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Research Article

Dynamics of an Infectious Disease Where Media Coverage Influences Transmission

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There is significant current interest in the application of media/psychosocial effects to problems in epidemiology. News reporting has the potential to reach and to modify the knowledge, attitudes, and behavior of a large proportion of the community. A susceptible-infected-hospitalized-recovered model with vital dynamics, where media coverage of disease incidence and disease prevalence can influence people to reduce their contact rates is formulated. The media function is incorporated into the model using an exponentially decreasing function. Qualitative analysis of the model reveals that the disease-free equilibrium is locally asymptotically stable when a certain threshold is less than unity. Numerical results show the potential short-term beneficial effect of media coverage.

1. Introduction

Populations worldwide have incurred significant losses from infectious disease outbreaks since the second century, both in terms of morbidity and mortality as well as social and economic costs [1]. As a result, a wide range of tools, including mass media, have been deployed in the effort to control and eliminate epidemic diseases. This study is motivated by the fact that information spreading in a population even though complex, has a great number of applications. Mass media (television, radio, newspapers, billboards, and booklets) have been used as a way of delivering preventive health messages as they have the potential to influence people's behavior [2], and deter them from risky behavior or from taking precautionary measures in relation to a disease outbreak, as concurrent presentation of objective information about the diseases can mitigate its severity [3]. There is a causal relationship between the mass media health education campaign and the increase in the demand for health services during a disease outbreak. However, there is a dearth of research documenting this, at least from the mathematical standpoint. It has been posited that mass media can enable people to demand direct TB smear tests and increase case finding [4]. Grilli et al. [5] noted that those engaged in promoting a better uptake of research information in clinical practice should consider mass media as one of the tools that may encourage the use of effective services and discourage those of unproved effectiveness.

The responsibilities of the media are to disseminate health information [6] and to frequently cover health-related topics and, as such, the media are the leading source of information about important health issues for many individuals. Media coverage of health-related events has become so important that several surveillance systems now rely on active trolling of internet news media to detect emerging disease threats [7, 8]. It is a relatively inexpensive way of exposing the population to information regarding their health [2] and has the potential to reach a large proportion of the population, particularly groups that may be difficult to access through more traditional approaches [5, 9, 9–11]. For instance, mass media campaigns based on communication for behavioral impact and social change have been shown to be an effective intervention for smoking cessation in adults [12–16]. In the case of a vaccine preventable disease, people may opt to vaccinate more, and, promptly, when the perceived risk from the disease is high, and little (and later)

otherwise [17]. Thus, communication and mass media are central to slowing the course of an epidemic [18, 19] and have the potential to modify the knowledge or attitudes of a large proportion of the community simultaneously [20].

Mathematical modeling is an indispensable tool for integrating information and leveraging further insight when data are not available. The role of media communications in altering the outcome of infectious disease outbreaks is gradually having a place in the mathematical epidemiologists repertoire [21–26], and the failure of the current influenza pandemic to spread may be attributable to massive news reporting, especially with regard to prevention, as the amount of media coverage garnered by the 2003 severe acute respiratory syndrome (SARS) and the recent 2009 H1N1 influenza epidemic was colossal [27]. The introduction of some psychological effects into infectious disease modeling can be traced back at least as far as the work of Capasso and Serio [22]. Chen [21] investigated how the quality of information about the prevalence of an infectious disease affects individuals' incentives to adopt self-protective actions to reduce the risk of infection. The choice of the incidence function describing the interplay between disease prevalence and contact rates is a major determinant of the transmission dynamics of the disease.

Various incidence functions such as a type II functional response [24] and a negative exponential function [23, 24] have been proposed to describe the reduction in contacts due to behavioral change when the number of infectious individuals increases [25]. For instance, Cui et al., [26] analyze a susceptible-infected-susceptible *SIS* infection model incorporating media coverage where higher media coverage results in a lower number of infectious persons due to a media-induced reduction in the contact rate. Liu et al. [23] incorporate the psychological impacts of media coverage on disease transmission and emphasize the importance of refining classical mathematical models to reflect the partially self-limiting nature of infectious disease outbreaks, due to the effects of widespread news coverage and fast information flow. Cui et al. [28] propose a model which exhibits multiple positive equilibria when the media effect is sufficiently strong (also see [23]), while Liu and Cui [24] noted that effective media coverage delays the arrival of the infection peak. Li and Cui [29] analyze the effects of constant and pulse vaccination of a susceptible-infected-vaccinated *SIV* epidemic models incorporating media coverage. Joshi et al. [30] model the effect of information campaigns on the HIV epidemic in Uganda and explicitly include a class reflecting the amount of educational information. Mummert and Weiss [31] examined the situation when the media *holds back* news of an outbreak and only after some time begins to report the disease morbidity and mortality with two types of delays. A model with pulse vaccination has shown that the media can trigger a vaccinating panic if the vaccine is imperfect and simplified messages may result in the vaccinated mixing with infected individuals without regard to disease risk [32]. Perlman [33] found that panic in the Israeli media resulted in a very significant reduction in the rate of vaccination with the flu vaccine at the Hadassah Mount Scopus Medical Center during the 2006-2007 flu season. This reduction in the rate of vaccination resulted in greater morbidity. There is a need to consider how to avoid a similar media-evoked panic reaction in the future which causes damage to the public's health as the timing and type of broadcast can make a significant difference on the epidemic spread as change in attitudes and (lack of) knowledge can generally affect the long-term success of any information campaigns in influencing the course of an epidemic. It has been reported that timing and annual receipt of influenza vaccination appear to be influenced by media coverage, particularly by headlines and specific reports on shortage/delay [34]. Sun et al. [27] consider a two-patch *SIS* model whereas we consider a single *SEIHR*.

