J. Math. Computer Sci., 31 (2023), 56–69

Online: ISSN 2008-949X

Journal of Mathematics and Computer Science

Journal Homepage: www.isr-publications.com/jmcs

Dynamics of a disease model for three infection phases with media awareness as a control strategy

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Abstract

In this paper, a non-linear mathematical model with three irresistible classes for the impacts of media awareness programs on the spread of irresistible infections, for example, influenza, has been proposed and analyzed. In the modeling process, it is expected that illness spreads because of the contact between the susceptibles and infectives, as it were. The growth rate of media awareness programs influencing the populace corresponds to the number of infective people. We examine the dynamical behavior and systematic investigation of the framework for the model, which demonstrates that the model has two equilibrium points, i.e., disease-free equilibrium (DFE) and interior (endemic) equilibrium. The outcomes show that the primary reproduction number determines the dynamics of the model. For the basic reproduction number $\mathcal{R}_0 < 1$, the disease-free equilibrium is locally as well as globally asymptotically stable under a specific parameter set. If $\mathcal{R}_0 > 1$, the model at the interior equilibrium is locally asymptotically stable. At long last, numerical arrangements of the model validate the analytical outcomes and facilitate a sensitivity analysis of the model parameters.

Keywords: Epidemic model, three phases of treatment, non-pharmaceutical interventions, fundamental reproduction number, global stability, local stability, sensitivity analysis.

2020 MSC: 92D30, 34D23, 93D20.

1. Introduction

Mathematical modeling has become an essential tool in analyzing the spread and control of infectious diseases taking into account the main factors governing the development of a disease, such as transmission and recovery rates [1]. Mathematical models are being used to predict how the infection will spread over a while. Lately, many attempts have been made to develop practical mathematical models for exploring the transmission dynamics of irresistible illnesses. The asymptotic behavior of these epidemic models is studied in [10, 11, 16, 28, 29]. The study of the behavior of epidemic models is beneficial in evaluating strategies to control infectious diseases in the population. The classical models governing the spread of contagious diseases depend mainly on the interactions between susceptibles and infectives. However,

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doi: 10.22436/jmcs.031.01.05

Received: 2022-02-15 *Revised:* 2022-12-09 *Accepted:* 2022-12-15

other factors, such as media coverage, vaccination, migration of population, etc, also affect the spread of infectious diseases [6, 14, 15, 19, 22, 24, 25, 30].

In particular, the media has a significant influence on the individual's behavior towards the diseases and the governmental health care interventions to control the spread of such diseases. The awareness program by the media that makes people know about the condition to take precautions such as social distancing, wearing protective masks, vaccination, etc, to reduce their chances of being infected. Therefore, the modeling process must consider the effect of media to predict the spread of infectious diseases [22]. A few compartmental models have been presented with the assumption that the media will decrease the contact rate of susceptibles with infectives [7, 8, 13, 20]. Liu et al. [21] focused on the psychological impact of epidemic outbreaks. They have considered that increasing infection level reduces effective contact but did not consider mandatory quarantine and isolation factors. In a recent article, Kiss et al. [18] proposed an SIS type compartmental model for sexually transmitted infectious with the assumption that the whole population is aware of the risk. Still, only a certain proportion chooses to respond by limiting their contact with infectives and seeking faster treatment. They have assumed that the total number of susceptibles remains relatively unchanged, and the demographic factors such as natural birth rate, death rate, immigration, etc, have been ignored.

Media coverage of an epidemic gives a sense of the risk level and the relative need for precautions in risk areas. It encourages the public to take preventive measures against the disease, such as wearing masks, avoiding public places, traveling when sick, frequent hand washing, etc. Non-pharmaceutical interventions (NPIs) are essential in the early stages of an epidemic when pharmaceutical interventions are not often possible because treatment or vaccination options have not yet been developed [3, 32]. Many researchers investigated the impact of media awareness using mathematical modeling [7–9, 13, 18, 20, 21, 23, 26, 32–34].

Cui et al. [7] used the transmission coefficient function of the form $\beta(I) = \beta e^{-mI}$ and established that multiple positive equilibria are possible when the media effect is sufficiently strong. In the modeling process of irresistible infections, the incidence function assumes a vital part, and it can decide the ascent and fall of infectious diseases [28]. In many epidemic models, the bilinear incidence rate $\beta\tilde{S}\tilde{I}$ and the standard incidence rate $\beta\tilde{S}\tilde{I}/\tilde{N}$ are frequently used, where β measures the effect of both the infectiousness of the disease and the contact transmission rates. However, these incidence functions do not consider the impact of media coverage on the spread and control of infectious diseases. The use of recommended non-pharmaceutical interventions (NPIs) through media coverage and alert has been found to reduce disease burden in some infectious diseases, e.g., SARS, pH1N1, etc, [17].

Liu et al. [21] and Cui et al. [7] used media-induced transmission rate of the form $\beta(I) = \beta e^{-mI}$ which has two major limitations. We consider media-induced transmission rate as $\beta(I) = \beta e^{-m\frac{I}{N}}$ in the proposed model which is more reasonable than $\beta(I) = \beta e^{-mI}$, because $\beta e^{-mI} \to 0$ as $I \to \infty$, independent of the value of m. Since the media coverage and alertness is not the intrinsic deterministic factor responsible for the transmission; hence it is reasonable to assume that the transmission rate cannot be reduced below a certain level merely through media alert. Moreover, even for a fixed m, the minimum transmission rate differs for different population sizes, regardless of the similarity in the social structure (i.e., education and awareness level) and climatic condition, which is not very realistic. On the other hand, $\min\{\beta e^{-m\frac{I}{N}}\} = \beta e^{-m}$ that remains unchanged with respect to the total population size.

This paper develops a general mathematical model that includes multiple infection stages on the basis of some existing models ([35] and references therein). The primary goal of this article is to study the impact of the use of NPIs stimulated by media coverage of an infectious disease in a community. The rest of the paper is organized as follows. In Section 2, the proposed model is formulated. In Section 3, the existence and local behavior of disease-free and endemic equilibria and global stability of DFE is established. In Section 4, numerical simulation is performed. Sensitivity analysis is performed for reproduction numbers concerning model parameters in Section 5. In the last section, the results are discussed.

