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Bifurcation for a disease model with the effect of mass media

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Abstract

In this paper, a four dimensional epidemic model was developed to quantify the effects of awareness created by the media strategy on the transmission and management of contagious diseases. The model was built on the assumption that infections occurred only through effective contact between the infectives and susceptibles. It was also assumed that the expansion of media coverage influencing the population was a function of the number of infectives. The model was studied qualitatively by employing stability theory and also quantitatively by employing a mathematical software. Both the qualitative and quantitative analyses revealed that the transmission of infectious diseases could be managed by employing awareness strategies but the disease might still persist in the population despite the implementation of awareness programmes, a phenomenon known as backward bifurcation.

Keywords: Mathematical model, Media, Infectious diseases, Effective contact, Backward bifurcation.

2020 MSC: 92B05, 34D05, 92D30.

1. Introduction

Worldwide, infectious diseases account for about 11 million deaths every year, the majority of which occur in the third world [4]. Tuberculosis, typhoid, pneumonia, cholera, measles, HIV/AIDS, malaria, Ebola, Lassa fever, rabies, hepatitis, mumps, zika, West Nile, chikungunya and recently COVID-19, etc. are the main fatal infectious diseases [5]. The transmission potential of infectious diseases is usually quantified in terms of interactions between the infected and susceptible individuals in classical models [6, 25, 27, 28, 29, 47]. However, disease spread and transmission can also be influenced by many other circumstances such as migration, income, climate change, sanitation, vaccination, media coverage, etc. [26]. The media in particular has a major impact during a disease outbreak not only on the people's behaviour but also on the government plan to contain the transmission of the disease [23].

The awareness campaigns through the media may inform individuals the precautions to take such as wearing of masks, vaccination, physical distancing, hand washing, etc. to minimise their tendencies of being infected [37]. Therefore, to forecast the dynamics of a disease, there is a need to consider the impact

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of media coverage. Even though, awareness about a disease may create panic if the number of reported cases is enormous, information about the disease may be beneficial because it could reduce transmission potential as the informed susceptibles would avoid contact with the infectious agents [32].

Generally, some diseases confer permanent immunity upon recovery, e.g. measles, influenza, chickenpox, while other diseases confer temporary immunity upon recovery, for example, toxoplasmosis, cholera, tuberculosis, Ebola, meningitis, gonorrhoea, etc. [8]. In another case, reliable vaccines have not yet developed for some diseases, e.g. dengue, AIDS, malaria, chikungunya, etc. [7]. For the diseases that have no proven vaccines, the best intervention is to stop individuals from being infected which can be achieved by appropriate awareness campaigns via the media. Due to the pivotal roles which the media play in shaping the course of epidemic, researchers have developed several mathematical models, which address the roles of media in disease dynamics, either by deterministic or stochastic mathematical models [2, 3, 21, 38, 48, 39, 41].

In deterministic models, media is mainly indicated in two ways: incorporating a number of new population classes, and modifying incidence function to adjust the ”psychological” consequences [32]. Based on the classical compartmental model ideals, several models have been developed by incorporating the hospitalised and the awareness populations to analyse the effect of the awareness intervention by the media on the course of the epidemic [18, 45, 46]. In studies concerning the modification of various incidence functions to indicate the psychological consequences of disease outbreaks, the key impression is to show that effective contacts between infectives and susceptibles diminish at elevated infective levels because of the isolation of the infectives or the protective strategies taken by the susceptibles after the awareness is created by the media.

Incidence functions have been generally expressed as decreasing functions by many authours to describe the impact of media reportage on the course of epidemic [3, 22, 23, 33]. The major dynamical behaviour explained in these studies was expressed in terms of a threshold quantity for incidence and prevalence of the disease under media coverage. While the awareness campaigns by the media can slow down the course of an epidemic or possibly eradicate it, the tendency of information fading which is one of the attributes of man can instigate disease persistence or reemergence, a phenomenon known as backward bifurcation in disease modelling.Several models have been presented in the literature; for example, see [9, 10, 11, 12, 13, 16, 17, 42, 43] and the references therein.

Motivated by the aforementioned studies, the present work incorporates a hospitalised compartment into the classical SIR model in [34] and a nonlinear SIHR compartmental model (susceptible, infectious, hospitalised, recovered) is developed to investigate the possibility of the existence of backward bifurcation for the spread of infectious diseases under media coverage. With the exception of few studies in the literature [24, 37, 40], backward bifurcation phenomenon in the epidemic models incorporating awareness intervention by the media has not received considerable attentions by the researchers.

2. Model Formulation and Basic Properties

A susceptible-infectious-hospitalised-recovered model is formulated based on the assumption of random mixing and nonexistence of spatial structure in the population. The population size (N) is subject to fluctuation over time and the population in each subset of (N) - susceptible (S), infectious (I), hospitalised (H), and recovered (R) could vary over time as well. Treatment is assumed for the infectives due to the presence of hospitals. The transmission model is represented by the following system of nonlinear

first-order ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= r - d_1 S - \left(\alpha_1 - \alpha_2 \frac{I}{m+I}\right) SI + \tau R \\
\frac{dI}{dt} &= \left(\alpha_1 - \alpha_2 \frac{I}{m+I}\right) SI - (d_1 + d_2 + \sigma + \gamma_1) I \\
\frac{dH}{dt} &= \sigma I - (d_1 + d_3 + \gamma_2) H \\
\frac{dR}{dt} &= \gamma_1 I + \gamma_2 H - (d_1 + \tau) R,
\end{aligned} \tag{2.1}$$