There are relatively few models that describe the impact of media coverage on the transmission dynamics of infectious diseases [23, 24, 26–29, 32]. Previous studies either use mass action or saturation incidence although data suggest that standard incidence is more realistic than mass action incidence for modeling human diseases [35]. The demographic processes (birth and death) are not taken into consideration in most of these studies. These shortcomings are addressed herein by formulating a model with standard incidence, including vital dynamics and transmission from clinical settings. The analysis adjusted for the number of new hospitalizations, to control for the fact that changed levels of reporting and worry might reflect the changing severity of the outbreak [36].

The rest of this work is organized as follows: Section 2 presents the model framework, some basic properties and stability analysis. Graphical representations are presented in Section 3, while Section 4 concludes the paper.

2. The Model

By assuming random mixing and therefore ignoring any spatial structure within the population, we can describe the model system by considering only the number of individuals in each possible state [37]. The population size (N) can vary over time and is subdivided into four subclasses according to individuals' disease status, namely, susceptible (S), infected/infectious (I), hospitalized (H), and recovered (R). It is assumed that some infectious individuals receive treatment in hospital settings.

The model equations are given by the following deterministic system of nonlinear ordinary differential equations:

$$\frac{dS}{dt} = \Lambda - e^{-M(t)}\lambda S - \mu S, \tag{1a}$$

$$\frac{dI}{dt} = e^{-M(t)}\lambda S - (\alpha_I + \mu + \tau + \delta_I)I, \tag{1b}$$

$$\frac{dH}{dt} = \tau I - (\alpha_H + \mu + \delta_H)H, \tag{1c}$$

$$\frac{dR}{dt} = \delta_H H + \delta_I I - \mu R, \tag{1d}$$

$$M(t) = \max\left\{0, aI(t) + b\frac{dI}{dt}(t)\right\}. \tag{1e}$$

This is an implicitly defined system for which the problem of the existence and uniqueness of solutions on a finite time interval has been studied by Banaszuk [38]. $M(t)$ is the signal function capturing the media coverage over time, Λ is the rate at which susceptible individuals are recruited into the population (no recruitment of infective individuals occurs), Λ is the constant recruitment rate, β is the baseline contact rate, $\lambda = \beta(I + \epsilon H)/N$ is the force of infection, $\epsilon \in [0, 1)$ is the modification parameter that accounts for the reduction in transmissibility of individuals under treatment, μ is the natural death rate, α_I and α_H are the death rates due to the disease and when under treatment, respectively, ($\alpha_I > \alpha_H$), τ is the rate at which individuals enter the health care settings seeking for treatment [23], and δ_H is the recovery rate. It is assumed that $\delta_I \leq \delta_H$ (i.e., the recovery rate of hospitalized individuals is greater than or equal to that of non hospitalized individuals). The choice of the function M (which is exponential to ensure the coefficient of the incidence term remains positive) reflects the fact that media coverage may increase either because the number of cases is large ($aI(t)$), or because there is a significant change in the number of cases ($b\dot{I}(t)$). The positive parameters a and b are difficult to interpret mechanistically. Rather, they are intended to capture the phenomenological effects of either the total number of cases (a) or a change in the number of cases (b) on media sentiment. Because they capture phenomenological effects, it is difficult to parameterize them directly but they could in principle be measured by fitting the model to data on media sentiment and/or cases over time, but we do not attempt that in the current work.

The constant inflow τ can be replaced by an appropriate function if delay in moving into the H class is accounted for (see the appendix A, for the generalization). The proposed model (which is closely related to the work in [22] when $a = 0 = b$ with λ being a monotone decreasing function of I that is strictly concave) can be applied to flu-like illnesses [32].

The above media function differs from the one proposed in [37], which reflects the current level of awareness within the susceptible population. We study the dynamics of the model system (1a), (1b), (1c), (1d), (1e) with permanent immunity in the feasible region

$$\Omega = \left\{(S, I, H) \in \mathbb{R}^3_+ : S + I + H \leq \frac{\Lambda}{\mu}\right\}, \tag{2}$$

which is positively invariant and attracting, and over the allowed domain, the model is well defined [38].

If recovery is nonpermanent, then, (1a) now reads

$$\frac{dS}{dt} = \Lambda - e^{-M(t)}\lambda S - \mu S + \delta_R R, \tag{3}$$

and it can be shown that the set $\Omega_0 = \{(S, I, H) \in \Omega : S \leq S_0\}$ is positively invariant and attracting and, hence, the dynamical properties of the model (such as boundedness, local stability, and persistence of the system) are unchanged [39].