2. Model formulation

This section is further divided into two subsections: Assumptions and Model description.

2.1. Assumptions

There are some assumptions we have to make based on our biological background, which are as follows.

1. The population is divided into various mutually exclusive compartments, namely, Susceptible (S), First Infected Individuals (I_1), Second Infected Individuals (I_2), Third Infected Individuals (I_3), and Recovered (R).
2. Population grows at a constant recruitment rate Λ and dies naturally at a rate μ.
3. The population in each compartment does not exhibit any structure (such as spatial location, age, etc.), and no delayed processes are considered.
4. The population is mixing and interacting homogeneously.
5. Disease transmission is horizontal, not vertical. There is no migration of population.
6. Recovered individuals develop disease-acquired temporary immunity.
7. A new media-induced effective transmission rate is proposed as $\tilde{\alpha}e^{-m\frac{\tilde{I}_1}{\tilde{N}}}$, m measures the impact of non-pharmaceutical interventions stimulated by media awareness.
8. In this case, the media-induced transmission rate lies in the range $[\alpha e^{-m}, \alpha]$.

2.2. Model description

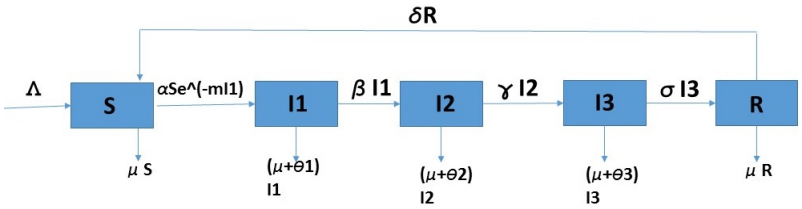

Figure 1: Schematic diagram of proposed disease model with media coverage.

In this section, we propose an $SI_1I_2I_3RS$ epidemic model with media induced transmission rate of the form $\tilde{\alpha}e^{-m\frac{\tilde{I}}{\tilde{N}}}$. The total population at time $\tilde{t}$ is divided into five mutually exclusive compartments, namely, susceptible ($\tilde{S}$), first infected individuals ($\tilde{I_1}$), second infected individuals ($\tilde{I_2}$), third infected individuals ($\tilde{I_3}$) and recovered ($\tilde{R}$). Let $\tilde{N}(\tilde{t}) = \tilde{S}(\tilde{t}) + \tilde{I_1}(\tilde{t}) + \tilde{I_2}(\tilde{t}) + \tilde{I_3}(\tilde{t}) + \tilde{R}(\tilde{t})$, be the total population at time $\tilde{t}$. The schematic diagram of the proposed mathematical model incorporating media coverage for homogeneously mixing population is shown in Figure 1, and the system is governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
&\frac{d\tilde{S}}{d\tilde{t}} = \Lambda - \tilde{\alpha}e^{-m\frac{\tilde{I_1}}{\tilde{N}}}\frac{\tilde{S}\tilde{I_1}}{\tilde{N}} - \tilde{\mu}\tilde{S} + \tilde{\delta}\tilde{R}, \quad \frac{d\tilde{I_1}}{d\tilde{t}} = \tilde{\alpha}e^{-m\frac{\tilde{I_1}}{\tilde{N}}}\frac{\tilde{S}\tilde{I_1}}{\tilde{N}} - (\tilde{\mu} + \tilde{\beta} + \tilde{\theta_1})\tilde{I_1},\\
&\frac{d\tilde{I_2}}{d\tilde{t}} = \tilde{\beta}\tilde{I_1} - (\tilde{\mu} + \tilde{\gamma} + \tilde{\theta_2})\tilde{I_2}, \quad \frac{d\tilde{I_3}}{d\tilde{t}} = \tilde{\gamma}\tilde{I_2} - (\tilde{\mu} + \tilde{\sigma} + \tilde{\theta_3})\tilde{I_3}, \quad \frac{d\tilde{R}}{d\tilde{t}} = \tilde{\sigma}\tilde{I_3} - (\tilde{\mu} + \tilde{\delta})\tilde{R}
\end{aligned} \tag{2.1}$$

with initial conditions:

$$\tilde{S}(0) = \tilde{S}^0 > 0,\ \tilde{I_1}(0) = \tilde{I_1}^0 > 0,\ \tilde{I_2}(0) = \tilde{I_2}^0 > 0,\ \tilde{I_3}(0) = \tilde{I_3}^0 > 0,\ \tilde{R}(0) = \tilde{R}^0 > 0.$$

The descriptions of all parameters are summarized in Table 1.

Table 1: Description of parameters for the system (2.1).

Parameter	Description (Unit)
Λ	Growth rate (days)
$\tilde{\alpha}$	Infection rate (days)
$\tilde{\mu}$	Natural death rate (days)
$\tilde{\beta}$	Transition rate of infected class from first stage to the second stage (days)
m	Coefficient of media coverage (——)
$\tilde{\gamma}$	Transition rate of infected class from second stage to third stage (days)
$\tilde{\sigma}$	Recovery rate from third infection stage (days)
$\tilde{\delta}$	Transfer rate from recovered to susceptible individuals (days)
$\tilde{\theta}_i$	Disease induced death rate of infected class in the 'ith' stage where $i = 1, 2, 3$ (days)

We consider only solutions with initial conditions inside the biologically feasible region

$$\Gamma = \left\{ (\tilde{S},\ \tilde{I_1},\ \tilde{I_2},\ \tilde{I_3},\ \tilde{R}) : 0 \leqslant \tilde{S},\ \tilde{I_1},\ \tilde{I_2},\ \tilde{I_3},\ \tilde{R} \leqslant \frac{\Lambda}{\tilde{\mu}} \right\}$$

in which the usual existence, uniqueness of solutions and continuation results hold.

We study the system (2.1) and claim that the region Γ is bounded and positively invariant with respect to the proposed system (2.1), similar as in [31].