where, $S(0) > 0, I(0) \geqslant 0, H(0) \geqslant 0$ and $R(0) \geqslant 0$.
In the system (1), the susceptibles are recruited at rates r and τ. The susceptible population is decreased through infection (moving to class I), and by natural mortality at rate d_1. The infective population increases through the effective contact between the susceptibles and the infectives. The population however, reduces through hospitalisation (moving to class H at rate σ), recovery unrelated to medical treatment (moving to class R at rate γ_1), natural mortality at rate d_1 and mortality due to infection at rate d_2. The population of hospitalised individuals increases when infected individuals seek medical attention at rate σ. However, it is reduced through successful medical treatment (moving to class R at rate γ_2), natural mortality at rate d_1 and treatment failure which results in death at rate d_3. The recovered compartment increases when individuals recover from infections either naturally or through treatment at rates γ_1 and γ_2 respectively but the compartment reduces when the recovered individuals die naturally at rate d_1 or when they are reinfected with the disease at rate τ. In the model, $\alpha(I) = \alpha_1 - \alpha_2 \left(\frac{I}{m+I}\right)$ is the effective contact rate induced by media intervention. The effective contact rate is considered in terms of the nonlinear function $\alpha(I)$ because people will take protective measures immediately after the infected individuals are reported by the media, which will slow down the spreading rate of the disease. Usually, the more the rate of appearance of infectious individuals in a population, the more the susceptibles reduce their contacts to prevent being infected. Therefore, the term $\alpha_2 \left(\frac{I}{m+I}\right)$ is used to indicate the minimised value of the spreading rate when infectives emerge and are reported. As $I \to \infty$, the minimised value of the spreading rate reaches its greatest value (i.e. α_2). Also, The minimised value of the spreading rate is equal to half its maximum value (i.e. α_2) when the number of reported infectious individuals reaches m. Because the implementation of the media intervention cannot stop disease transmission totally, it is obvious that $\alpha_1 \geqslant \alpha_2$. m indicates the responsive velocity of the population to the disease in terms of the media intervention. As media coverage increases, it is believed that the propensity to contract a disease decreases. Media coverage expansion and the disease propagation are inversely related. If $\alpha_2 = 0$ then $\alpha(I) = \alpha_1$ and the spreading rate is constant, a phenomenon that has been investigated by some scholars [1, 32, 36].

For the bounded and nonnegative solutions of the system (1), the domain of attraction exists within the set:

$$\Omega = \left\{(S, I, H, R) \in \mathbb{R}^4_+ : S + I + H + R \leqslant \frac{r}{d_1}\right\},$$

and attracts every solution in the interior of nonnegative octant.

Proof.

$$\begin{aligned}
N &= S + I + H + R \Rightarrow \\
\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{dH}{dt} + \frac{dR}{dt} \Rightarrow \\
\frac{dN}{dt} &\leqslant r - d_1 N \Rightarrow
\end{aligned}$$

$$\frac{d}{dt}(Ne^{d_1 t}) \leqslant re^{d_1 t}. \tag{2.2}$$

Integrating (2) within the limit 0 to t then

$$N(t) \leqslant N_0 e^{-d_1 t} + \frac{r}{d_1}.$$

Thus, following differential inequality theory in [30], $\lim_{t\to\infty} \text{Sup}\, N(t) \leqslant \frac{r}{d_1}$, i.e. the total population cannot grow beyond $N(t) \to N_\infty \leqslant \frac{r}{d_1}$. □

3. Existence of Equilibria and Stability

It is easy to confirm that the system (1) admits a single disease-free equilibrium $\mathcal{E}_\circ = (S_0, I_0, H_0, R_0) = \left(\frac{r}{d_1}, 0, 0, 0\right)$. The reproduction number, $\mathcal{R}_\circ$ is the quantity that measures the anticipated quantity of resultant infections instigated by an infective upon emergence in a completely susceptible population [19, 20]. The quantity is important not only in quantifying the transmission potential of a disease but also in providing information for checking the transmission of the disease [26]. Following [20], the stability of the infection-free equilibrium $\mathcal{E}_\circ$ of system (1) is determined by the size of the $\mathcal{R}_\circ$ of the system. Following the method in [20], we obtained

$$\mathcal{F} = \left(\left(\alpha_1 - \alpha_2 \frac{I}{m+I}\right) SI, 0\right)^T$$

and,

$$\mathcal{V} = \begin{pmatrix} (d_1 + d_2 + \sigma + \gamma_1)I \\ -\sigma I + (d_1 + d_3 + \gamma_2)H \end{pmatrix}.$$