2.1. Disease-Free Equilibrium and Its Stability. The model has a disease-free equilibrium (DFE) given by

$$E_0 = (S_0, I_0, H_0) = \left(\frac{\Lambda}{\mu}, 0, 0\right). \tag{4}$$

For the models (1a), (1b), (1c), (1d), (1e), the next generation matrix calculation [40] shows that the reproduction number (or epidemic threshold) is

$$\mathcal{R}_\tau = \frac{\beta S_0}{\alpha_I + \mu + \tau + \delta_I}\left(1 + \frac{\epsilon\tau}{\alpha_H + \mu + \delta_H}\right), \tag{5}$$

which can be written as $\mathcal{R}_\tau = \mathcal{R}_0(1 + \mathcal{R}_1)$, where $\mathcal{R}_0$ is the basic reproduction number. When a disease breaks out in a human population, changes in behavior in response to the outbreak can alter the progression of the infectious agent [37], resulting in lowering the epidemic size (Figure 4), but does not affect the threshold for disease invasion $\mathcal{R}_\tau$, and, thus, the potential for an outbreak remains unchanged.

Because media coverage is assumed to have no effect in the absence of the disease and that the basic reproduction number is evaluated in the absence of the disease, media coverage does not play a role in $\mathcal{R}_0$. Hence, changing the intensity of media coverage cannot be used to trigger the passage of $\mathcal{R}_\tau$ from values larger to values smaller than unity. Increasing the intensity of media coverage can however greatly reduce the number of infectious individuals at the endemic equilibrium in an endemic situation, that is, when $\mathcal{R}_\tau > 1$. On the other hand, if other means are used to bring $\mathcal{R}_\tau$ to values less than unity, then media coverage can help speed up the extinction of the epidemic [27]. From [40], the following result holds.

Lemma 1. *The DFE E_0 of model system* (1a), (1b), (1c), (1d), (1e) *is locally asymptotically stable provided $\mathcal{R}_\tau < 0$.*

2.2. Existence of the Endemic Equilibrium. At equilibrium, $M(t) = aI(t)$ since $dI/dt = 0$. Solving equations (1a) and (1c) when the right hand sides equal zero yields

$$\begin{aligned} S^* &= \frac{\Lambda}{e^{-aI^*}\lambda^* + \mu}, \\ H^* &= \frac{\tau I^*}{\alpha_H + \mu + \delta_H}, \end{aligned} \tag{6}$$

which upon substitution into the second equation of (1b) gives after some little rearrangements

$$I^*\left(c_1c_2\beta e^{-aI^*}I^* + c_2N\mu - c_1\Lambda\beta e^{-aI^*}\right) = 0, \tag{7}$$

where $c_1 = \alpha_H + \mu + \epsilon\tau + \delta_H$ and $c_2 = (\alpha_H + \mu + \delta_H)(\alpha_I + \mu + \tau + \delta_I)$. $I^* = 0$ is a root of (7) which corresponds to the DFE. An endemic equilibrium exists if the following transcendental equation $c_1c_2\beta e^{-aI^*}I^* + c_2\Lambda - c_1\Lambda\beta e^{-aI^*} = 0$ has a positive root for biological relevance. Using the fact that in the limit superior $N = \Lambda/\mu$ at most, we then rewrite this equation as follows:

$$Y_1(I^*) = I^* - \frac{\Lambda}{c_2}, \qquad Y_2(I^*) = \frac{c_2\Lambda e^{-aI^*}}{c_1\beta}. \tag{8}$$

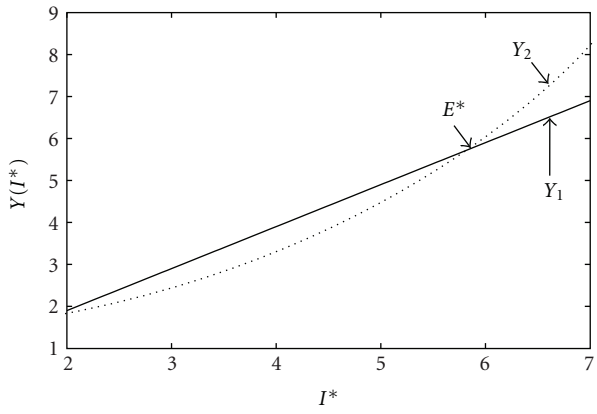

Figure 1: Existence of the the endemic equilibrium E^* when the reproduction number $\mathcal{R}_\tau$ is greater than unity ($\mathcal{R}_\tau > 1$).

Their slopes are

$$y_1 = \frac{dY_1}{dI^*} = 1, \qquad y_2 = \frac{dY_2}{dI^*} = \frac{-a\Lambda}{\beta c_1} e^{-aI^*}. \tag{9}$$

Thus either Y_1 does not intercept Y_2, or Y_1 does intercept Y_2 in two distinct points. Plotting (9) in the y_1y_2-plane yields a single intersecting point which indeed corresponds to the endemic equilibrium E^* (Figure 1). Thus, the model system (1a), (1b), (1c), (1d), (1e) has at least one endemic equilibrium when $\mathcal{R}_\tau > 1$ and none when $\mathcal{R}_\tau < 1$ (for which $I^* = 0$). The outcome (one or two endemic equilibria) strongly depends on the initial conditions.