Proposition 2.1. *All the solution trajectories of system* (2.1) *initiating inside* Γ*, approach, enter, or stay within the interior of* Γ*.*

Proof. Let $\mathbb{R}^5_+ = \{(\tilde{S}, \tilde{I_1}, \tilde{I_2}, \tilde{I_3}, \tilde{R}) \in \mathbb{R}^5 : \tilde{S} \geqslant 0, \tilde{I_1} \geqslant 0, \tilde{I_2} \geqslant 0, \tilde{I_3} \geqslant 0, \tilde{R} \geqslant 0\}$ denotes the nonnegative cone in five-dimensional Euclidean space. From the system (2.1), we observe that

$$\frac{d\tilde{S}}{d\tilde{t}}|_{\tilde{S}=0} = \Lambda + \tilde{\delta}\tilde{R} > 0, \qquad \frac{d\tilde{I_1}}{d\tilde{t}}|_{\tilde{I_1}=0} = \tilde{\alpha} e^{-m\frac{\tilde{I_1}}{\tilde{N}}} \frac{\tilde{S}\tilde{I_1}}{\tilde{N}} > 0,$$

$$\frac{d\tilde{I_2}}{d\tilde{t}}|_{\tilde{I_2}=0} = \tilde{\beta}\tilde{I_1} > 0, \qquad \frac{d\tilde{I_3}}{d\tilde{t}}|_{\tilde{I_3}=0} = \tilde{\gamma}\tilde{I_2} > 0, \qquad \frac{d\tilde{R}}{d\tilde{t}}|_{\tilde{R}=0} = \tilde{\sigma}\tilde{I_3} > 0$$

and $\tilde{S}(\tilde{t}), \tilde{I_1}(\tilde{t}), \tilde{I_2}(\tilde{t}), \tilde{I_3}(\tilde{t}), \tilde{R}(\tilde{t})$ are continuous functions of $\tilde{t}$. Thus the vector field on each bounding hyperplane of $\mathbb{R}^5_+$, is pointing inward direction of $\mathbb{R}^5_+$. Hence all the solution trajectories initiating in $\mathbb{R}^5_+$, will remain inside $\mathbb{R}^5_+$ for all the time. This establishes the fact that $\mathbb{R}^5_+$ is positively invariant for the system (2.1). Also, the total population $\tilde{N}(\tilde{t}) = \tilde{S}(\tilde{t}) + \tilde{I_1}(\tilde{t}) + \tilde{I_2}(\tilde{t}) + \tilde{I_3}(\tilde{t}) + \tilde{R}(\tilde{t})$ satisfies $\frac{d\tilde{N}}{d\tilde{t}} = \Lambda - \tilde{\mu}\tilde{N} - \tilde{\theta}_1\tilde{I_1} - \tilde{\theta}_2\tilde{I_2} - \tilde{\theta}_3\tilde{I_3}$. Then, $\frac{d\tilde{N}}{d\tilde{t}} < \Lambda - \tilde{\mu}\tilde{N}$, applying Birkhoff's and Rota's theorem on differential inequality [2], as $\tilde{t} \to \infty$, we have $0 \leqslant \tilde{N}(\tilde{t}) \leqslant \frac{\Lambda}{\tilde{\mu}} = \tilde{N}^0$. Therefore the solution of system (2.1) is bounded and hence any solution of the system originated from Γ remains in Γ. □

Now, we consider not-dimensionally the above system using

$$S = \frac{\tilde{S}}{\tilde{N}},\ I_1 = \frac{\tilde{I_1}}{\tilde{N}},\ I_2 = \frac{\tilde{I_2}}{\tilde{N}},\ I_3 = \frac{\tilde{I_3}}{\tilde{N}},\ \ R = \frac{\tilde{R}}{\tilde{N}},\ \ N = \frac{\tilde{N}}{\tilde{N}^0},\ \ t = \tilde{\mu}\tilde{t}.$$

Since $S = 1 - (I_1 + I_2 + I_3 + R)$, dropping the equation

$$\frac{dS}{dt} = \frac{1}{N} - \alpha e^{-mI_1} S I_1 + \delta R - \frac{S}{N} + \theta_1 S I_1 + \theta_2 S I_2 + \theta_3 S I_3,$$

the equivalent non-dimensional system is given by:

$$\frac{dI_1}{dt} = \alpha e^{-mI_1}(1 - I_1 - I_2 - I_3 - R) I_1 - \beta I_1 - \frac{I_1}{N} + \theta_1 I_1^2 + \theta_2 I_1 I_2 + \theta_3 I_1 I_3 - \theta_1 I_1 := f_1,$$

$$\begin{aligned}
\frac{dI_2}{dt} &= \beta I_1 - \gamma I_2 - \frac{I_2}{N} + \theta_1 I_1 I_2 + \theta_2 I_2^2 + \theta_3 I_2 I_3 - \theta_2 I_2 := f_2,\\
\frac{dI_3}{dt} &= \gamma I_2 - \sigma I_3 - \frac{I_3}{N} + \theta_1 I_1 I_3 + \theta_2 I_2 I_3 + \theta_3 I_3^2 - \theta_3 I_3 := f_3,\\
\frac{dR}{dt} &= \sigma I_3 - \delta R - \frac{R}{N} + \theta_1 I_1 R + \theta_2 I_2 R + \theta_3 I_3 R := f_4,\\
\frac{dN}{dt} &= 1 - (1 + \theta_1 I_1 + \theta_2 I_2 + \theta_3 I_3) N := f_5,
\end{aligned} \tag{2.2}$$

where $\beta = \frac{\tilde{\beta}}{\tilde{\mu}}$, $\delta = \frac{\tilde{\delta}}{\tilde{\mu}}$, $\sigma = \frac{\tilde{\sigma}}{\tilde{\mu}}$, $\gamma = \frac{\tilde{\gamma}}{\tilde{\mu}}$, $\alpha = \frac{\tilde{\alpha}}{\tilde{\mu}}$, $\theta_1 = \frac{\tilde{\theta}_1}{\tilde{\mu}}$, $\theta_2 = \frac{\tilde{\theta}_2}{\tilde{\mu}}$, $\theta_3 = \frac{\tilde{\theta}_3}{\tilde{\mu}}$ and with the initial condition:

$$I_1(0) = I_1^0 > 0, I_2(0) = I_2^0 > 0, I_3(0) = I_3^0 > 0 R(0) = R^0 > 0, N(0) = N^0 > 0. \tag{2.3}$$

3. Model analysis

In the following sections, we will study the dynamical behavior of the system (2.2) with initial condition (2.3). We will calculate the basic reproduction number and all feasible steady states and analyze the equilibria's local stability for the proposed system.

