Role of media coverage and delay in controlling infectious diseases: A mathematical model

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Abstract

The aim of this paper is to investigate the effect of awareness coverage and delay in controlling infectious diseases. We formulate an SIS model considering individuals' behavioral changes due to the influences of media coverage and divide the susceptible class into two subclasses: aware susceptible and unaware susceptible. Other model variables are infected human and media campaign. It is assumed that the rate of becoming aware (unaware), from unaware to aware susceptible human (from aware to unaware susceptible human), is a function of media campaign. A time delay is considered for the time that is taken by an unaware (aware) susceptible individual to become aware (unaware). An additional time delay is considered due to the time lag needed in organising awareness campaigns. The model exhibits two equilibria: the disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium is stable if the basic reproduction number is smaller than unity and the endemic equilibrium exhibits a Hopf-bifurcation, in both delayed and non-delayed system, whenever it exists. Analytical and numerical results prove the significance of awareness and delay on the prevalence of infectious diseases.

Key words: Infectious disease, Awareness program, Mathematical model, Time delay, Hopf-bifurcation.

1. Introduction

Infectious diseases are major nuisances to mankind. They cause mortality, disability, as well as social and economic disturbance for society. Pneumonia, Tuberculosis (TB), Diarrheal diseases (Cholera), Malaria, Measles and more

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recently HIV/AIDS etc. are the major deadly infectious diseases. Including premature deaths and deaths of young children, in developing countries approximately 11 million people die each year due to infectious diseases [1, 2, 3, 4].

At the beginning of an epidemic outbreak, the initial step to try to control it, is to make people aware of the disease through the media and to let them know preventive measures that can be adopted. The media coverage is obviously not the most important factor responsible for fighting the transmission of the infectious disease, but it is a very important issue which has to be considered seriously. In the case of a large number of infected cases, on one hand, media coverage may cause panic in the society, while on the other hand, it can certainly reduce the opportunity of contact among the alerted susceptible populations, which in turn helps to control the transmission probability and prevent the disease from further spreading [5].

It is well known that some diseases upon recovery give permanent immunity, e.g. influenza, measles, chickenpox, while for other ones the immunity is only temporary, for instance gonorrhea, meningitis, tuberculosis. For a third class of illnesses, no vaccination is yet available: malaria, dengue, chikungunya, AIDS. In the latter case, the best control at the moment is to prevent people to become infected. This can be obtained for instance by suitable campaigns through the media, [6, 7].

Disease propagation in an epidemic outbreak is heavily dependent on the people's behavior. In Bari, South-East Italy, the cholera outbreak of 1973 has been curbed within a few days because the local individuals implemented severe measures to avoid its further spread. This gave rise to one of the now classical models, the so-called Capasso-Serio epidemic model, [8], which incorporates the individual behavior in the Monod-Haldane functional response. The infection rate decreases with an increasing number of reported infected, as susceptibles take stricter measures to avoid to be infected themselves, a fact that is well known in the literature, [9, 6, 10]. The essential mean that contributed to this change in behavior in the cholera epidemics was the information that media were providing. Indeed, during the initial phase of the epidemics, most people and public mass media are in general unaware of the disease, but as the awareness of it disseminates, people respond and eventually change their behavior to reduce their susceptibility. Media familiarizes people with the diseases and the possible preventive means to avoid becoming infected, e.g. social distancing, wearing protective masks, practice of better hygiene, use of preventive medicaments, vaccination, voluntary quarantine. People aware of the danger of the epidemic spread adopt practices to try to minimize their exposure to contagion, a fact that may deeply influence the epidemic pattern, [11, 12].

Recent investigations have begun to introduce explicitly the role that media campaigns possess to influence people's behavior during epidemics outbreaks, see for instance [13, 14, 15]. In the mathematical epidemic models, this in general is obtained by partitioning the whole susceptible population among aware people and those that are not knowledgeable of the proper ways to reduce the risks of infection, [16]. However, because of the initial lag in realizing the danger of the epidemics spread and render it public, generally the response from the people

is not immediate. This fact should be taken into consideration for a proper modeling of the situation. A formulation that suitably incorporates time delays becomes imperative in this situation. Models of this kind have been considered in the literature, [17, 18, 19, 20]. Among the findings, the time delay between the advertising campaigns and the moment in which people start to act deeply influences the endemic equilibrium stability leading to periodic oscillations when the basic reproduction number exceeds unity. The similar delay in reporting the epidemic outbreak has been considered in [21], while a modification with the assumption that the growth rate of aware people increases at a rate proportional to the infective population is presented in [22]. Two delays, one in reporting of the infected cases, and the other due to the fading away of disease awareness after a fixed period of time are studied in [14], with the findings that an increase in the duration of awareness reduces the equilibrium level of infected. Both time delays can destabilize the endemic equilibrium and trigger persistent oscillations.