To prove the local stability of the endemic equilibrium, we first reduce the model system (1a), (1b), (1c), (1d), (1e) into an equivalent system. Let $I+H = P$, since $N_1 = S+I+H$, then, with $d = \min(\alpha_I + \delta_I, \alpha_H + \delta_H)$ and $M(t)$ given by (1e), the model system (1a), (1b), (1c), (1d), (1e) is equivalent to the following system:

$$\begin{aligned} N_1' &= \Lambda - \mu N_1 - dP, \\ P' &= \beta e^{-M(t)} \lambda (N_1 - P) - (\alpha_I + \mu + \tau + \delta_I) P, \\ H' &= \tau P - (\alpha_H + \mu + \delta_H + \tau) H, \end{aligned} \tag{10}$$

from which it can be shown that any nonnegative solution of (10) will eventually enter the convex set

$$\Omega_1 = \left\{ (N_1, P, H) \in \mathbb{R}_+^3 : H \le P \le N_1 \le \frac{\Lambda}{\mu} \right\}. \tag{11}$$

If $\Phi(t) = (N_1(t), P(t), H(t))$ is a non-negative solution of system (10), then

$$\liminf_{t \to \infty} \left(\beta e^{-M} \frac{N_1 - P}{N_1} - (\alpha_I + \mu + \tau + \delta_I) \right) \ge 0. \tag{12}$$

The proof is straightforward and is adapted from [41]. If $\mathcal{R}_\tau > 1$, the DFE is unstable and then, either $H(t) = 0$ (there are no hospitalized people in the community) or $\exists t_0 > 0$ and $\xi > 0$ such that

$$H(t) \ge \xi, \qquad \forall t \ge t_0. \tag{13}$$

If $H(t) = 0$, then $P' = I' = \beta e^{-M} I(1 - (I/N_1)) - (\alpha_I + \mu + \tau + \delta_I)I$, and

$$\begin{aligned} &\liminf_{t \to \infty} \left(\beta e^{-M} \left(1 - \frac{I}{N_1} \right) - (\alpha_I + \mu + \tau + \delta_I) \right) \\ &\quad \approx \beta - (\alpha_I + \mu + \tau + \delta_I) \\ &\quad = (\alpha_I + \mu + \tau + \delta_I) \times (\mathcal{R}_0 - 1) \ge 0. \end{aligned} \tag{14}$$

If $I(t) = 0$, then, $P' = H' = -(\alpha_I + \mu + \tau + \delta_I)$ and $\lim_{t \to \infty} H(t) = 0$.

The epidemicity of a disease is closely related to the stability/persistence of the solutions [39]. For some models, uniform persistence criteria are exactly the conditions used to establish the existence of at least one interior equilibrium [42].

Lemma 2. *If $\mathcal{R}_\tau > 1$, then model system* (1a), (1b), (1c), (1d), (1e) *is locally persistent in $\overset{\circ}{\Omega}$.*

Proof. Persistence in $\overset{\circ}{\Omega}$, the interior of Ω implies there exists a constant $\xi > 0$ (and is independent of initial data in Ω) such that

$$\begin{gathered} \liminf_{t \to \infty} S(t) > \xi, \qquad \liminf_{t \to \infty} I(t) > \xi, \\ \liminf_{t \to \infty} H(t) > \xi. \end{gathered} \tag{15}$$

It can be shown that

$$0 \le \liminf_{t \to \infty} (S(t), I(t), H(t)) \le \limsup_{t \to \infty} \le \frac{\Lambda}{\mu}. \tag{16}$$

Define the following Korobeinikov-Maini-type Lyapunov function [43]:

$$\begin{aligned} L(S, I, H) = \kappa_1 \left(\frac{S^*}{S} + \ln \frac{S}{S^*} \right) + \kappa_2 \left(\frac{I^*}{I} + \ln \frac{I}{I^*} \right) \\ + \kappa_3 \left(\frac{H^*}{H} + \ln \frac{H}{H^*} \right). \end{aligned} \tag{17}$$

Obviously, $L(S, I, H)$ is well defined and continuous for all non negative constant c_1, c_2, c_3, and for all $S, I, H > 0$ with

$$\begin{gathered} \frac{\partial L}{\partial S} = \frac{\kappa_1}{S} \left(1 - \frac{S^*}{S} \right), \qquad \frac{\partial L}{\partial I} = \frac{\kappa_2}{I} \left(1 - \frac{I^*}{I} \right), \\ \frac{\partial L}{\partial H} = \frac{\kappa_3}{H} \left(1 - \frac{H^*}{H} \right). \end{gathered} \tag{18}$$

Equation (18) shows that the endemic equilibrium or positive equilibrium E^* is the only extremum of $L(S, I, H)$ in the positive quadrant and

$$\lim_{(S,I,H) \to 0} L(S, I, H) = \lim_{(S,I,H) \to \infty} L(S, I, H) = +\infty. \tag{19}$$

Thus, from (18) and (19), it is clear that E^* is the global minimum, that is,

$$L(S, I, H) > L(S^*, I^*, H^*) = \kappa_1 + \kappa_2 + \kappa_3 > 0, \quad \forall S, I, H > 0. \tag{20}$$