Observe that the biologically feasible region for the non-dimensional system is

$$\Omega = \{(I_1, I_2, I_3\ R,\ N) : 0 \leqslant S,\ I_1,\ I_2,\ I_3,\ R,\ N \leqslant 1\},$$

which is positively invariant for the system (2.2). Hence, we consider only solution with initial conditions inside the region Ω. The system (2.2) always has the disease-free equilibrium (DFE) $(E^0 = 0, 0, 0, 0, 1)$.

3.1. Basic reproduction number

The basic reproduction number, R_0, is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population [12]. The basic reproduction number, sometimes called basic reproductive rate or basic reproductive ratio, is one of the most useful threshold parameters which mathematically characterizes the spreading of infections diseases. This metric is useful because it helps to determine whether or not an infectious disease will spread through the population. We calculate the basic reproduction number similarly as in [12]. Let $x = (I_1,\ I_2,\ I_3)$, then from model (2.2), it follows: $\frac{dx}{dt} = \mathcal{F} - \mathcal{V}$, where

$$\mathcal{F} = \begin{pmatrix} \alpha e^{-mI_1}(1 - I_1 - I_2 - I_3 - R) I_1 \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} \beta I_1 + \frac{I_1}{N} - \theta_1 I_1 \\ -\beta I_1 + \gamma I_2 + \frac{I_2}{N} - \theta_2 I_2 \\ -\gamma I_2 + \sigma I_3 + \frac{I_3}{N} - \theta_2 I_2 \end{pmatrix}.$$

We get

$$F = \text{Jacobian of } \mathcal{F} \text{ at DFE} = \begin{bmatrix} \alpha & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

and

$$V = \text{Jacobian of } \mathcal{V} \text{ at DFE} = \begin{bmatrix} (\beta + \theta_1 + 1) & 0 & 0 \\ -\beta & (\gamma + \theta_2 + 1) & 0 \\ 0 & -\gamma & (\sigma + \theta_3 + 1) \end{bmatrix}.$$

Hence, next generation matrix for the model is

$$K = FV^{-1} = \begin{bmatrix} \frac{\alpha}{(\beta + \theta_1 + 1)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

The spectral radius R_0 of the next generation matrix $K = FV^{-1}$, is the basic reproduction number of the model, i.e., $R_0 = \rho(FV^{-1})$, hence

$$R_0 = \frac{\alpha}{(\beta + \theta_1 + 1)}.$$

3.2. Existence of endemic equilibrium

The system (2.2) also has an interior equilibrium called endemic equilibrium (EE) given by

$$\bar{E} = (\ I_1^*,\ I_2^*,\ I_3^*,\ R^*,\ N^*),$$

where

$$N^* = \frac{1}{1-[\theta_1 + \frac{\beta\theta_2}{(\gamma+\theta_2+1)} + \frac{\beta\gamma\theta_3}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)+(\delta+1)}]I_1^*}, \qquad I_2^* = \frac{\beta}{(\gamma+\theta_2+1)}I_1^*,$$

$$I_3^* = \frac{\beta\gamma}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)}I_1^*, \qquad R^* = \frac{\beta\gamma\sigma}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)(\delta+1)}I_1^*.$$

The value of I_1^* is given by the solution of the equation

$$\frac{e^{mI_1^*}}{R_0} = 1 - \left(1 + \frac{\beta}{(\gamma+\theta_2+1)} + \frac{(\beta)(\gamma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)} + \frac{(\beta)(\gamma)(\sigma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)(\delta+1)})\right) I_1^*. \tag{3.1}$$

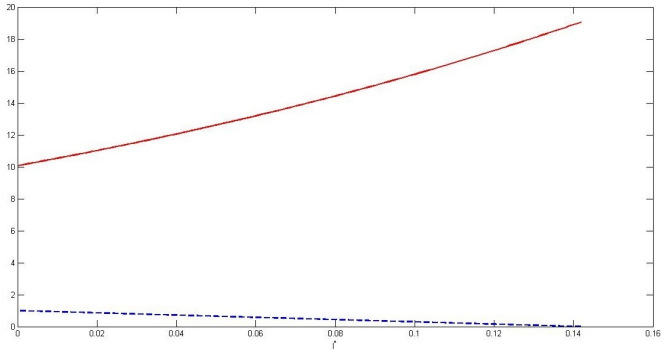

Figure 2: Non existence of EE for $\tilde{\alpha} = 0.09$, $m = 4.5$, and $R_0 = 0.9091 < 1$.

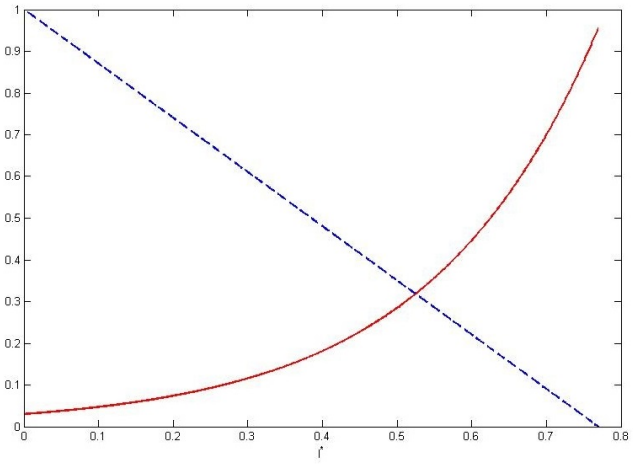

Figure 3: EE exists for $\tilde{\alpha} = 0.12$, $m = 4.5$, and $R_0 = 1.8182 > 1$.