In this study, a mathematical model is proposed to investigate the change in prevalence of an infectious disease when an awareness program through the media is employed. Our main aim is the study of the impact on the epidemic outbreak of the combined action of the awareness program and the time delays. The former is not present in [21]. In [19] an SIRS model is presented with an infected-dependent rate in the transmission process, but no explicit modelization of the media coverage is included. In [17] there is only one delay, also in the infected reports in the media advertisements. Note also that awareness recruitments are not constant, but rather assumed to depend on the media campaigns, $M(t)$ following an explicit saturation function. For us instead, this functional dependence is not only nonlinear, as for instance in [22], but expressed via a generic, monotonically increasing function f. Both these aspects are more realistic because in reality it is difficult to properly estimate these behavioral rates. A model closer to ours is presented in [14]. The functional responses for the media campaign are assumed of specific Holling type II form, for recruitment of aware people, while we generalize it through the generic function f bearing similar mathematical properties. Instead, a broader view is taken in [14] as far as the effect of the advertisement has on the contact rate, which is expressed by a hyperbola tending to vanish when M becomes large. In [11], the authors consider the levels of human awareness to model the effect of awareness and a time delay is introduced to take into account the time needed by unaware people to become aware. They have shown the existence of a Hopf bifurcation when the time delay parameter crosses a critical value. In the model proposed here, we simply take this contact rate constant. Finally, the fading away of implementing the safety measures occurs here via the generic nonlinear monotonically decreasing function G instead than at a constant rate. There are two delays also in [14], one for the time to implement the preventive measures, while the other one involves memory fading away. In our case instead, two time delays are also considered. The first one accounts for the time that is taken by unaware (aware) susceptible individuals to become aware (unaware). The second one is considered for the organisation and implementation of awareness campaigns. The endemic equilibrium exhibits a Hopf-bifurcation, in both delayed

and non-delayed system.

The paper is organized as follows. The model is described next, followed in Section 3 by the analysis of the system with no delay. Delays are treated in the subsequent Section while Section 5 contains the numerical experiments. A final discussion concludes the paper.

2. The mathematical model

Let $S(t)$ and $I(t)$ be the density of the susceptible and infected populations respectively at time t. Further, the total susceptible population is divided into two subclasses: the susceptible population unaware of the disease fighting means, S_u and the aware susceptible population S_a. Here we stress that "aware" does not mean "informed" of the existence of the epidemics, but also knowledgeable of ways of avoiding disease propagation and further implementing these prevention mechanisms. $M(t)$ represents the amount of media campaign, measured possibly by the time exposure to TV and radio advertising, or in the number and size of posters placed in public areas.

As the awareness disseminates, people respond to it and eventually modify their behavior to reduce their susceptibility. Usually, aware susceptible individuals contract the disease at a lower rate than unaware individuals. The disease is transmitted from infected to susceptible individuals following a mass action functional form. Also, infected individuals recover through appropriate treatment. After recovery, a fraction p of recovered people will join the aware susceptible class, whereas the remaining fraction $1-p=q$, hopefully small, will join the unaware class. We ignore demographic issues and assume only that in the system there is a constant recruitment of new susceptible individuals assumed all to be unaware of the epidemics threat, at rate π.

The model reads:

$$\begin{aligned}
\frac{dS_u}{dt} &= \pi - \beta S_u I - \alpha f(M) S_u + \lambda g(M) S_a - d S_u + qrI \\
\frac{dS_a}{dt} &= \alpha f(M) S_u - \lambda g(M) S_a - \gamma S_a I - d S_a + prI \\
\frac{dI}{dt} &= \beta S_u I + \gamma S_a I - (d+e+r) I \\
\frac{dM}{dt} &= \eta e I - \theta M,
\end{aligned} \tag{1}$$

with the initial conditions:

$$S_u(0) = S_{0u}, \quad S_a(0) = S_{0a}, \quad I(0) = I_0, \quad M(0) = M_0.$$

In the first equation the unaware individuals are modeled. After recruitment they may become infected at rate β, by contact with an infected individual, but they can also listen to the advertisement campaigns and become aware, at rate α. The modeling of the recruitment campaign is expressed by the function $f(M)$, discussed below. Similarly, the function $g(M)$ models the fading away of

disease prevention measures among the unaware people, which makes them to return into the class of unaware at rate λ, fourth term in the first equation. The natural mortality rate of the population is d. The last term represents the input of successfully recovered infected individuals. Despite their previous exposure to the disease, the latter keep on not following suitable prevention means.

The second equation models the aware population, recruited through successful advertisements, whose correct behavior however might fade away. They can leave this class by becoming infected by contact with a diseased individual, at rate γ. They are exposed to natural mortality. Finally new recruitment into this class also come from all the disease-recovered individuals that have learnt the lesson and keep on applying preventive measures against disease propagation.

In the third equation the dynamics of the infected appears, recruited from unaware susceptible individuals at rate β via "successful" contacts among infected and from the aware class at rate γ. They disappear from this class by dying naturally, or by disease-related causes, at rate e, or by recovering, at rate r. Here η is the proportionality constant which governs the implementation of awareness programs.

The equation for the media campaign is assumed to model the fact that these advertisements grow with the report of increasing disease-induced deaths, and fade away at rate θ.

In the model, $f(M)$ is an increasing function of M with $f(0) = 0$ and $\sup f(M) = 1$. The memory fading and/or carelessness for which aware people stop taking preventive measure for simplicity is taken as $g(M) = 1 - f(M)$. Therefore, $g(M)$ is a decreasing function of M with $g(0) = 1$ and $\inf g(M) = 0$.

The delay model considers the fact that between the moment an advertising is seen or heard and the moment in which people act accordingly, there is a lag τ_1, due to a "pondering" time to take the decision to follow the preventive measures. Further, there is a need of time to organize the media campaign after the disease-induced deaths reports, modeled by the variable τ_2.