For simplicity, let $\kappa_1 = \kappa_2 = \kappa_3 = 1$, then, calculating the derivative of $L(S, I, H)$ along the solution of (1a), (1b), (1c), (1d), (1e), we have

$$\begin{aligned}\dot{L} &= \frac{1}{S}\left(1 - \frac{S^*}{S}\right)\dot{S} + \frac{1}{I}\left(1 - \frac{I^*}{I}\right)\dot{I} + \frac{1}{H} \times \left(1 - \frac{H^*}{H}\right)\dot{H} \\ &= -\mu\frac{(S - S^*)^2}{S} - (\alpha_I + \mu + \tau + \delta_I) \times \frac{(I - I^*)^2}{I} \\ &\quad - (\alpha_H + \mu + \delta_H)\frac{(H - H^*)^2}{H} - F(S, I, H),\end{aligned} \tag{21}$$

where $F(S, I, H) = \beta(S - S^*)/S(e^{-M}\lambda S - e^{-M^*}\lambda^* S^*)$. Let $I/N_1 = y$, $I^*/N_1^* = y^*$ and $H/N = z$, $H^*/N_1^* = z^*$, without any ambiguity and loss of reality, assuming that $S^*/S \leq 1$ and substituting the expression for λ and rearranging, we have

$$F(S, I, H) \leq \beta(S - S^*)[\alpha(y - y^*) + \epsilon(z - z^*)], \tag{22}$$

where $\alpha = \min(e^{-(I-I^*)}, 1)$. Applying a modified version of Barbalat's Lemma [44], we have $\alpha(y - y^*) + \epsilon(z - z^*) \to 0$ as $t \to \infty$. Therefore, $\dot{L}(S, I, H) < 0$, for all $S, I, H > 0$ except at E^* where equality holds. Thus, the maximal invariant set in $\{(S, I, H) : \dot{L} = 0\}$ is the singleton $\{E^*\}$ and $L(S, I, H)$ satisfies Lyapunov asymptotic stability Theorem (Lyapunov second method). Hence, from LaSalle-Lyapunov invariance principle, E^* is locally stable and system (1a), (1b), (1c), (1d), (1e) is locally persistent. □

3. Graphical Representations

To complement the mathematical analysis carried out in the previous section, we now investigate some of the numerical properties of system (1a), (1b), (1c), (1d), (1e). The following parameter values are assumed within possible range for illustrative purpose (with unit of day^{-1}). $S_0 = 15$, $I_0 = 6$, $H_0 = 2$, $\Lambda = 0.35$, $\mu = 0.015$, $\beta = 0.73$, $\epsilon = 0.43$, $\delta_I = 0.02$, $\delta_H = 0.04$, $\delta_R = 0.27$, $\tau = 0.65$, $\alpha_I = 0.03$, $\alpha_H = 0.01$. The media parameters a and b control the number of infected cases; it is difficult to estimate their values. Hence, to understand model dynamics for a range of values of a and b, we simulated the model and plotted the equilibrium values of I and H for a range of values of $a, b \in [0.1\ \ 10]$. Small values of a, b (such that the media impact is not strongly dependent on overall prevalence) translate into larger disease prevalence (Figure 2). This is related to over/underreporting which is closely related to the adverse effect of media [32, 45]. The parameter a has a larger effect than b, which is reasonable because a represents the media response to prevalence whereas b represents the media response to incidence. Increasing the parameter a causes the number of infected individuals to decrease. This is not surprising because increasing a causes the population to become more sensitive to the prevalence of infection, which results in a decrease in transmission. However, increasing b can actually cause an increase in the number of infected individuals when a is low, suggesting that a population that is very sensitive to incidence (b) but not very sensitive to prevalence (a) can exhibit surprising responses to media coverage.

Figure 3 depicts the level curves (contour plot) of $\mathcal{R}_\tau$ with respect to some of the parameter values. We observe from Figure 3(a) that $\mathcal{R}_\tau$ increases when the hospitalization rate τ decreases. This occurs because transmission is assumed to be reduced in hospital settings relative to nonhospital settings (due to better infection control) and individuals leave the hospital more quickly due to treatment, relative to untreated individuals who are not hospitalized. Also, $\mathcal{R}_\tau$ increases when the rate δ_H at which people successfully recover and leave the hospital decreases. However, if the rate δ_H is small such that individuals spend a long time in hospital, changing the value of τ has little impact on $\mathcal{R}_\tau$. Figure 3(b) can similarly be interpreted, except that the values of ϵ and δ_H have little impact on $\mathcal{R}_\tau$ at these parameter values. In Figure 3(c) we observe that ϵ again has little impact on $\mathcal{R}_\tau$ whereas δ_I, the rate at which individuals leave the infectious compartment through recovery, has a large impact.

When there are no media reports ($a = 0 = b$), the disease runs its normal course (this was also noted in previous studies [23, 26, 28]) and the number of infected and hospitalized individuals is higher (see Figure 4(a)) than the number in the presence of media coverage (Figure 4(b)). News coverage gets diluted, and the signal triggered by mass reporting at the onset of an epidemic fades away after some time. Also, there is a transient period for small times when the prevalence of infection is slightly higher in the case where media is included. However, media coverage does contribute in controlling the epidemic spread and maintains a low long-term prevalence.

The contour lines (not shown here) of: β versus δ_1 and τ versus δ_I are similar to that of Figure 3(c), while those of β versus τ, δ_I, δ_H are also similar. However, $\mathcal{R}_\tau$ is large when natural recovery δ_I is small, independent of the rate at which hospitalized individuals recover δ_H (this may mean that a high number of infected people in the community continue to transmit the infection, while only a small number are hospitalized).

