equilibrium.

The disease-free equilibrium (no presence of HIV in the population) is given by $S(t) = H(t) = A(t) = 0$, so the DFE is $(S_0, 0, 0)$, where the entire population is susceptible, and no portion of the population is infected with HIV or has progressed to AIDS. We note that the DFE is unstable.

The endemic equilibrium is given by

$$\begin{aligned} S^* &= \frac{S_0}{\beta\lambda + \mu_S} \\ H^* &= \frac{S_0\beta\lambda}{(\beta\lambda + \mu_S)(\nu + \mu_H)} \\ A^* &= \frac{S_0\beta\lambda\nu}{(\beta\lambda + \mu_S)(\nu + \mu_H)(\mu_A + \delta_A)} \end{aligned}$$

However, we note that the Jacobian of the system is given by

$$\begin{pmatrix} -\beta\lambda - \mu_S & 0 & 0 \\ \beta\lambda & -(\nu + \mu_H) & 0 \\ 0 & \nu & -(\mu_A + \delta_A) \end{pmatrix}$$

Since the Jacobian is an upper triangular matrix, then the eigenvalues are components of the main diagonal, given by

$$\lambda_1 = -\beta\lambda\mu_S, \;\; \lambda_2 = -(\nu + \mu_H), \;\; \lambda_3 = -(\mu_A + \delta_A).$$

We note that the equilibrium is stable, regardless of initial conditions, since all eigenvalues are real and negative.

Behavior-HIV model

Using the same process as described in the behavior-disease model, we consider the contagion term within the context of the HIV model described in the previous section,

$$\beta_F(1 - e^{-\delta(H(t)+A(t))})$$

Thus we construct the following system of nonlinear differential equations

$$\begin{aligned}
\frac{dS}{dt} &= S_0 - S(t)[\beta\lambda + \mu_S] - \beta_F S(t)(1 - e^{-\delta(H(t)+A(t))}) + \mu_F S^F(t)S(t) \\
\frac{dS^F}{dt} &= -r_\beta \beta\lambda S^F(t)H(t) + \beta_F S(t)(1 - e^{-\delta(H(t)+A(t))}) + \mu_F S^F(t)S(t) \\
\frac{dH}{dt} &= -H(t)(\nu + \mu_H) + \beta\lambda S(t) + r_\beta \beta\lambda S^F(t)H(t) \\
\frac{dA}{dt} &= H(t)\nu A(t)(\mu_A + \delta_A)
\end{aligned}$$

where

$$\frac{dN}{dt} = S_0 - \mu_S S(t) - \mu_H H(t) - (\mu_A - \delta_A)A(t) = 0$$

and thus we have no net flux, and the parameters are as above.

The equilibria of this model are slightly more complicated because there are two competing contagion processes. If we assume that $S(t) = S^F(t) = I(t) = H(t) = 0$, then we have a DFE that is the same as the HIV model, where the DFE is given by $(S_0, 0, 0, 0)$. Similarly, if we consider the endemic equilibrium of the model where there is no fear contagion process ($S^F = 0$), then the endemic equilibrium is identical to the HIV endemic equilibrium. In this particular model, we note that that an endemic equilibrium for the fear contagion after the disease process has been eradicated ($H(t) = A(t) = 0$) forces $S^F(t) = S(t) = 0$ and thus the endemic equilibrium of just the fear contagion without the presence of the biological disease is $(S_0, 0, 0, 0)$. Thus this particular model does not exhibit an institutional memory; once the biological disease is not present in the society, the fear dissipates and does not continue to impact behavior. This is not always true; analysis of Perra et al. [7] shows that some types of fear do create an endemic equilibrium of the fear process once the disease has been eradicated, creating the idea of an "institutional memory," or long-term behavior changes that continue to impact a population after the catalyst for those changes has

been removed.

One unusual aspect of this model that is adapted from Perra et al. [7] and differs from many standard models is the presence of the exponential transmission rate of the fear contagion, rather than a mass action incidence rate or a standard incidence rate. The transmission rate is given by

$$\beta_F(1 - e^{-\delta(H(t)+A(t))})$$

for $0 < \delta \leq 1$ and is the primary rate that describes the interaction between the two competing disease processes. We note that the term is dependent on the number of individuals in the population that are infected with either HIV or AIDS. If we consider the behavior of δ, we have that

$$\delta \to 0 \Rightarrow \beta_F(1 - e^{-\delta(H(t)+A(t))}) \to 0$$

and the term becomes obsolete. However, we also note that

$$\delta \to 1 \Rightarrow \beta_F(1 - e^{-\delta(H(t)+A(t))}) \to \beta_F(1 - e^{-[H(t)+A(t)]})$$

We note that as $H(t) + A(t)$ increase, the entire contagion rate increases; that is, as the number of people in the population that are HIV positive increase, fear of the disease increases accordingly. The presence of this exponential term can be understood in terms of how it impacts the behavior of the model; however, the presence and dependency on $I(t)$ make an explicit analytical solution to this system difficult, if not impossible, to achieve. A simultaneous endemic equilibrium of both disease processes, for example, cannot be found explicitly as a result of the exponential transmission rate that is dependent on both $A(t)$ and $H(t)$.

Clearly, this model is substantially more complex than the previous models, and may require numerical approximations in order to understand and interpret the model. However, it serves as one possible application to the coupled behavior-disease model presented by Perra et al. [7] and helps further understand how HIV and social response interact to change the dynamics of HIV transmission.

Discussion

This paper explored the construction of epidemiological modeling, beginning with an understanding of the framework of continuous time, compartmental models. From there, we considered different methods of coupling behavior models with epidemiological models, building off the construction presented by Perra et al. [7]. Finally, we performed some basic analysis of several of the models, and presented a possible

behavioral-HIV model for future consideration and analysis.

One of the most interesting findings of Perra et al. [7] was the possibility of a biologically disease free equilibrium coupled with an endemic fear contagion equilibrium, raising the possibility of population memory, where the behavioral changes become ingrained within the population. In an application to HIV, this could manifest as a change in sexual practices such as a greater emphasis on barrier protection and frequent disease testing, where these practices remain in the population even when the disease has been eradicated. Further construction and analysis of coupled disease-behavior models with respect to specific disease processes can provide insight into the interaction between biological diseases and social responses, which can be invaluable in studying diseases that are coupled with social stigma to better understand the interplay between the two contagion processes.

References

[1] Linda J.S. Allen. *An Introduction to Mathematical Biology*. Pearson, 2007.

[2] CP Bhunu, W Garira, and G Magombedze. Mathematical analysis of a two strain hiv/aids model with antiretroviral treatment. *Acta biotheoretica*, 57(3):361–381, 2009.

[3] Michael Hubbell, Kyle Hunsberger, Kimberly Waite, and Rebecca Wilson. Module 791: Immunological and epidemiological hiv/aids modeling. *The UMAP Journal*, 26(1):49–90, 2005.

[4] James M Hyman and E Ann Stanley. Using mathematical models to understand the aids epidemic. *Mathematical Biosciences*, 90(1):415–473, 1988.

[5] Kathleen M MacQueen. The epidemiology of hiv transmission: trends, structure and dynamics. *Annual review of anthropology*, pages 509–526, 1994.

[6] Alan S Perelson and Patrick W Nelson. Mathematical analysis of hiv-1 dynamics in vivo. *SIAM review*, 41(1):3–44, 1999.

[7] Nicola Perra, Duygu Balcan, Bruno Gonçalves, and Alessandro Vespignani. Towards a characterization of behavior-disease models. *PloS one*, 6(8):e23084, 2011.