The delay model is then:

$$\begin{aligned}
\frac{dS_u}{dt} &= \pi - \beta S_u I - \alpha f(M(t-\tau_1))S_u + \lambda g(M) S_a - dS_u + qrI \\
\frac{dS_a}{dt} &= \alpha f(M(t-\tau_1))S_u - \lambda g(M) S_a - \gamma S_a I - dS_a + prI \\
\frac{dI}{dt} &= \beta S_u I + \gamma S_a I - (d+e+r)I \\
\frac{dM}{dt} &= \eta e I(t-\tau_2) - \theta M,
\end{aligned} \tag{2}$$

with the initial conditions:

$$S_u(\phi) = S_{0u} > 0, S_a(\phi) = S_{0a} > 0, I(\phi) = I_0 > 0, M(\phi) = M_0 > 0, \tag{3}$$

where $\phi \in (-\tau, 0]$, and $\tau = \max\{\tau_1, \tau_2\}$.

For later use, let us rewrite (2) in the following compact form,

$$\frac{dX}{dt} = P(X), \quad X = (x_1, x_2, x_3, x_4)^T, \quad P = (P_1, P_2, P_3, P_4)^T, \tag{4}$$

where P_i's represent the right hand sides of (2).

2.1. Positive invariance

Biologically, positivity for a population implies its survival. We have the following result.

Theorem 2.1. *All the solution of (2) with initial conditions (3) are positive.*

Proof. Using the lemma in [23, 24], the solution of the system (2) exists in the region $\mathbb{R}^4_+$ and all solutions remain non-negative for all $t > 0$. Indeed, it is easy to check in system (4) that whenever choosing $X(\phi) \in R_+$ such that $S_u = 0, S_a = 0, I = 0, M = 0$, it follows $P_i(X)|_{x_i=0, X\in R^4_+} \geq 0$, with $x_1(t) = S_u(t), x_2(t) = S_a(t), x_3(t) = I(t), x_4(t) = M(t)$.

Now, using the lemma in [23], and the theorem in [24], any solution of (1) with $X(\phi) \in C$, say $X(t) = X(t, X(\phi))$, satisfies $X(\phi) \in R^4_+$ for all $t \geq 0$. Hence the solution of the system (2) exists in the region R^4_+ and all components remain non-negative for all $t > 0$. Therefore, the positive cone $\mathbb{R}^4_+$ is an invariant region. □

For the analysis of model (1), the region of attraction is given by the set:

$$B = \left\{(S_u, S_a, I, M) \in R^4_+ : 0 \leq S_u + S_a + I \leq \frac{\pi}{d}, 0 \leq M \leq \frac{\eta e \pi}{\theta d}\right\}. \tag{5}$$

This result implies that all the populations in the model are bounded for all times, which biologically implies robustness of the model.

3. Equilibria analysis

System (1) has two equilibria, the disease-free equilibrium, $E_0(S^0_u, 0, 0, 0)$ with $S^0_u = \pi d^{-1}$ and the endemic equilibrium, $E^*(S^*_u, S^*_a, I^*, M^*)$. Note that in the absence of the disease, also the precautious behavior of the population disappears, $S^0_a = 0$. For the populations at coexistence level, we find

$$\begin{aligned} S^*_u &= \frac{(d + e + qr)\gamma I^* + (d + e + r)[\gamma g(M^*) + d]}{\beta(\gamma I^* + \lambda g(M^*) + d) + \alpha f(M^*)\gamma}, \\ S^*_a &= \frac{(d + e + r)\alpha f(M^*) + \beta p r I^*}{\beta(\gamma I^* + \lambda g(M^*) + d) + \alpha f(M^*)\gamma}, \quad I^* = \frac{\theta M^*}{e\eta}. \end{aligned} \tag{6}$$

and M^* is a positive, real root of the following equation:

$$\begin{aligned} F(M) &= \pi\Big[\beta\Big\{\gamma\frac{\theta M}{\eta e} + \lambda g(M) + d\Big\} + \alpha f(M)\gamma\Big] \\ &-\Big[\frac{\theta\beta M}{e\eta} + \alpha f(M) + d\Big](d + e + r)\Big[\gamma\frac{\theta M}{\eta e} + \gamma g(M) + d\Big] \\ &+\lambda g(M)\Big[(d + e + r)\alpha f(M) + \beta p r\frac{\theta M}{\eta e}\Big] + \frac{qr\theta M}{e\eta} = 0. \end{aligned} \tag{7}$$

This can be recast in the form $\mathcal{L} = \mathcal{V}$ with

$$\mathcal{L}(M) = \pi\Big[\beta\Big\{\gamma\frac{\theta M}{\eta e} + \lambda g(M) + d\Big\} + \alpha f(M)\gamma\Big] \tag{8}$$

$$+\lambda g(M)\Big[(d+e+r)\alpha f(M) + \beta pr\frac{\theta M}{\eta e}\Big] + \frac{qr\theta M}{e\eta} = L_1(M)M + L_0(M),$$

$$\mathcal{V}(M) = \Big[\frac{\theta\beta M}{e\eta} + \alpha f(M) + d\Big](d+e+r)\Big[\gamma\frac{\theta M}{\eta e} + \gamma g(M) + d\Big]$$

$$= V_2(M)M^2 + V_1(M)M + V_0(M),$$

Since $0 \leq f(M), g(M) \leq 1$, let us define the following curves that bound from below and from above the previous ones and do not depend on the nonlinear functions f and g:

$$\mathcal{L}^-(M) = L_1^- M + L_0^-, \quad L_1^- = \frac{\theta}{\eta e}\left[\beta\gamma\pi + qr\right], \quad L_0^- = \beta\pi d, \tag{9}$$

$$\mathcal{L}^+(M) = L_1^+ M + L_0^+, \quad L_1^+ = L_1^- + \lambda\frac{\theta}{\eta e}\beta pr,$$

$$L_0^+ = L_0^- + \pi\Big[\beta\lambda + \alpha\gamma\Big] + \alpha\lambda(d+e+r)$$

$$\mathcal{V}^-(M) = V_2^- M^2 + V_1^- M + V_0^-, \quad V_0^- = d^2(d+e+r),$$

$$V_1^- = \frac{\theta}{e\eta}(\beta+\gamma)d(d+e+r), \quad V_2^- = \frac{\theta^2\beta\gamma}{e^2\eta^2}(d+e+r)$$

$$\mathcal{V}^+(M) = V_2^+ M^2 + V_1^+ M + V_0^+, \quad V_1^+ = V_1^- + \frac{\gamma\theta}{e\eta}[\alpha+\gamma](d+e+r),$$

$$V_0^+ = V_0^- + (d+e+r)[\alpha\gamma + d(\alpha+\gamma)], \quad V_2^+ = V_2^-.$$

These pairs of curves define two stripes in the $M - \mathbb{R}$ plane, that contain the original curves, i.e.

$$\mathcal{L}^- \leq \mathcal{L} \leq \mathcal{L}^+, \quad \mathcal{V}^- \leq \mathcal{V} \leq \mathcal{V}^+. \tag{10}$$

Further $\mathcal{L}$ is a straight line with both positive slope and height at the origin and $\mathcal{V}$ is a convex quadratic, with also positive slope and height at the origin. Thus $\mathcal{L}^\pm$ and $\mathcal{V}^\pm$ inherit these properties. If we require $V_0^+ \leq L_0^-$, it follows that these four curves intersect at four points, that together with $\mathcal{L}^-$, $\mathcal{L}^+$, $\mathcal{V}^-$ and $\mathcal{V}^+$ delimit a compact set Γ. In view of the bounds (10), it follows that $\mathcal{L}$ and $\mathcal{V}$ must intersect in Γ at a positive abscissa M^* and therefore ensure an intersection so that the existence of E^* in the first quadrant is guaranteed. Explicitly, the sufficient condition becomes

$$\beta\pi d \geq (\alpha+d)(d+\gamma)(d+e+r). \tag{11}$$

Alternatively, in view of these considerations, the two curves will meet also if their points for $M = 0$ are interlaced, namely $\mathcal{L}(0) > \mathcal{V}(0)$, i.e. the following condition holds:

$$\beta\pi(\lambda+d) \geq d(d+\gamma)(d+e+r). \tag{12}$$

4. Stability analysis

In this section we analyze the model (2) without and with delay. We also derive the stability conditions for the equilibrium E^* as well as the conditions for Hopf-bifurcation.

4.1. Stability analysis without delay (i.e. $\tau_1 = \tau_2 = 0$)

The Jacobian matrix $V = [V_{ij}]$ of (1) at any generic equilibrium point is:

$$V = \begin{pmatrix} -\beta I - \alpha f(M) - d & \lambda g(M) & -\beta S_u + qr & V_{14} \\ \alpha f(M) & V_{22} & pr - \gamma S_a & V_{24} \\ \beta I & \gamma I & V_{33} & 0 \\ 0 & 0 & \eta e & -\theta \end{pmatrix},$$

with

$$V_{14} = -\alpha f'(M)S_u + \lambda g'(M)S_a, \quad V_{22} = -\lambda g(M) - d - \gamma I,$$
$$V_{24} = \alpha f'(M)S_u - \lambda g'(M)S_a, \quad V_{33} = \beta S_u + \gamma S_a - (d + e + r).$$

At the disease-free equilibrium E_0, the characteristic equation of the Jacobian matrix has only negative eigenvalues if

$$\beta\pi - d(d + e + r) < 0. \tag{13}$$

Let

$$R_0 = \frac{\beta\pi}{d(d + e + r)}$$

be the basic reproduction number. Then the above result can be rewritten as

Theorem 4.1. *The system is stable at E_0 if $R_0 < 1$ and unstable for $R_0 > 1$, consequently, a transcritical bifurcation occurs at $R_0 = 1$.*

Remark Note that even if $R_0 < 1$, and the disease-free state is stable, it is still possible to have a feasible endemic steady state, provided that $R_0 > d^{-2}(\alpha + d)(d + \gamma)$, compare with (11) or (12). This situation gives rise to a backward bifurcation and is very well-known to possibly occur in the literature, [25].