4. Conclusion

The question of what factors influence people to change their behavior is a difficult one, probably more in the areas of psychology and sociology than epidemiology and public health [46]. Our overall approach was guided by a psychological theory that suggests that higher levels of worry about a hazard, coupled with perceiving a specific action to be effective in protecting against the hazard, increases the likelihood of an individual performing that action [36]. A susceptible-infected-hospitalized-recovered model with vital dynamics, where media coverage of disease incidence and disease prevalence can influence people to reduce their contact rates is formulated. A signal function capturing the media coverage over time is incorporated into the model using an exponentially decreasing function. In Cui et al. [28], three endemic equilibria may occur, but it is important to note that one of the curves will pass through the fourth quadrant, where values are biologically meaningless. Their

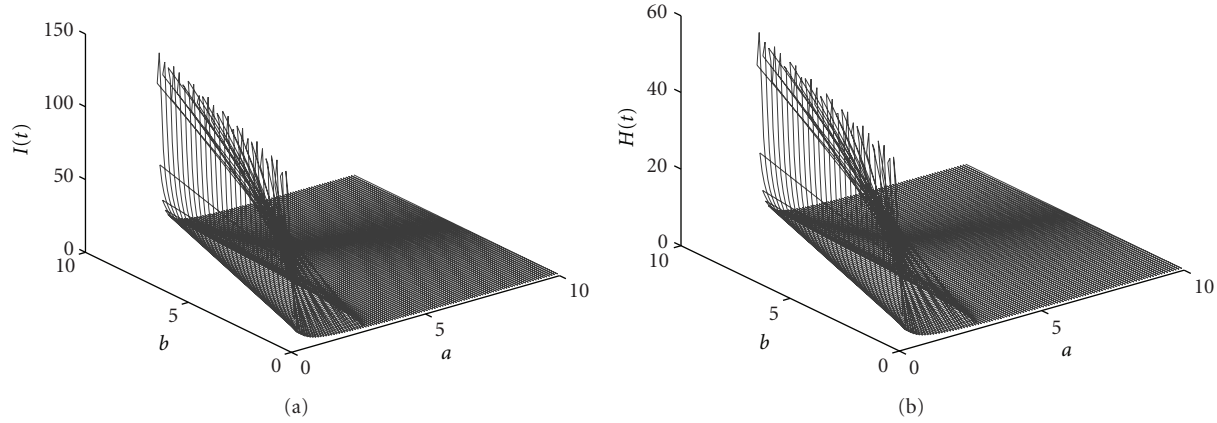

Figure 2: Surface plot of the change in (a) the number of infected individuals, I, and (b) the number of hospitalized individuals, H, for possible combination of the parameters a, b ($\in [0.1\ 10]$) which represent the effect of education/media or psychological impact from media coverage.