A direct computation gives

$$\mathcal{F} = \begin{pmatrix} \frac{r\alpha_1}{d_1} & 0 \\ 0 & 0 \end{pmatrix}, \tag{3.1}$$

$$\mathcal{V} = \begin{pmatrix} d_1 + d_2 + \sigma + \gamma_1 & 0 \\ -\sigma & d_1 + d_3 + \gamma_2 \end{pmatrix}. \tag{3.2}$$

$$\mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{d_1 + d_2 + \sigma + \gamma_1} & 0 \\ \frac{\sigma}{(d_1 + d_2 + \sigma + \gamma_1)(d_1 + d_3 + \gamma_2)} & \frac{1}{(d_1 + d_3 + \gamma_2)} \end{pmatrix} \tag{3.3}$$

$$\mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} \frac{r\alpha_1}{d_1(d_1 + d_2 + \sigma + \gamma_1)} & 0 \\ 0 & 0 \end{pmatrix} \tag{3.4}$$

and,

$$\mathcal{R}_0 = \frac{r\alpha_1}{d_1(d_1 + d_2 + \sigma + \gamma_1)}. \tag{3.5}$$

The endemic equilibrium $\mathcal{E}^*$ exists when the disease invades the population in such a way that $I^* > 0$. This equilibrium with the point (S^*, I^*, H^*, R^*) is obtained when each equation in the system (1) is reduced

to zero such that

$$S^* = \frac{d_1 + d_2 + \sigma + \gamma_1}{\alpha_1 - \alpha_2 \dfrac{I^*}{m + I^*}}, \tag{3.6}$$

$$H^* = \frac{\sigma I^*}{d_1 + d_3 + \gamma_2}, \tag{3.7}$$

$$R^* = \frac{1}{d_1 + \tau}\left[\gamma_1 I^* + \frac{\sigma\gamma_2 I^*}{d_1 + d_3 + \gamma_2}\right]. \tag{3.8}$$

The endemic equilibrium satisfies the condition $S^* > 0$, $I^* > 0$, $H^* > 0$ and $R^* > 0$. I^* can be determined from the first equation of the system (1) by appropriate substitution.

Theorem 3.1. *There exists a disease-free equilibrium for the system (1) that is locally and globally asymptotically stable if $\mathcal{R}_\circ < 1$ [14].*

Proof. The Jacobian of the system (1) about the disease-free equilibrium $\mathcal{E}_\circ$ is

$$J\left(\frac{r}{d_1}, 0, 0, 0\right) = \begin{pmatrix} -d_1 & -\dfrac{r\alpha_1}{d_1} & 0 & \tau \\ 0 & (d_1 + d_2 + \sigma + \gamma_1)(\mathcal{R}_\circ - 1) & 0 & 0 \\ 0 & \sigma & -(d_1 + d_3 + \gamma_2) & 0 \\ 0 & \gamma_1 & \gamma_2 & -(d_1 + \tau) \end{pmatrix} \tag{3.9}$$

The characteristic equation of (11) about $\mathcal{E}_\circ$ has the eigenvalues

$$\lambda_1 = -d_1, \lambda_2 = -(d_1 + \tau), \lambda_3 = -(d_1 + d_3 + \gamma_2) \text{ and } \lambda_4 = (d_1 + d_2 + \sigma + \gamma_1)(\mathcal{R}_\circ - 1).$$

Obviously, $\lambda_4 < 0$ if $\mathcal{R}_\circ < 1$ and the equilibrium about $\mathcal{E}_\circ$ is locally asymptotically stable otherwise $\mathcal{E}_\circ$ is locally asymptotically unstable. To examine the global stability of the model about $\mathcal{E}_\circ$, the disease-free equilibrium, the Lyapunov function $V = I$ is chosen and the differentiation of V about the solution of system (1) is considered thus

$$\begin{aligned} \dot{V} = \dot{I} &= \left(\alpha_1 - \alpha_2 \frac{I}{m + I}\right) S_0 I - (d_1 + d_2 + \sigma + \gamma_1) I, \\ &\leqslant \alpha_1 S_0 I - (d_1 + d_2 + \sigma + \gamma_1) I, \\ &\leqslant (d_1 + d_2 + \sigma + \gamma_1)(\mathcal{R}_\circ - 1), \\ &\leqslant 0. \end{aligned}$$

If $\mathcal{R}_\circ < 1$ then $\dot{V} < 0$ and $\mathcal{E}_\circ$ is globally asymptotically stable. However, if $\mathcal{R}_\circ > 1$, infections emerge in the population and the equilibrium switches from $\mathcal{E}_\circ$ to $\mathcal{E}^*$. The stability about $\mathcal{E}^*$, the endemic equilibrium, can be examined following linearisation approach as in [34]. The Jacobian about $\mathcal{E}^*$, is computed as

$$J(S^*, I^*, H^*, R^*) = \begin{pmatrix} -d_1 - A_1 & A_2 & 0 & \tau \\ A_1 & A_3 & 0 & 0 \\ 0 & \sigma & -(d_1 + d_3 + \gamma_2) & 0 \\ 0 & \gamma_1 & \gamma_2 & -(d_1 + \tau) \end{pmatrix}, \tag{3.10}$$

where

$$\begin{aligned} A_1 &= \left(\alpha_1 - \alpha_2 \frac{I^*}{m + I^*}\right) I^*, \\ A_2 &= -(d_1 + d_2 + \sigma + \gamma_1) + \frac{\alpha_2 m I^* (d_1 + d_2 + \sigma + \gamma_1)}{[\alpha_1 m + (\alpha_1 - \alpha_2) I^*](m + I^*)}, \\ A_3 &= -\frac{\alpha_2 m I^* (d_1 + d_2 + \sigma + \gamma_1)}{[\alpha_1 m + (\alpha_1 - \alpha_2) I^*](m + I^*)}. \end{aligned}$$