If there is no media effect, i.e., $m = 0$, then

$$I_1^* = \frac{1 - 1/\mathcal{R}_0}{(1 + \frac{\beta}{(\gamma+\theta_2+1)} + \frac{(\beta)(\gamma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)} + \frac{(\beta)(\gamma)(\sigma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)(\delta+1)})}.$$

Clearly, in absence of media effect I_1^* exists if and only if $R_0 > 1$. In presence of media effect, the value of I_1^* is given from equation (3.1). When $R_0 \leqslant 1$, EE does not exist (no point of intersection, see Figure 2) but as $R_0 > 1$ EE exists (see Figure 3). In Figures 2 and 3 the red curve represents $e^{mI_1^*}/R_0$ and blue dotted curve represents

$$1 - \left(1 + \frac{\beta}{(\gamma+\theta_2+1)} + \frac{(\beta)(\gamma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)} + \frac{(\beta)(\gamma)(\sigma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)(\delta+1)})\right) I_1^*$$

at $m = 4.5$. Note that

$$I_1^* \leqslant 1/(1 + \frac{\beta}{(\gamma+\theta_2+1)} + \frac{(\beta)(\gamma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)} + \frac{(\beta)(\gamma)(\sigma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)(\delta+1)})).$$

3.3. Local stability of disease-free and endemic equilibrium

In this section, we explore the local stability of the system (2.2) around the disease-free and endemic equilibria which are stated as follows.

Theorem 3.1. *The disease-free equilibrium (DFE)* E^0 *is*

1. *locally asymptotically stable, if* $R_0 < 1$*;*
2. *unstable, if* $R_0 > 1$.

Proof. The variational matrix at DFE point is given by

$$J_0 = \begin{bmatrix} (\alpha-\beta-\theta_1-1) & 0 & 0 & 0 & 0 \\ \beta & -\gamma-\theta_2-1 & 0 & 0 & 0 \\ 0 & \gamma & -\sigma-\theta_3-1 & 0 & 0 \\ 0 & 0 & \sigma & -\delta-1 & 0 \\ 0 & 0 & 0 & 0 & -1 \end{bmatrix}.$$

The characteristic equation of J_0 is given by

$$(\alpha-\beta-\theta_1-1-\lambda_1)[(-(\gamma+\theta_2+1)-\lambda_2)(-(\sigma+\theta_3+1)-\lambda_3)(-(\delta+1)-\lambda_4)(-1-\lambda_5)] = 0,$$

i.e.,

$$\lambda_5 = -1,\ \ \lambda_4 = -(\delta+1),\ \ \lambda_3 = -(\sigma+\theta_3+1),\ \ \lambda_2 = -(\gamma+\theta_2+1),\ \ \lambda_1 = (\alpha-\beta-\theta_1-1).$$

Clearly if $\mathcal{R}_0 < 1$, then all the five eigen values of $\mathbf{J_0}$ have negative real parts. If $\mathcal{R}_0 > 1$, then four eigen values of $\mathbf{J_0}$ have negative real parts and one eigen value has positive real part. Hence, DFE is locally asymptotically stable, if $\mathcal{R}_0 < 1$ and unstable, if $\mathcal{R}_0 > 1$. □

Theorem 3.2. *The endemic equilibrium* $\bar{E}$ *is locally asymptotically stable for* $\mathcal{R}_0 > 1$*, but close to* 1.

Proof. Here, we use the method based on the central manifold theory to establish the local stability of endemic equilibrium taking α as bifurcation parameter [5]. Critical value of bifurcation parameter α at $\mathcal{R}_0 = 1$ is $\alpha^* = \beta + \theta_1 + 1$. It can be easily verified that the Jacobian $\mathbf{J_0}$ at $\alpha = \alpha^*$ has a right eigenvector (corresponding to the zero eigenvalue) given by $\mathbf{W} = (w_1, w_2, w_3, w_4, w_5)^T$, where

$$w_1 = (\gamma+\theta_2+1),\ w_2 = \beta,\ w_3 = \frac{(\sigma+\theta_3+1)}{\gamma},\ w_4 = 0,\ w_5 = 0.$$

Furthermore, the components of the left eigenvector (corresponding to the zero eigenvalue), $\mathbf{V} = (v_1, v_2, v_3, v_4, v_5)$, must satisfy the equalities $\mathbf{V}.\mathbf{J_0} = 0$ and $\mathbf{V}.\mathbf{W} = 1$, so that we obtain

$$v_1 = \frac{1}{(2+\gamma+\sigma+\theta_2+\theta_3)},\ \ v_2 = 0,\ \ v_3 = \frac{\gamma}{(2+\gamma+\sigma+\theta_2+\theta_3)},\ \ v_4 = 0,\ \ v_5 = 0.$$

The associated non-zero partial derivatives of $\mathbf{F} = (f_1, f_2, f_3, f_4, f_5)^T$ at the DFE and $\alpha = \alpha^*$ are given by

$$\frac{\partial^2 f_1}{\partial I_1 \partial I_2} = \frac{\partial^2 f_1}{\partial I_2 \partial I_1} = -(\beta + 1 - \theta_2), \ \frac{\partial^2 f_1}{\partial I_1 \partial N} = \frac{\partial^2 f_1}{\partial N \partial I_1} = 1,$$
$$\frac{\partial^2 f_1}{\partial I_1^2} = -2(1+m)(1+\beta) + \theta_1, \ \frac{\partial^2 f_1}{\partial R \partial J_1} = \frac{\partial^2 f_1}{\partial J_1 \partial R} = -(1+\beta), \ \frac{\partial^2 f_1}{\partial I_1 \partial I_3} = \frac{\partial^2 f_1}{\partial I_3 \partial I_1} = -(\beta + 1 - \theta_3),$$
$$\frac{\partial^2 f_2}{\partial I_1 \partial I_2} = \frac{\partial^2 f_2}{\partial I_2 \partial I_1} = \theta_1, \ \frac{\partial^2 f_2}{\partial I_1 \partial I_3} = \frac{\partial^2 f_2}{\partial I_3 \partial I_1} = \theta_3, \ \frac{\partial^2 f_2}{\partial I_2^2} = 2\theta_2, \ \frac{\partial^2 f_2}{\partial I_2 \partial N} = \frac{\partial^2 f_2}{\partial N \partial I_2} = 1,$$
$$\frac{\partial^2 f_3}{\partial I_1 \partial I_3} = \frac{\partial^2 f_3}{\partial I_3 \partial I_1} = \theta_1, \ \frac{\partial^2 f_3}{\partial I_2 \partial I_3} = \frac{\partial^2 f_3}{\partial I_3 \partial I_2} = \theta_2, \ \frac{\partial^2 f_3}{\partial I_3^2} = 2\theta_3, \ \frac{\partial^2 f_3}{\partial I_3 \partial N} = \frac{\partial^2 f_3}{\partial N \partial I_3} = 1,$$
$$\frac{\partial^2 f_4}{\partial R \partial I_1} = \frac{\partial^2 f_4}{\partial I_1 \partial R} = \theta_1, \ \frac{\partial^2 f_4}{\partial I_2 \partial R} = \theta_2, \ \frac{\partial^2 f_4}{\partial I_3 \partial R} = \theta_3, \ \frac{\partial^2 f_4}{\partial R \partial N} = \frac{\partial^2 f_4}{\partial N \partial R} = 1,$$
$$\frac{\partial^2 f_5}{\partial I_1 \partial N} = \frac{\partial^2 f_5}{\partial N \partial I_1} = -\theta_1, \ \frac{\partial^2 f_5}{\partial I_2 \partial N} = \frac{\partial^2 f_5}{\partial N \partial I_2} = -\theta_2, \ \frac{\partial^2 f_5}{\partial I_3 \partial N} = \frac{\partial^2 f_5}{\partial N \partial I_3} = -\theta_3.$$