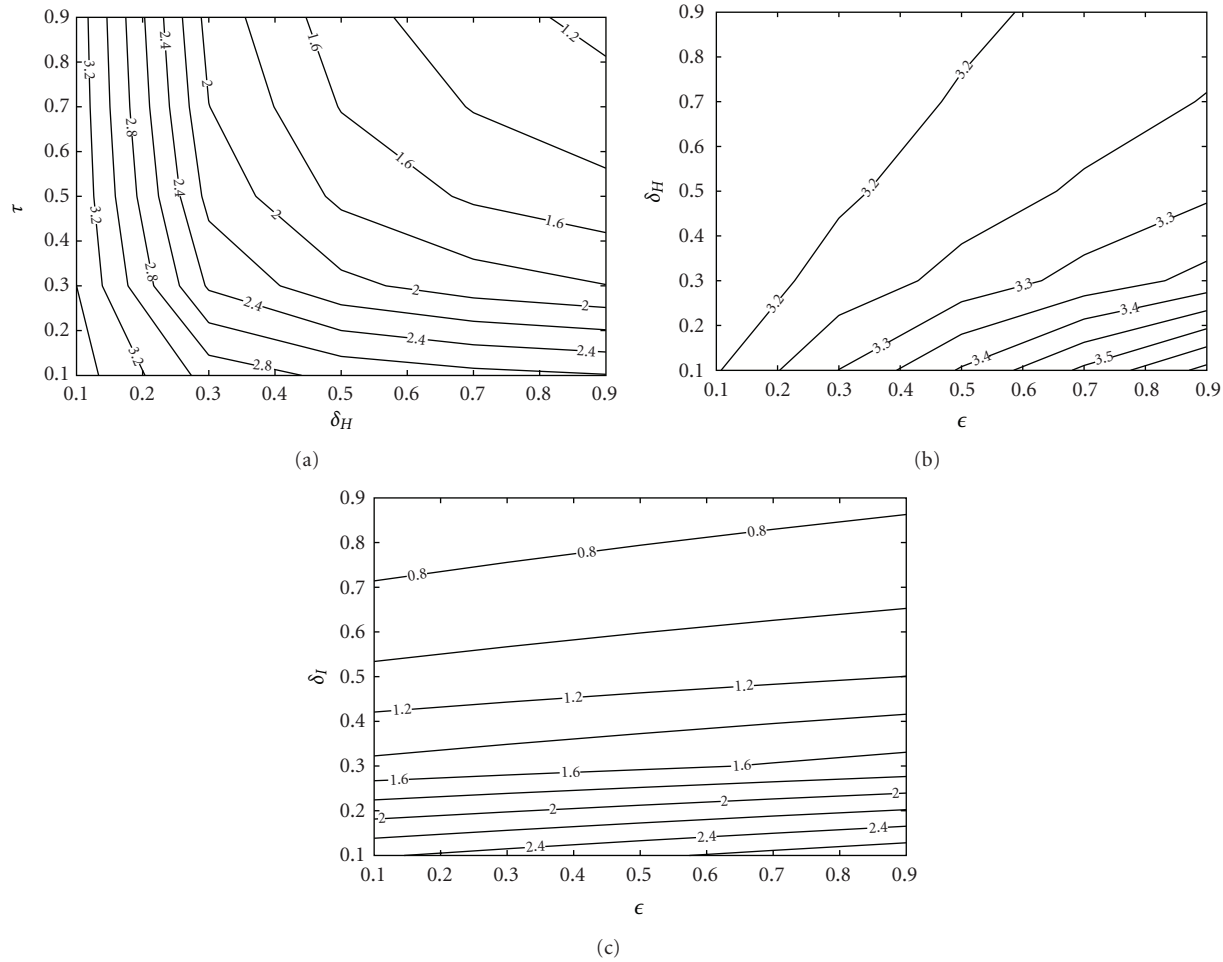

Figure 3: Contour plots of the reproduction number $\mathcal{R}_\tau$ with respect to (a) the recovery rate of hospitalized individuals, δ_H and the rate at which individuals enter the health care settings to seek for treatment, τ (b) the rate at which individuals enter the health care settings to seek for treatment, τ and the recovery rate of nonhospitalized individuals, δ_I (c) the parameter that accounts for the reduction in transmissibility of individuals under treatment, ϵ; and the recovery rate of nonhospitalized individuals, δ_I.

origin is a hyperbolic saddle point (unstable) and multiple endemic equilibria arise, but this is basically driven by their choice of the logistic growth over the constant inflow.

From the graphical representations, it is observed that media coverage (which encompasses designed programs that take efforts to a critical breadth and depth of effort) does not eradicate the disease because the media signal fades when the prevalence and incidence decline to small values, but it contributes in the control process/strategy via information dissemination, which can help to a greater extend to reverse the escalation of some epidemics such as HIV with greater availability of infection prevention mechanisms and resources. Thus, awareness through media and education plays a tremendous role in limiting the spread of infectious disease, and, therefore, the effect of media coverage cannot be underestimated as public health educational campaigns via this medium can help in slowing down the progress of an epidemic outbreak [18, 19]. Also, news reporting at rates dependent upon the number of cases and the rate of change in cases may not destabilize an endemic steady-state, but it can significantly reduce prevalence. For instance, part of Uganda's success has been attributed to a formalized information, education, and communication strategy, lowering estimated HIV/AIDS infection rates from 18.5% in 1995 to 4.1% in 2003 [30]. Vigorous media reporting can have a substantial effect on reducing the impact of an outbreak as public health agencies constant updating of the media about the number of infections as they immediately pass on the information to the general population could result is a dramatic decrease in the severity of the outbreak, see Figure 4. This agrees with the conclusion in [31].

This study is not exhaustive and has some limitations. Construction of a suitable media function is a daunting task as there are many factors that come into play. We proposed a seemingly new function for which the suitable range of its coefficients is determined graphically. One concern is that data on media coverage is hard to gather and in most cases when available are limited. As noted in [30], refining the susceptible class based on behavior change and including the dynamics of the information level are valid features. Finding data and including other features to make the proposed model more realistic are therefore very important if the model is to be calibrated to real data for comparison. Additional research addressing how people's experiences during an outbreak affects their perceptions of health warnings as propagated by the media or the government, what impact this might have on their response to future warnings about a potentially more severe pandemic or how best to ameliorate any scepticism is warranted, informed by evidence-based theories of behavior change [36]. Provision of efficacy information coverage is also viable [47].

The process described in this paper does not in any way take into account the almost endless number of complexities which actually arise. It is an idealization of the real situation in which the complex process is reduced to its essential properties [48]. Therefore, the proposed model can be extended in various ways, by assuming that information decreases at each transmission event while it is gradually lost within each individual and eventually disappears if not refreshed [37, 49], a dynamics that would be most appropriate especially in some rural communities in the developing world where the major means of communication is by word of mouth. Funk et al. [37] observed that disease transmission is dependent on the quality of the information available to a given susceptibility as the tendency to act is reduced with decreasing quality of information, but their model does not include the hospitalized class. An important shortcoming of our model is that the potential benefit of reporting the number of successfully vaccinated individuals (if any) in the previous year in regards to the subsequent level of vaccination the next year (for a seasonal disease such as influenza for instance) is not considered. Human reactions to the presence of a disease abound, yet they have rarely been systematically investigated [50]. Even though the question of what factors influence people to change their behavior is a difficult one [46], accounting for behavioral change in a heterogeneous mixing population is of interest.