Row operation reduces (12) to

$$J(S^*, I^*, H^*, R^*) = \begin{pmatrix} -(d_1 + A_1) & A_2 & 0 & \tau \\ 0 & \frac{(d_1 + A_1)}{A_1} A_3 + A_2 & 0 & 0 \\ 0 & \sigma & -(d_1 + d_3 + \gamma_2) & 0 \\ 0 & \gamma_1 & \gamma_2 & -(d_1 + \tau) \end{pmatrix}, \tag{3.11}$$

The characteristic equation of (13) has the eigenvalues

$$\lambda_1 = -(d_1 + A_1), \lambda_2 = -(d_1 + \tau), \lambda_3 = -(d_1 + d_3 + \gamma_2) \text{ and } \lambda_4 = \frac{(d_1 + A_1)}{A_1} A_3 + A_2$$

λ_2 and λ_3 are negative. Therefore, the endemic equilibrium of the model is locally asymptotically stable if $\lambda_1 < 0$ and $\lambda_4 < 0$. The existence of global stability of the endemic equilibrium $\mathcal{E}^*$ of the model can also be verified via Lyapunov function. □

Theorem 3.2. *The endemic equilibrium* E^* *of system (1) is globally asymptotically stable in* Ω *if* $\dot{W} < 0$ *, where* W *is the Lyapunov function.*

Proof. Construct the Lyapunov functional

$$W = \frac{1}{2} k_1 (S - S^*)^2 + \frac{1}{2} k_2 (I - I^*)^2 + \frac{1}{2} k_3 (H - H^*)^2 + \frac{1}{2} k_4 (R - R^*)^2, \tag{3.12}$$

where k_1, k_2, k_3 and k_4 are nonnegative constants.

$$\begin{aligned} \Rightarrow \dot{W} &= k_1 (S - S^*) \dot{S} + k_2 (I - I^*) \dot{I} + k_3 (H - H^*) \dot{H} + k_4 (R - R^*) \dot{R} \\ &= k_1 (S - S^*) \left[r - d_1 S - \left(\alpha_1 - \alpha_2 \frac{I}{m + I} \right) SI + \tau R \right] \\ &+ k_2 (I - I^*) \left[\left(\alpha_1 - \alpha_2 \frac{I}{m + I} \right) SI - (d_1 + d_2 + \sigma + \gamma_1) I \right] \\ &+ k_3 (H - H^*) [\sigma I - (d_1 + d_3 + \gamma_2) H] \\ &+ k_4 (R - R^*) [\gamma_1 I + \gamma_2 H - (d_1 + \tau) R] \end{aligned} \tag{3.13}$$

$$\begin{aligned} \Rightarrow \dot{W} &= [r k_1 (S - S^*) + \tau k_1 (S - S^*) R + \alpha_1 k_2 (I - I^*) + k_3 \sigma (H - H^*) I \\ &+ k_4 \gamma_1 (R - R^*) I + k_4 \gamma_2 (R - R^*) H] \\ &- [k_1 d_1 (S - S^*) S + k_1 (S - S^*) \left(\alpha_1 - \alpha_2 \frac{I}{m + I} \right) SI \\ &+ k_2 (I - I^*) \left(\alpha_1 - \alpha_2 \frac{I}{m + I} \right) SI + k_2 (d_1 + d_2 + \sigma + \gamma_1) (I - I^*) I^* \\ &+ k_3 (d_1 + d_3 + \gamma_2) (H - H^*) H + k_4 (d_1 + \tau) (R - R^*) R] \end{aligned} \tag{3.14}$$

Suppose

$$\dot{W} = N_1 - N_2 \tag{3.15}$$

then, $\dot{W} < 0$ if and only if $N_1 < N_2$. Also, $\dot{W} = 0$ if $S = S^*, I = I^*, H = H^*$ and $R = R^*$. Following LaSalle's invariance principle [31], E^* remains globally asymptotically stable in Ω if $N_1 < N_2$ where E^* is the endemic equilibrium of the model. □

Assuming the conditions $\lambda_1 < 0$, $\lambda_4 < 0$ and $N_1 < N_2$ are not satisfied then the endemic equilibrium of the model is locally and globally asymptotically unstable. It means that $\mathcal{R}_\circ < 1$ and the disease either fails to take off or dies out. In case the disease dies out (i.e. disappears gradually from the population),

there is a tendency for a reemergence as in the case of recently experienced second wave of COVID-19 pandemic. The epidemiological concept that addresses the possibility of disease persistence when $\mathcal{R}_\circ < 1$ is bifurcation [7]. Bifurcation can be forward or backward. It is forward if the condition $\mathcal{R}_\circ < 1$ is sufficient to eradicate a disease from the population while it is backward when the condition $\mathcal{R}_\circ < 1$ is only necessary but not sufficient to eliminate the disease from the population [7]. We shall verify the existence of bifurcation phenomenon for our model using the Centre Manifold Theory formulated in [15]. By changing the variables S to x_1, I to x_2, H to x_3 and R to x_4 such that $\mathbf{X} = (x_1, x_2, x_3, x_4)^T$ and $\frac{dX}{dt} = F(X)$ where $F = (f_1, f_2, f_3, f_4)$ then the system (1) is transformed to

$$\frac{dX_1}{dt} = f_1 = r - d_1 x_1 - \left(\alpha_1 - \alpha_2 \frac{x_2}{m + x_2}\right) x_1 x_2 + \tau x_4 \tag{3.16}$$

$$\frac{dX_2}{dt} = f_2 = \left(\alpha_1 - \alpha_2 \frac{x_2}{m + x_2}\right) x_1 x_2 - (d_1 + d_2 + \sigma + \gamma_1) x_2 \tag{3.17}$$

$$\frac{dX_3}{dt} = f_3 = \sigma x_2 - (d_1 + d_3 + \gamma_2) x_3 \tag{3.18}$$

$$\frac{dX_4}{dt} = f_4 = \gamma_1 x_2 + \gamma_2 x_3 - (d_1 + \tau) x_4 \tag{3.19}$$