Here we use notations $x_1 \equiv I_1, x_2 \equiv I_2, x_3 \equiv I_3, x_4 \equiv R, x_5 \equiv N$. Hence, we get

$$\begin{aligned}
\mathbf{a} &= \sum_{k,i,j=1}^{4} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \\
&= -\frac{1}{(2+\gamma+\sigma+\theta_2+\theta_3)\gamma}\big[\big(\gamma(\gamma+\theta_2+1)^2[2(1+m)(1+\beta)-\theta_1]\big)(+2\beta\gamma(\gamma+\theta_2+1)(\beta-\theta_3+1) \\
&\quad + (\gamma+\theta_2+1)(\beta-\theta_3+1)(\sigma+\theta_3+1))(-\gamma\theta_1(\gamma+\theta_2+1)(\sigma+\theta_3+1) \\
&\quad - \beta\gamma\theta_2(\sigma+\theta_3+1) - \theta_3(\sigma+\theta_3+1))\big], \\
\mathbf{b} &= \sum_{k,i=1}^{4} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0) = v_1 w_2 = \frac{(\gamma+\theta_2+1)}{(2+\gamma+\sigma+\theta_2+\theta_3)}.
\end{aligned} \tag{3.2}$$

Since, either $a < 0$ or $a > 0$ and $b > 0$ at $\alpha = \alpha^*$, there are two cases arise.

(i.) $\mathbf{a} > 0, \mathbf{b} > 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable and their exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable, and their exists a negative and locally asymptotically stable equilibrium.

(ii.) $\mathbf{a} < 0, \mathbf{b} > 0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Therefore using the Theorem and Remark stated above, a transcritical bifurcation occurs at $R_0 = 1$ and the unique endemic equilibrium is locally asymptotically stable for $R_0 > 1$. □

3.4. Global Stability of disease-free equilibrium

In this section, we analyze the global stability of the disease-free steady states for a special case. We state the following theorem.

Theorem 3.3. *Suppose* $R_0 < 1$ *and* $\theta_1 = \theta_2 = \theta_3 = 0$. *The disease-free equilibrium* E^0 *is globally asymptotically stable.*

Proof. Here, we prove global stability of DFE applying the method used by Castillo-Chavez et al. [4, 29]. According to Castillo-Chavez et al. the following conditions (H1) and (H2) must be met to guarantee a local asymptotic stability:

1. H1 For $\frac{dX}{dt} = F(X, 0), X^0$ is globally asymptotically stable;
2. H2 $G(X, Z) = BZ - \widehat{G}(X, Z)$, where $\widehat{G}(X, Z) \geqslant 0, \forall (X, Z) \in \Omega$.

Let disease-induced death rate $\theta_1 = \theta_2 = \theta_3 = 0$, then $\frac{dN}{dt} = 1 - N$. In limiting case $N(t) \to 1$, as $t \to \infty$. Taking the total population in limiting case, i.e, $N = 1$, then the system (2.2) reduces to

$$\frac{dI_1}{dt} = \alpha e^{-mI_1}(1 - I_1 - I_2 - I_3 - R)I_1 - (\beta + 1)I_1, \qquad \frac{dI_2}{dt} = \beta I_1 - (\gamma + 1)I_2,$$
$$\frac{dI_3}{dt} = \gamma I_2 - (\sigma + 1)I_3, \qquad \frac{dR}{dt} = \sigma I_3 - (\delta + 1)R,$$

and the modified basic reproduction number is $R_0 = \frac{\alpha}{(\beta+1)}$. Let $X = (R)$ and $Z = (I_1, I_2, I_3)$, and

$$Q_0 = (R^0,\ 0), \quad \text{where} \quad R^0 = 0.$$

We have

$$\frac{dX}{dt} = F(X,\ Z) = \sigma I_3 - (\delta + 1)X.$$

At $R = R^0$, $G(X,\ 0) = 0$. Now $\frac{dX}{dt} = F(X,\ 0) = -(\delta + 1)X$, as $t \to \infty$, $X \to X^0$. Hence, $X = X^0 (= R^0 = 0)$ is GAS. Thus, condition (H1) is satisfied. From the reduces system, we get

$$G(X,\ Z) = \begin{bmatrix} (\alpha - \beta - 1) & 0 & 0 \\ \beta & -(1+\gamma) & 0 \\ 0 & \gamma & -(1+\sigma) \end{bmatrix} \begin{bmatrix} I_1 \\ I_2 \\ I_3 \end{bmatrix} - \begin{bmatrix} \alpha(1 - e^{-mI_1})I_1 + \alpha e^{-mI_1}(1 + I_1 + I_2 + I_3 + R)I_1 \\ 0 \\ 0 \end{bmatrix} = BZ - \widehat{G}(X,\ Z),$$

where,

$$B = \begin{bmatrix} (\alpha - \beta - 1) & 0 & 0 \\ \beta & -(1+\gamma) & 0 \\ 0 & -\gamma & -(1+\sigma) \end{bmatrix},$$

and

$$\widehat{G}(X,\ Z) = \begin{bmatrix} \alpha(1 - e^{-mI_1})I_1 + \alpha e^{-mI_1}(1 + I_1 + I_2 + I_3 + R)I_1 \\ 0 \\ 0 \end{bmatrix}.$$