At E^* the characteristic equation is given by:

$$D(\rho) = \rho^4 + \sigma_1\rho^3 + \sigma_2\rho^2 + \sigma_3\rho + \sigma_4 = 0, \tag{14}$$

where

$$\begin{aligned}
\sigma_1 &= -[V_{11}^* + V_{22}^* + V_{44}^*], \qquad (15)\\
\sigma_2 &= V_{11}^*V_{22}^* - V_{12}^*V_{21}^* - V_{13}^*V_{31}^* - V_{23}^*V_{32}^* + V_{11}^*V_{44}^* + V_{22}^*V_{44}^*,\\
\sigma_3 &= V_{12}^*V_{21}^*V_{44}^* - V_{11}^*V_{22}^*V_{44}^* + V_{13}^*V_{31}^*V_{44}^* + V_{23}^*V_{32}^*V_{44}^* - V_{14}^*V_{31}^*V_{43}^*\\
&\quad + V_{14}^*V_{32}^*V_{43}^* + V_{13}^*V_{22}^*V_{31}^* - V_{12}^*V_{23}^*V_{31}^* - V_{13}^*V_{21}^*V_{32}^* + V_{11}^*V_{23}^*V_{32}^*,\\
\sigma_4 &= V_{12}^*V_{14}^*V_{31}^*V_{43}^* + V_{14}^*V_{22}^*V_{31}^*V_{43}^* - V_{11}^*V_{14}^*V_{32}^*V_{43}^* - V_{14}^*V_{21}^*V_{32}^*V_{43}^*\\
&\quad - V_{13}^*V_{22}^*V_{31}^*V_{44}^* + V_{12}^*V_{23}^*V_{31}^*V_{44}^* + V_{13}^*V_{21}^*V_{32}^*V_{44}^* - V_{11}^*V_{23}^*V_{32}^*V_{44}^*,
\end{aligned}$$

with $V_{ik}^* = V_{ik}(E^*)$, $i,k = 1,\ldots,4$.

Here $\sigma_1 > 0$, so according to the Routh-Hurwitz criterion, all the eigenvalues of the Jacobian matrix at E^* are negative or have negative real part if:

$$\sigma_4 > 0, \quad \sigma_1\sigma_2 - \sigma_3 > 0, \quad (\sigma_1\sigma_2 - \sigma_3)\sigma_3 - \sigma_1^2\sigma_4 > 0. \qquad (16)$$

In summary we can state the following result.

Proposition 1. *The coexistence equilibrium point E^* is stable if the conditions (16) are satisfied.*

4.1.1. Hopf-Bifurcation Analysis

At E_0, in view of the strict inequality in the condition (13), the quadratic will not possess purely imaginary eigenvalues, hence there is no possibility of occurrence of Hopf-bifurcations at this equilibrium point. For the endemic equilibrium E^* we consider the Hopf-bifurcation as a function of the generic parameter $\kappa \in \mathbf{R}$.

Let $\Psi : (0,\infty) \to \mathbf{R}$ be the following continuously differentiable function of κ:

$$\Psi(\kappa) := \sigma_1(\kappa)\sigma_2(\kappa)\sigma_3(\kappa) - \sigma_3^2(\kappa) - \sigma_4(\kappa)\sigma_1^2(\kappa).$$

Then for the occurrence of a supercritical Hopf-bifurcation, there should exist a $\kappa^* \in (0,\infty)$ in the spectrum $\sigma(\kappa) = \{\rho : D(\rho) = 0\}$ of the characteristic equation, at which a pair of complex eigenvalues $\rho(\kappa^*), \bar{\rho}(\kappa^*) \in \sigma(\kappa)$ satisfy

$$\mathrm{Re}\rho(\kappa^*) = 0, \quad \mathrm{Im}\rho(\kappa^*) = \omega_0 > 0,$$

along with the transversality condition

$$\frac{dRe(\rho_j(\kappa))}{d\kappa}|_{\kappa=\kappa^*} \neq 0, \quad j = 1,\ 2. \qquad (17)$$

Furthermore, all other eigenvalues must have negative real parts.

Theorem 4.2. *The endemic equilibrium E^* of the system (2) undergoes a Hopf bifurcation at $\kappa = \kappa^* \in (0,\infty)$ if and only if*

$$\begin{aligned}
&\sigma_2(\kappa^*) > 0, \quad \sigma_3(\kappa^*) > 0, \quad \sigma_4(\kappa^*) > 0, \quad \sigma_1(\kappa^*)\sigma_2(\kappa^*) - \sigma_3(\kappa^*) > 0,\\
&\Psi(\kappa^*) = 0, \text{ and } \sigma_1^3\sigma_2'\sigma_3(\sigma_1 - 3\sigma_3) \neq (\sigma_2\sigma_1^2 - 2\sigma_3^2)(\sigma_3'\sigma_1^2 - \sigma_1'\sigma_3^2).
\end{aligned} \qquad (18)$$

Moreover, at $\kappa = \kappa^*$, the characteristic equation has a pair of purely imaginary eigenvalues, and the other two have negative real parts. Here, primes denote the differentiation with respect to κ.

Proof. By the condition $\Psi(\kappa^*) = 0$, the characteristic equation can be written as

$$\left(\rho^2 + \frac{\sigma_3}{\sigma_1}\right)\left(\rho^2 + \sigma_1\rho + \frac{\sigma_1\sigma_4}{\sigma_3}\right) = 0.$$

Let its four roots in the complex domain be denoted by ρ_i, (i=1,2,3,4) with the pair of purely imaginary roots at $\kappa = \kappa^*$ being $\rho_1 = \bar{\rho}_2$, then we have

$$\rho_3 + \rho_4 = -\sigma_1, \quad \omega_0^2 + \rho_3\rho_4 = \sigma_2, \quad \omega_0^2(\rho_3 + \rho_4) = -\sigma_3, \quad \omega_0^2\rho_3\rho_4 = \sigma_4, \quad (19)$$

where $\omega_0 = \mathrm{Im}\rho_1(\kappa^*)$. By dividing the third and the first equations in (19), we find $\omega_0 = \sqrt{\sigma_3\sigma_1^{-1}}$. Now, if ρ_3 and ρ_4 are complex conjugate, from (19), it follows that $2\mathrm{Re}\rho_3 = -\sigma_1$; if they are real roots, then by (14) and (19) $\rho_3 < 0$ and $\rho_4 < 0$. To complete the discussion, it remains to verify the transversality condition.