Suppose α_1 is the bifurcation parameter then at the change of stability when $\mathcal{R}_\circ = 1$, it is obvious from the relation $\mathcal{R}_\circ = 1$ that

$$\alpha_1 = \frac{(d_1 + d_2 + \sigma + \gamma_1)}{S_0}. \tag{3.20}$$

If the system (18)-(21) is captured in terms of the new transmission parameter α_1 in (22) indicated by α_1^* then the Jacobian of the system (18)-(21) about $\mathcal{E}_\circ$, the disease-free equilibrium is computed as

$$J(\mathcal{E}_\circ)|_{\alpha_1 = \alpha_1^*} = \begin{pmatrix} -d_1 & -\frac{r\alpha_1^*}{d_1} & 0 & \tau \\ 0 & (d_1 + d_2 + \sigma + \gamma_1)(\mathcal{R}_\circ - 1) & 0 & 0 \\ 0 & \sigma & -(d_1 + d_3 + \gamma_2) & 0 \\ 0 & \gamma_1 & \gamma_2 & -(d_1 + \tau) \end{pmatrix} \tag{3.21}$$

The Jacobian (23) has the right eigenvectors

$$w_1 = -\frac{1}{d_1}\left[\frac{r\alpha_1^*}{d_1} - \frac{\tau[\gamma_1(d_1 + d_3 + \gamma_2) + \sigma\gamma_2]}{(d_1 + \tau)(d_1 + d_3 + \gamma_2)}\right] w_2 \tag{3.22}$$

$$w_2 = w_2 > 0 \tag{3.23}$$

$$w_3 = \frac{\sigma}{(d_1 + d_3 + \gamma_2)} w_2 > 0 \tag{3.24}$$

$$w_4 = \left[\frac{\gamma_1(d_1 + d_3 + \gamma_2) + \sigma\gamma_2}{(d_1 + \tau)(d_1 + d_3 + \gamma_2)}\right] w_2 > 0 \tag{3.25}$$

Also, the left eigenvectors of the Jacobian (23) are derived as

$$v_1 = v_3 = v_4 = 0 \ \text{ but } \ v_2 > 0.$$

The bifurcation coefficients

$$a = \sum_{k,i,j=1}^{4} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

and,

$$b = \sum_{k,i=1}^{4} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha_1^*}(0,0), \tag{3.26}$$

are computed as

$$a = -2\nu_2 \frac{1}{d_1}\left[\frac{r\alpha_1^*}{d_1} - \frac{\tau[\gamma_1(d_1+d_3+\gamma_2)+\sigma\gamma_2]}{(d_1+\tau)(d_1+d_3+\gamma_2)}\right]\alpha_1^* w_2^2 \tag{3.27}$$

and,

$$b = \frac{r}{d_1}\nu_2 w_2 > 0. \tag{3.28}$$

Following item (iv) in ([15], Theorem 4.1), b is always positive and the bifurcation type which the model undergoes is forward if $a < 0$. The existence of forward bifurcation has no adverse health implication as the condition $\mathcal{R}_\circ < 1$ remains the necessary and sufficient condition to eradicate the disease. If $a > 0$, backward bifurcation exists. Looking at equation (29) in our result, it is obvious that a change in the transmission parameter α_1 to α_1^* may instigate backward bifurcation for our model since $a > 0$ is possible. If there is no relapse (i.e. if $\tau = 0$) in (29) then backward bifurcation is sure to be prevented in the disease dynamics addressed by our model but if there is a relapse (i.e. if $\tau > 0$) then backward bifurcation may exist. The existence of backward bifurcation has a major health implication. The condition $\mathcal{R}_\circ < 1$ is just a necessary but not a sufficient condition for disease eradication. The presence of relapse in our model and the possibility of the existence of backward bifurcation in our analysis show that the media awareness programme that reduces the disease spread in the population to the point where $\mathcal{R}_\circ < 1$ has to be maintained if the global elimination of the disease is to be sustained.

4. Numerical Simulations and Discussion of Results

To harmonise the theoretical analysis performed in section 3, numerical behaviour of system (1) is examined. The parameter and variable values are assumed for illustrative purposes within the possible range and the unit of measurement is considered day^{-1} as in [4, 23, 32, 35, 37, 44]. We decide to investigate the bifurcation type that exists with reference to the reproduction number $\mathcal{R}_\circ$ as well as visualise the behaviour of the system. To achieve the purpose, the initial values for the variables are taken as $S_0 = 80, I_0 = 5, H_0 = 3$ and $R_0 = 2$. Also, we fixed $r = 0.15, d_1 = 0.015, d_2 = 0.0143, d_3 = 0.011, \gamma_1 = 0.4$ and $\gamma_2 = 0.8$ while the values for the remaining parameters are varied to carried out the computations. The result of the computations is presented in Table 1 followed by the graphical profiles of the system in Figures 1-8.