If $R_0 < 1$, then the matrix B is a M-Matrix and $\widehat{G}(X,\ Z) \geqslant 0$. Thus, both conditions (H1) and (H2) are satisfied. Therefore the DFE is gas if $R_0 < 1$. □

4. Numerical simulations

In this section, we provide numerical simulations to illustrate previously established results with the biological feasible parametric values as shown in Table 2 taking time unit in days. Most of the values of parameters are taken in the reference from the existing literature ([27, 29] and references therein) and the rest of the parametric values are assumed for the numerical computation. The system (2.2) is simulated taking initial population size $\tilde{S_0} = 40, \tilde{I_1^0} = 35, \tilde{I_2^0} = 30, \tilde{I_3^0} = 25, \tilde{R_0} = 20$, and hence $\tilde{N} = 100$. The corresponding initial condition in non-dimensional form is $S_0 = 0.4, {I_1}^0 = 0.35, {I_2}^0 = 0.32, {I_3}^0 = 0.23, R_0 = 0.2$, and $N_0 = 0.02$. Numerical simulations are performed using ODE solver of Matlab to justify the analytical findings of previous sections. Now we consider the following two cases.

Case (a): When $m = 4.5$ and $\tilde{\alpha} = 0.09$ (i.e., $\alpha = 90.0$), then $\mathcal{R}_0 = 0.9091 < 1$ and the DFE is globally asymptotically stable shown in Figure 5, which is in accordance with the results stated in Theorems 3.1 and 3.3.

Table 2: Parameter values used in the simulation for the system (2.2).

Parameter	Value
Λ	5.0
$\tilde{\delta}$	0.07
$\tilde{\mu}$	0.01
$\tilde{\gamma}$	0.1
$\tilde{\theta}_1$	0.01
$\tilde{\sigma}$	0.03
$\tilde{\beta}$	0.2
m	4.5
$\tilde{\theta}_2$	0.04
$\tilde{\theta}_3$	0.03
$\tilde{\alpha}$	[0.09,0.12]

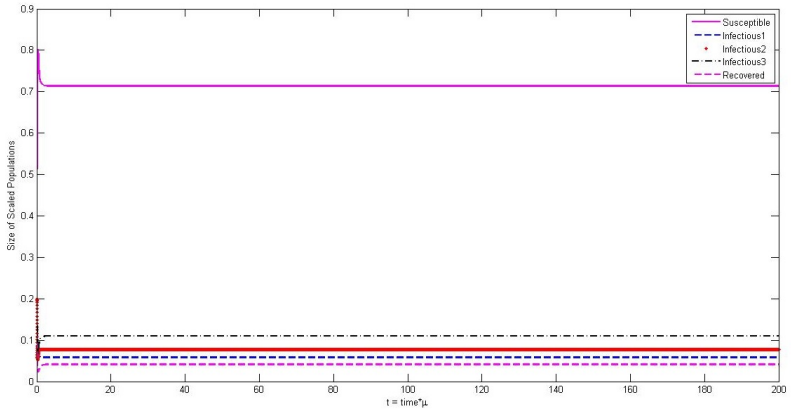

Figure 4: The variation of population in scaled time taking $\tilde{\alpha} = 0.12$, $m = 4.5$, and $\mathcal{R}_0 = 1.8182 > 1$.

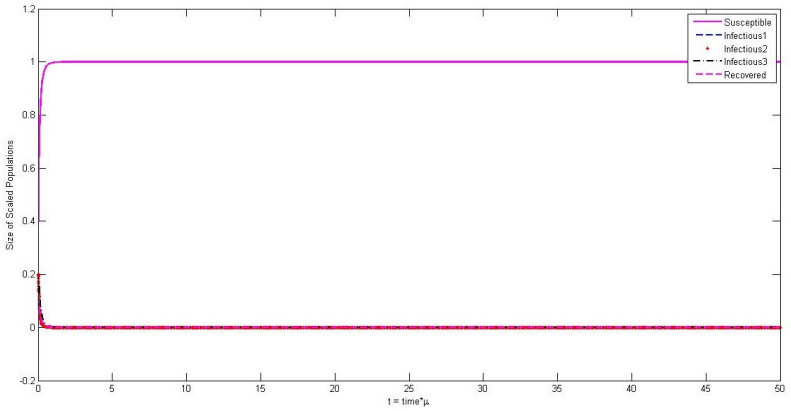

Figure 5: The variation of population in scaled time taking $\tilde{\alpha} = 0.09$, $m = 4.5$, and $\mathcal{R}_0 = 0.9091 < 1$.

Case (b): When $m = 4.5$ and $\tilde{\alpha} = 0.12$ (i.e., $\alpha = 120$), then $\mathcal{R}_0 = 1.8182 > 1$ and it follows that EE is locally asymptotically stable from Theorem 3.2 as shown in Figure 4.

The coefficient of media coverage m should depend on the disease under consideration, the population's social structure (education, awareness, responsiveness, economy, etc), and the NPIs used in a particular region. Here, we use the formula $m = -\log e(p + q - pq)$ to quantify the coefficient m of media coverage, where q quantifies the response of the population aware to media recommended NPIs

concerning the number of infective individuals. If people are not responding to media alerts, then $q = 1$, and if all the people are adopting the recommended NPIs, then $q = 0$. It is assumed that the disease transmission rate can be reduced by p fraction when all individuals follow the media urged NPIs to protect themselves. It is observed from the analysis that the coefficient of media coverage m does not affect R_0 and the qualitative features of the model remain unaltered. From 3.2, we observe that a is always negative, which precludes the existence of backward bifurcation in the system and hence ensures transcritical (i.e., forward) bifurcation about $R_0 = 1$. Hence, the classical requirement of $R_0 < 1$ is necessary and sufficient for disease control in this case. Moreover, from (3.1), we observe that

$$\frac{\partial I_1}{\partial m} = \frac{-e^{mI_1^*}I_1^*}{1-\left(1+\frac{\beta}{(\gamma+\theta_2+1)}+\frac{(\beta)(\gamma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)}+\frac{(\beta)(\gamma)(\sigma)}{(\gamma+\theta_2+1)(\sigma+\theta_3+1)(\delta+1)}\right)R_0+me^{mI_1^*}} < 0.$$

One can easily observe that using NPIs stimulated by media coverage helps mitigate the disease burden from the environment by lowering the level of infectious individuals to a steady-state. The effect of m on the fraction of infectious individuals (I_1) is shown in Figures 6 and 7 taking same parametric values as in the cases (a) and (b), respectively with different values of m. It is observed that the level of endemic equilibrium is significantly affected by media coefficient of m.