Now, we shall verify the transversality condition (17). Substituting $\rho_j(\kappa) = \chi(\kappa) \pm i\nu(\kappa)$, into (14) and differentiating, we have

$$K(\kappa)\chi'(\kappa) - L(\kappa)\nu'(\kappa) + M(\kappa) = 0, \quad L(\kappa)\chi'(\kappa) + K(\kappa)\nu'(\kappa) + N(\kappa) = 0, \quad (20)$$

where

$$\begin{aligned}
K(\kappa) &= 4\chi^3 - 12\chi\nu^2 + 3\sigma_1(\chi^2 - \nu^2) + 2\sigma_2\chi + \sigma_3, \\
L(\kappa) &= 12\chi^2\nu + 6\sigma_1\chi\nu - 4\chi^3 + 2\sigma_2\chi, \\
M(\kappa) &= \sigma_1\chi^3 - 3\sigma_1'\chi\nu^2 + \sigma_2'(\chi^2 - \nu^2) + \sigma_3'\chi, \\
N(\kappa) &= 3\sigma_1'\chi^2\nu - \sigma_1'\nu^3 + 2\sigma_2'\chi\nu + \sigma_3'\chi.
\end{aligned}$$

Solving (20) for $\chi'(\kappa^*)$ we have

$$\begin{aligned}
\left[\frac{dRe(\rho_j(\kappa))}{d\kappa}\right]_{\kappa=\kappa^*} &= \chi'(\kappa)_{\kappa=\kappa^*} = -\frac{L(\kappa^*)N(\kappa^*) + K(\kappa^*)M(\kappa^*)}{K^2(\kappa^*) + L^2(\kappa^*)} \\
&= \frac{\sigma_1^3\sigma_2'\sigma_3(\sigma_1 - 3\sigma_3) - 2(\sigma_2\sigma_1^2 - 2\sigma_3^2)(\sigma_3'\sigma_1^2 - \sigma_1'\sigma_3^2)}{\sigma_1^4(\sigma_1 - 3\sigma_3)^2 + 4(\sigma_2\sigma_1^2 - 2\sigma_3^2)^2} > 0
\end{aligned}$$

which holds in view of (18). Thus the transversality conditions holds and consequently a Hopf bifurcation occurs at $\kappa = \kappa^*$. □

4.2. Analysis of the system with delays

In this section, the local stability of the delayed system (2) is studied around the coexisting equilibrium point only. Without loss of generality we assume E^* to be the endemic equilibrium point of the system (2). The expressions of S_u^*, S_a^*, I^*, M^* have already been obtained in (6), (7). We are now interested

in the local asymptotic stability of the endemic steady state E^* for the delayed system.

Linearizing the system (2) about E^*, we get

$$\frac{dX}{dt} = JX(t) + GX(t-\tau_1) + HX(t-\tau_2), \tag{21}$$

where $J = [J_{ij}]$, $G = [G_{ij}]$, $H = [H_{ij}]$ are the following 4×4 matrices:

$$J = \begin{pmatrix} -\beta I^* - \alpha f(M^*) - d & \lambda g(M^*) & -\beta S_u^* + (1-p)r & \lambda g'(M^*) S_a^* \\ \alpha f(M^*) & J_{22} & pr - \gamma S_a^* & -\lambda g'(M^*) S_a^* \\ \beta I^* & \gamma I^* & J_{33} & 0 \\ 0 & 0 & 0 & -\theta \end{pmatrix},$$

with $J_{22} = -\lambda g(M^*) - d - \gamma I^*$, $\quad J_{33} = \beta S_u^* + \gamma S_a^* - (d+e+r) = 0$, from (1),

$$H = \begin{pmatrix} 0 & 0 & 0 & -\alpha f'(M^*) S_u^* \\ 0 & 0 & 0 & \alpha f'(M^*) S_u^* \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad G = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \eta e & 0 \end{pmatrix}.$$

The characteristic equation of system (2) is

$$\triangle(\xi) = \mid \xi I - J - e^{-\xi\tau_1} G - e^{-\xi\tau_2} H \mid = 0.$$

This equation can be written as

$$\psi(\xi, \tau_1, \tau_2) = \xi^4 + a_1\xi^3 + a_2\xi^2 + a_3\xi + a_4 + e^{-\xi(\tau_1+\tau_2)}[b_1\xi + b_2] = 0. \tag{22}$$

where

$$\begin{aligned} a_1 &= -[J_{11} + J_{22} + J_{44}], \\ a_2 &= J_{11}J_{22} - J_{12}J_{21} + J_{11}J_{44} + J_{22}J_{44} + J_{13}J_{31} - J_{32}J_{23}, \\ a_3 &= J_{31}J_{13}J_{44} - J_{11}J_{22}J_{44} + J_{44}J_{21}J_{12} \\ &\quad -J_{12}J_{31}J_{23} + J_{13}J_{31}J_{22} + J_{32}J_{23}J_{44}, \\ a_4 &= J_{44}[J_{31}(J_{12}J_{23} - J_{22}J_{13}) - J_{32}(J_{11}J_{23} - J_{21}J_{13})], \\ b_1 &= J_{32}H_{24}G_{43} + J_{31}G_{43}H_{14}, \\ b_2 &= J_{31}G_{43}(J_{22}H_{14} - J_{12}H_{24}) + J_{32}G_{43}(J_{11}H_{24} - J_{21}H_{14}), \end{aligned} \tag{23}$$

4.2.1. Delay length and Hopf bifurcations

The coexisting equilibrium point E^* will be LAS if all the roots of the corresponding characteristic equation (22) are negative or have negative real parts. The classical Routh-Hurwitz criterion cannot be used to investigate the stability of the system as the equation (22) is a transcendental equation in ξ. The following cases may arise.