Table 1. Bifurcation results at the critical point $\mathcal{R}_\circ = 1$

S/No.	α_1	α_1^*	σ	τ	$\mathcal{R}_\circ$	a	Bifur. type	Remarks
1.	0.05	0.06	0.6	0.6	0.48	0.76	Backward	Persistence
2.	0.06	0.07	0.7	0.6	0.53	0.79	Backward	Persistence
3.	0.07	0.08	0.8	0.6	0.57	0.78	Backward	Persistence
4.	0.08	0.09	0.9	0.00	0.60	-5.40	Forward	Eradication
5.	0.06	0.05	0.6	0.6	0.59	0.97	Backward	Persistence
6.	0.07	0.06	0.7	0.6	0.62	1.07	Backward	Persistence
7.	0.08	0.07	0.8	0.6	0.65	1.15	Backward	Persistence
8.	0.09	0.08	0.9	0.00	0.68	-4.20	Forward	Eradication

As argued earlier, a reduction in $\mathcal{R}_\circ$ below unity can only guarantee an eradication for a disease which is not associated with a relapse. This is confirmed in S/No. 4 and S/No. 8 in Table 1 where backward bifurcation is inhibited only when the relapse parameter τ is zero. This shows that media coverage or appropriate use of media strategy has the tendency not only to eradicate but also to prevent reemergence of a disease which does not relapse upon recovery. On the other hand, at a point where $\mathcal{R}_\circ < 1$, it may mean that the disease persists in the population. A stable disease-free equilibrium co-exists with a stable endemic equilibrium with the existence of backward bifurcation. This is observable in S/No. 1-3 and S/No. 5-7 in Table 1. The existence of $\mathcal{R}_\circ < 1$ at these points is not sufficient to eradicate the disease.

The implication of the results at these points is that while awareness strategies by the media can play a major role in controlling and eradicating infectious diseases, the efforts must be sustained especially for a disease that is characterised by a relapse if total eradication and reemergence are to be achieved.

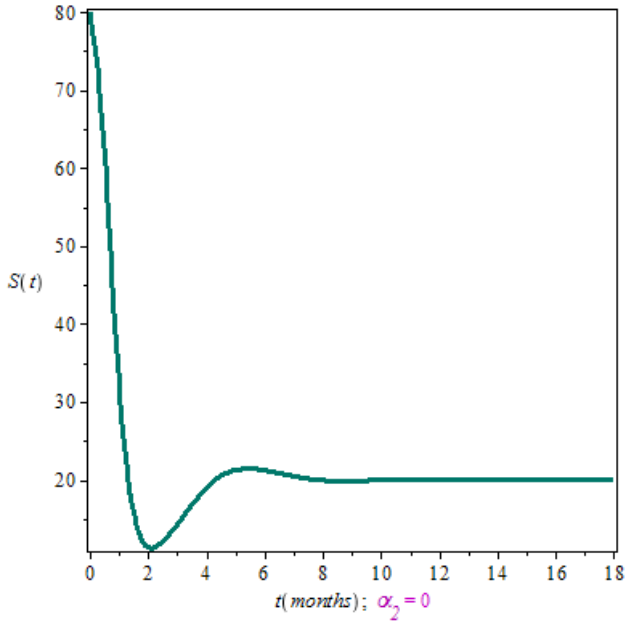

Figure 1: The effect of absence of media intervention on the population of susceptible individuals.

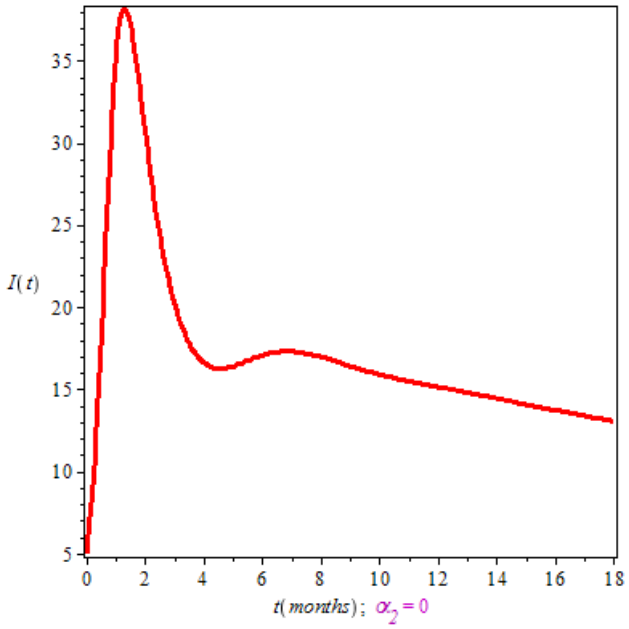

Figure 2: The effect of absence of media intervention on the population of infectious individuals.