Appendix

Hint for Model Generalization

The model can be generalized as follows. First, note that other functional types may also be considered for the transition to the Hclass such as an integral operator of the form $h(I) = \int_0^T K(t-u)\dot{I}(u)du$ to capture the delay effects leading to a neutral functional differential system, however, we will not consider it further. One contribution in the direction of studying this area is [31]. The right choice of the kernel K could approximate I or for $\dot{I}$ which in the simplest case might just be the source-term τI, which leads us back to the current model. Secondly, let the media function be abstractly represented by $g(I)$, a certain bounded function tending to a saturation [22]. The spread of rumors which is analogous to the spread of infectious disease described as *infection of the mind* [51] or *thought contagion* [52] follows this saturation dynamics [32]. Then, the model system (1a), (1b), (1c), (1d), (1e) now reads

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - g(I)S - \mu S,\\
\frac{dI}{dt} &= g(I)S - (\alpha_I + \mu + \delta_I)I - h(I)I,\\
\frac{dH}{dt} &= h(I)I - (\alpha_H + \mu + \delta_H)H,\\
\frac{dR}{dt} &= \delta_H H - (\mu + \delta_R)R.
\end{aligned} \tag{A.1}$$

System (A.1) can be written as

$$\frac{d\mathbf{z}(t)}{dt} = f(\mathbf{z}(t)), \quad t > 0,\ \mathbf{z} \in \Omega, \tag{A.2}$$

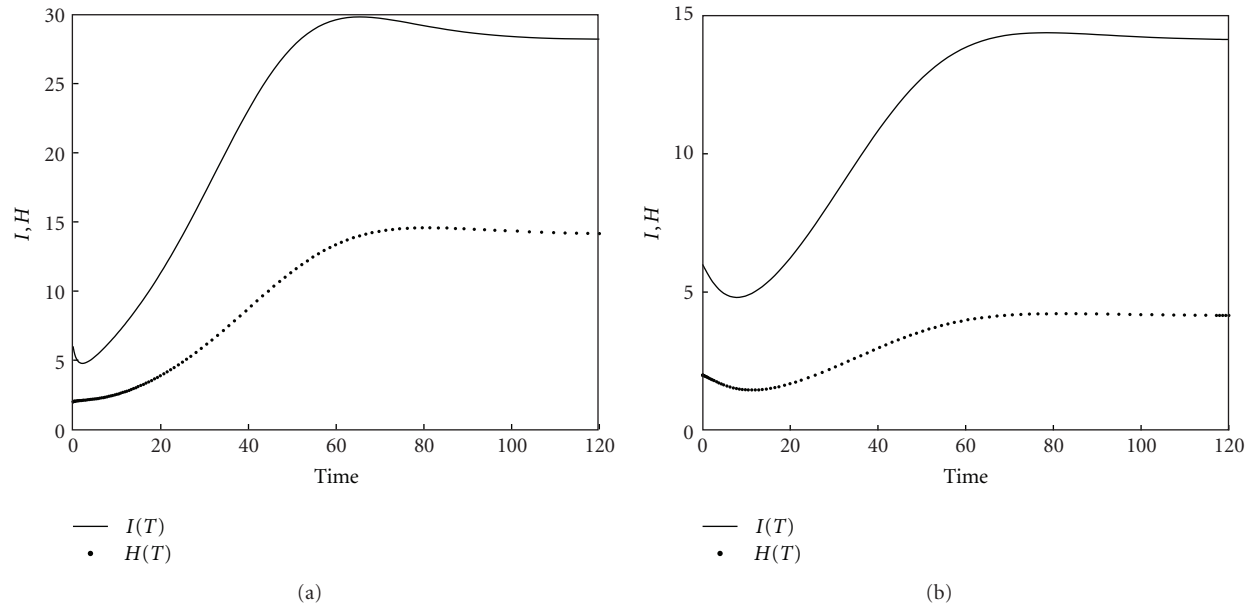

Figure 4: Graphical evolution of the disease (a) without media coverage ($a = 0 = b$), the disease runs its normal course, (b) with media coverage ($a = 2$, $b = 3$), there is a great reduction of the number of infective and hospitalized individuals.

where

$$\begin{aligned} f(\mathbf{z}(t)) &= \left(f_1(z), f_2(z), f_3(z), f_4(z)\right)^T, \\ f_1(z) &= \Lambda - g(z_2)z_1 - \mu z_1, \\ f_2(z) &= g(z_2)z_1 - (\alpha_I + \mu + \delta_I)z_2 - h(I)I, \\ f_3(z) &= h(I)z_2 - (\alpha_H + \mu + \delta_H)z_3, \\ f_4(z) &= \delta_H z_3 - (\mu + \delta_R)z_4, \end{aligned} \tag{A.3}$$

with the superscript denoting transpose.

If $g(z) \leq g'(z)z$, for all $z \in \mathbb{R}_+$, and f is monotone, then

$$\begin{aligned} \forall S, I, H, R \in \Omega : \frac{dS}{dt} &= f_1(S, I, H, R) < 0, \\ \forall S, I, H, R \in \Omega \ni S < \frac{\alpha_1 + h(I)}{g(I)} I : \frac{dI}{dt} &= f_2(S, I, H, R) < 0, \\ \forall S, I, H, R \in \Omega \ni S > \frac{\alpha_1 + h(I)}{g(I)} I : \frac{dI}{dt} &= f_2(S, I, H, R) > 0, \end{aligned} \tag{A.4}$$

where $\alpha_1 = \alpha_I + \mu + \tau + \delta_I$. Thus, the curve $q_1 = S = (\alpha_1 + h(I))/(g(I))I$ will have some kind of threshold character, which was also observed in the simple SI case [22] since the media function appears in the S and I equations only. Similarly, it can be shown that the curve $q_2 = I = (\alpha_2/h(I))I$ (with $\alpha_2 = \alpha_H + \mu + \delta_H$) will also have some kind of threshold character, while the ratio $q_1/q_2 = \alpha_2/(\alpha_1 + h(I)) := \sigma(I)$ is a monotonically decreasing function, and the rest of the analysis will then follow.

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