Case I: $\tau_1 = 0, \tau_2 > 0$

The characteristic equation becomes

$$\psi(\xi, \tau) = \xi^4 + a_1\xi^3 + a_2\xi^2 + a_3\xi + a_4 + e^{-\xi\tau_2}[b_1\xi + b_2] = 0, \tag{24}$$

A necessary condition for stability changes of E^* is that the characteristic equation (24) has purely imaginary solutions. Let $i\theta$, $\theta \in \mathbf{R}$, be a root of equation (24). We then get

$$b_1 \sin\theta\tau_2 + b_2 \cos\theta\tau_2 = -\theta^4 + a_2\theta^2 - a_4 \tag{25}$$

$$b_1 \cos\theta\tau_2 - b_2 \sin\theta\tau_2 = a_1\theta^3 - a_3\theta \tag{26}$$

Squaring and adding the above two equations, and substituting $\theta^2 = l$ we obtain

$$l^4 + \omega_1 l^3 + \omega_2 l^2 + \omega_3 l + \omega_4 = 0. \tag{27}$$

Here

$$\omega_1 = {a_1}^2 - 2a_2, \quad \omega_2 = a_2^2 + 2a_4 - 2a_1a_3,$$
$$\omega_3 = -2a_2a_4 + a_3^2 - b_1^2, \omega_4 = a_4^2 - (b_1^2 + b_2^2).$$

The roots of equation (27) have negative real parts if and only if the Routh-Hurwitz criterion is satisfied. In such case (24) does not have purely imaginary roots. Thus, we summarize the results in the following proposition.

Proposition 2. *Suppose that the system without delay is stable. The endemic equilibrium E^* is LAS for all $\tau_2 > 0$ if the following conditions are satisfied:*

$$\omega_1 > 0, \quad \omega_4 > 0, \quad \omega_1\omega_2 - \omega_3 > 0, \quad (\omega_1\omega_2 - \omega_3)\omega_3 - \omega_1^2\omega_4 > 0.$$

If $\omega_4 < 0$ holds then equation (27) will admit at least one positive root. If θ_0^2 is the minimum positive root of (27), then θ will be a purely imaginary root, $\pm i\theta_0$ corresponding to the delay τ_2. By Butler's lemma, [26], the endemic equilibrium E^* remains stable for $\tau_2 < \tau_2^*$. We now evaluate the critical value of τ_2 for which the delayed system (22) remains stable.

From equation (25),

$$\tau_2^* = \frac{1}{\theta_0} cos^{-1}\left[\frac{b_2(-\theta_0^4 + a_2\theta_0^2 - a_4) + b_1a_1\theta_0^3}{b_1^2 + b_2^2}\right] + \frac{2\pi n}{\theta_0}, \quad n = 0, 1, 2, 3, \ldots$$

From the above analysis the following theorem follows.

Theorem 4.3. *If $\omega_4 < 0$ is satisfied then the steady state E^* is LAS for $\tau_2 < \tau_2^*$ and becomes unstable for $\tau_2 > \tau_2^*$. Furthermore, the system will undergo a Hopf-bifurcation at E^* when $\tau_2 = \tau_2^*$ provided $4\theta_0^6 + A_1\theta_0^4 + A_2\theta_0^2 + A_3 > 0$, where*

$$A_1 = 3a_1 - 6a_2, \quad A_2 = 2a_2 + 4a_4 - 4a_1a_3, \quad A_3 = a_3^2 - 2a_2a_4 - b_1^2.$$

Proof. We need to prove the last conditions only. Now, differentiating (24) with respect to τ_2 we get:

$$\frac{d\tau_2}{d\xi} = \frac{4\xi^3 + 3a_1\xi^2 + 2a_2\xi + a_3}{b_1\xi^2 + b_2} e^{\xi\tau_2} + \frac{b_1}{b_1\xi^2 + b_2\xi} - \frac{\tau_2}{\xi}.$$

Now, using the relation (25) one can obtain:

$$\begin{aligned} sgn\left[\frac{d(Re\xi)}{d\tau_2}\right]_{\tau_2=\tau_2^*} &= sgn\left[Re\left(\frac{d\xi}{d\tau_2}\right)^{-1}\right]_{\xi=i\theta_0}, \\ &= sgn\left[\frac{4\theta_0^6 + A_1\theta_0^4 + A_2\theta_0^2 + A_3}{b_1\theta_0^2 + b_2^2}\right] \end{aligned} \tag{28}$$

and the latter is positive if $4\theta_0^6 + A_1\theta_0^4 + A_2\theta_0^2 + A_3 > 0$ i.e. the transversality condition holds and the system undergoes Hopf bifurcation at $\tau_2 = \tau_2^*$.