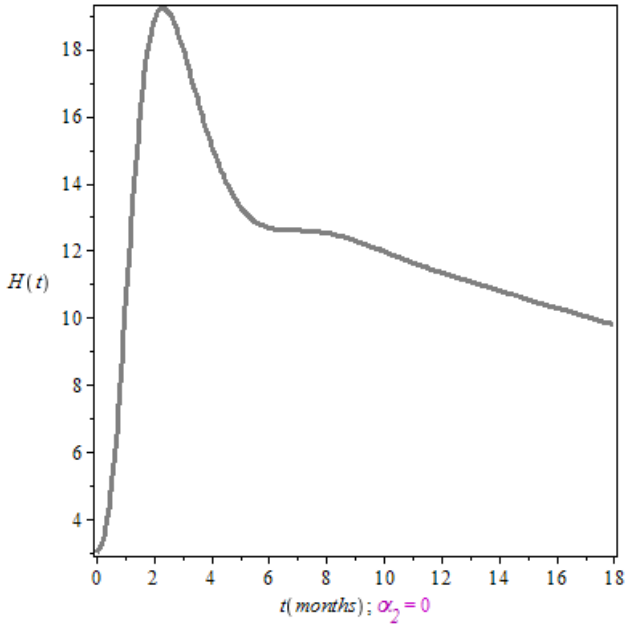

Figure 3: The effect of absence of media intervention on the population of hospitalised individuals.

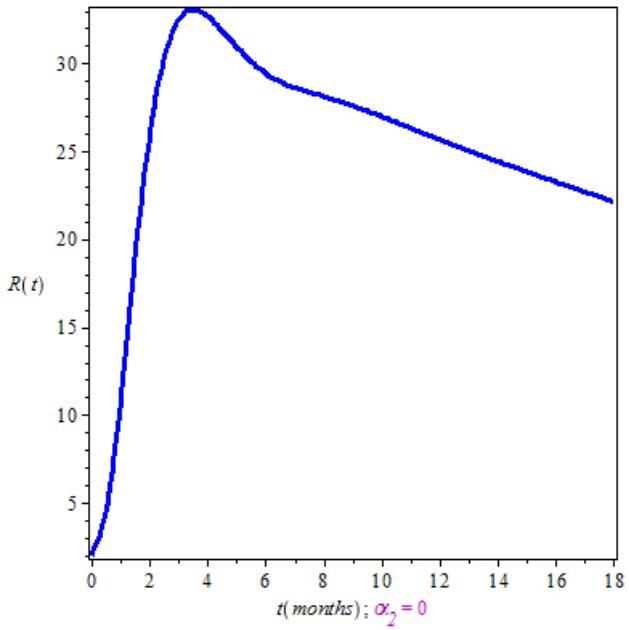

Figure 4: The effect of absence of media intervention on the population of Recovered individuals.

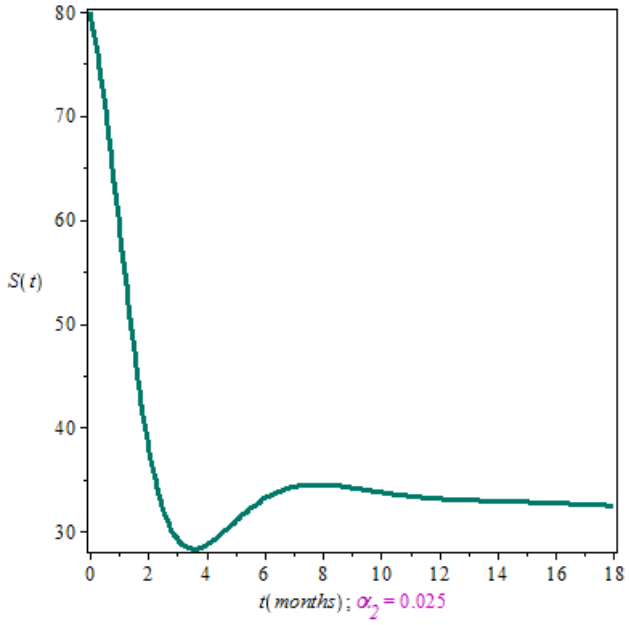

Figure 5: The effect of media intervention on the population of susceptible individuals.

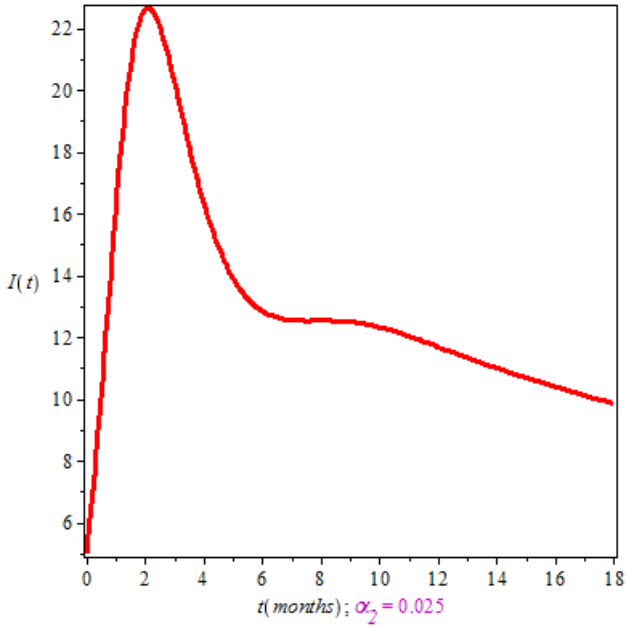

Figure 6: The effect of media intervention on the population of infectious individuals.