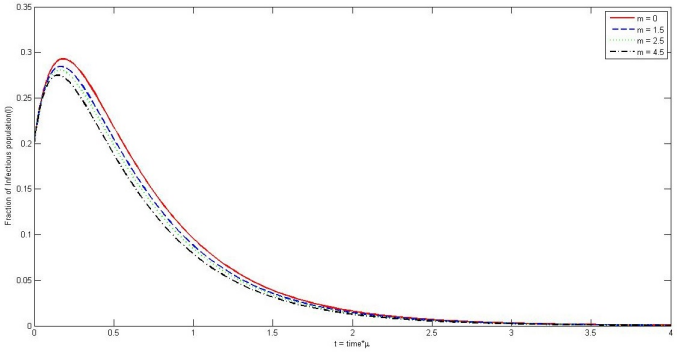

Figure 6: Effect of m on I_1 at $\tilde{\alpha} = 0.09$ with $\mathcal{R}_0 = 0.9091 < 1$.

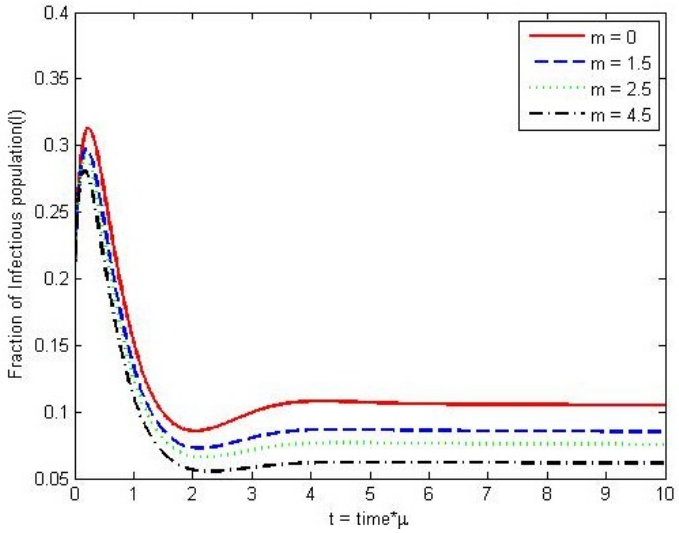

Figure 7: Effect of m on I_1 at $\tilde{\alpha} = 0.12$ with $\mathcal{R}_0 = 1.8182 > 1$.

5. Sensitivity analysis

In this section, we perform the sensitivity analysis of effective reproduction number $\mathcal{R}_C$ taking parametric values given in Table 2. The normalized sensitive indices of effective reproduction number $\mathcal{R}_C$ with respect to parameters are shown in Table 3.

Table 3: The sensitivity indices, $\Upsilon_{y_j}^{\mathcal{R}_C} = \frac{\partial \mathcal{R}_C}{\partial y_j} \times \frac{y_j}{\mathcal{R}_C}$, of the effective reproduction number $\mathcal{R}_C$ to the parameters y_j for parameter values given in Table 2.

Parameter (y_j)	Sensitivity index of $\mathcal{R}_C$ w.r.t. y_j ($\Upsilon_{y_j}^{\mathcal{R}_C}$)
β	-0.9708
α	1.000
σ	0
γ	0
m	0
θ_1	-0.01941
θ_2	0
θ_3	0

From Table 3, we observe that α has a positive impact on $\mathcal{R}_C$, and the rest of the parameters have negative impact. For example, 10% increase (decrease) in β, resulting in 0.9708% increase (decrease) in $\mathcal{R}_C$. Moreover, parameters α and β are most sensitive to $\mathcal{R}_C$, hence we observe significant change in $\mathcal{R}_C$ by small changes in these parameters.

6. Results and discussion

In this paper, the mathematical model for spreading contagious disease is formulated to assess the impact of non-pharmaceutical interventions stimulated by media coverage. For mathematical convenience, it is assumed that the population is in a homogeneous environment. Further, the people in each compartment do not exhibit any structure (such as space, location, age, etc) with instantaneous shifting from one compartment to another; the system of ordinary differential equations describes the time evolution of such compartments. The acquired immunity is supposed to be temporary so that individuals who recovered from infection can become susceptible again over time. This proposed model deals with a nonlinear mathematical model reflecting the effect of awareness programs on a specific population with constant requirements. We have studied the impact of awareness as a novel intervention for controlling epidemiological disease. In this modeling process, it is assumed that media campaign creates awareness regarding personal protection, for example, control of HIV/AIDS. Our analytical study shows that the basic reproduction number that determines a disease's existence does not contain awareness-related terms. We have also shown that this model undergoes trans-critical bifurcation at $R_0 = 1$, and there exists endemic equilibrium when R_0 exceeds one. Hence, it does not change the qualitative behavior of the model, but it helps mitigate the disease burden by lowering the level of infection over time. With media coverage, transmission dynamics of infectious diseases have already been carried out in previous studies, but these models do not account for multi-stage infection class. The main mathematical finding of this research paper is that adding a multi-stage infection class with the use of NPIs stimulated by media does not alter the basic model's essential qualitative features (about the disease's persistence or elimination).

Acknowledgment

The authors are thankful to the referees for their valuable suggestions. They are also grateful to the anonymous reviewers for their helpful tips and comments that have improved the quality and presentation of the manuscript.

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