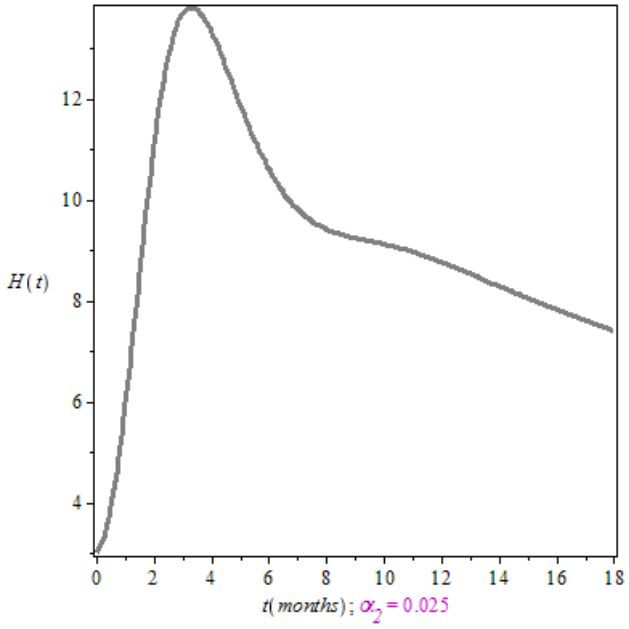

Figure 7: The effect of media intervention on the population of hospitalised individuals.

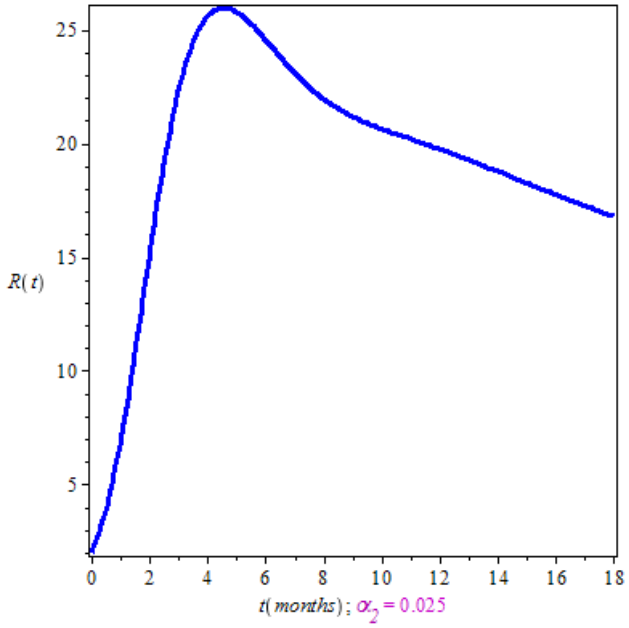

Figure 8: The effect of media intervention on the population of recovered individuals.

Two scenarios are considered in plotting the graphs - a case when there is no media intervention such that $\alpha_2 = 0$ and another case when there is media reports such that $\alpha_2 = 0.025$. All the figures are plotted using the variables and parameters values stated in section 4 except α_2 whose values are stated under each plot. Figures 1-4 depict a scenario when there is no media intervention while Figures 5-8 describe a situation when there is media reports in disease dynamics. In both instances, it is shown that the dynamics of the disease follows the same trend among the susceptible, infectious, hospitalised and recovered individuals as depicted in Figures 1 and 5 for S(t), Figures 2 and 6 for I(t), Figures 3 and 7 for H(t) and Figure 4 and 8 for R(t) respectively. Generally, during an outbreak of a disease, whether there is a media intervention to curb the disease or not, susceptibility and infectivity tend to fall while hospitalisation and recovery tend to rise as illustrated in all the figures. The reason is that disease outbreaks instill fear and panic in individuals and as a result, every individual keeps away from infectious agents to avoid being infected.

Besides, the pain and agony that do accompany infections always propel those who have been infected with a disease to look for the way out which is usually through the medical means. However, awareness campaigns by the media can be a game changer in disease control and management and the impact of awareness created by the media on the disease dynamics has been indicated in our plots especially the plots for the susceptible and infectious individuals. In Figures 1 and 5, it is shown that while susceptibility rises after two months in Figure 1, it rises after four months in Figure 5. The time lag before the increase in susceptibility in Figure 5 is attributable to the presence of media intervention. Also, in Figures 2 and 6, while infections spread to thirty-eight individuals within a month in Figure 2, infections spread to just twenty-three individuals in two months in Figure 6. The delay in the spread of the disease in Figure 6 is as a result of the intervention by the media.

5. Conclusion

A mathematical model of infectious diseases is a theoretical epidemiological method, which we use to simulate various factors that shape the dynamics of infectious diseases. In this work, a four dimensional epidemic model has been proposed to quantify the effects of awareness created by the media strategy on the transmission and management of contagious diseases. We have derived the equilibria of the model, investigated equilibria stability and examined bifurcation phenomenon at the critical point. We have also established the necessary and sufficient conditions for the model equilibria to be locally and globally asymptotically stable. Numerical simulations have been conducted to visualise and complement the theoretical results. Although the awareness campaigns by the media might not be sufficient to eradicate communicable diseases that relapse, media intervention is indispensable in disease control and can instigate delay in susceptibility and infection spread. Awareness programme by the media is therefore a major option to tackle the spread of communicable diseases should there be an outbreak.

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