□

Case II: When $\tau_1 > 0, \tau_2 = 0$
The analysis is similar to **Case I** and is therefore given without proof by the following theorem.

Theorem 4.4. *If the system without delay is asymptotically stable, then the steady state E^* is LAS for $\tau_1 < \tau_1^*$ and becomes unstable for $\tau_1 > \tau_1^*$. Furthermore, the system will undergo a Hopf-bifurcation at E^* when $\tau_1 = \tau_1^*$ provided*

$$\left[\frac{d(Re\xi)}{d\tau_1}\right]_{\tau_1=\tau_1^*} > 0.$$

Case III: When $\tau_1 > 0, \tau_2 > 0$
This analysis is complicated and it is difficult to obtain information on the nature of the eigenvalues and the conditions for occurrence of stability switches. But we can investigate the nature of the eigenvalues at the endemic state with the traceDDE package in Matlab which allows us to find the characteristic roots and region of stability [27]. We provide the result without proof in the following theorem.

Theorem 4.5. *Suppose that the non-delayed system is asymptotically stable. Now, if $\omega_4 < 0$ holds then there exists a τ^* such that the steady state E^* is LAS for $\tau_1 + \tau_2 < \tau^*$, and becomes unstable for $\tau_1 + \tau_2 > \tau^*$. Furthermore, E^* will undergo a Hopf-bifurcation when $\tau_1 + \tau_2 = \tau^*$, provided that*

$$\left[\frac{d(Re\xi)}{d\tau}\right]_{\tau=\tau^*} > 0.$$

Table 1: List of parameters used in numerical simulations and their references.

Parameter	Definition	Value	Unit
π	Constant recruitment rate	30	person day^{-1}
β	Disease transmission rate	0.00025	day^{-1}
α	Maximum rate of awareness from unaware to aware human	0.02	day^{-1}
λ	Maximum rate of transfer rate of people from aware to unaware	0.0002	day^{-1}
d	Susceptible class natural death rate	0.005	day^{-1}
e	Additional death rate due to infection	0.02	day^{-1}
η	Proportionality constant	0.02	–
γ	Contacts among infected and from the aware class	0.000002	day^{-1}
θ	Depletion rate of awareness program due to ineffectiveness	0.05	day^{-1}

5. Numerical Simulations

In this section, numerical simulations are performed to investigate the dynamics of the system and to support the findings of the theoretical findings. To carry out the numerical simulations on the epidemic models we need to make a specific choice for the function f, here taken as $f(M) = M(1 + M)^{-1}$.

The dynamic behavior of the system has been observed for different values of the two delays τ_1 and τ_2, in order also to assess its global dynamic behavior. Numerical simulations are performed to examine the disease spread at first with no time delay and subsequently with time delays. The parameters values used are obtained from [12, 17, 28, 29].

5.1. Numerical simulations of un-delayed system

Figure 1 to Figure 5 contain the numerical results of the un-delayed system. The set of parameters are given in Table 1. The time series solution of the system without delay is plotted in Figure 1 with the parameters values as given in Table 1.

Here, $R_0 > 1$ and the conditions given in (15) are satisfied, thus the endemic equilibrium is LAS. The phase portrait of the infective population, the unaware population and the cumulative density of awareness programs, not shown, have the property that all the trajectories initiating inside the region of attraction approach the equilibrium values. This indicates the nonlinear stability of the coexistence equilibrium (M^*, S_u^*, I^*) in the $M - S_u - I$ population subspace.

The bifurcation diagram of the system without delay is shown in Figure 2 as function of the parameter η. For lower values of η the system is stable, but above the threshold value η^* the system loses its stability and periodic solutions arise

through Hopf-bifurcation. The system with no delay shows periodic oscillations for $\eta = 0.1$ in the plane (see Figure 3) where other parameter values are the same as in Figure 1. But this oscillation depends on the immigration rate π and on the disease contact rate β.

In Figure 4, the stability region of E^* in $\eta - \beta$ and $\eta - \pi$ parameter spaces is shown. From Figure 4(a), if β increases then the critical value of η^* increases and from Figure 4(b), the periodic oscillation will disappear for some higher values of immigration rate, π.

5.2. Numerical simulations of delayed system

For the system with time delays, steady state E^* is asymptotically stable for $\tau_1 = 15, \tau_2 = 0$ (Figure 5). This indicates that sometimes the number of infective will be high and sometimes low and it may be difficult to make the forecasts regarding the size of epidemic. The system takes more time to settle to a stable state than the system with no delay.

In Figure 6, the trajectories of the system populations are plotted. Periodic sustained oscillations are observed for $\tau_1 = 75$. Figure 7 shows that the critical value of delay τ_1 depends on η. Similarly periodic oscillations for the delay $\tau_2 = 78$ can be observed (not shown). Figure 7 shows the that critical value of delay τ_1 i.e. τ_1^* depends on η (shown in Figure 7(a)) and that of τ_2 depends on α (shown in Figure 7(b)). In Figure 8, stability of E^* of the multi-delayed system in the $\tau_1 - \tau_2$ plane is shown. The critical value τ_1^* is clearly seen to depend on τ_2 and vice versa. In Figure 9, for the pair $\tau_1 = 30, \tau_2 = 48$ periodic solutions are observed. In Figure 10, using traceDDE, we plot the roots of characteristic equation (22). A pair of purely imaginary roots observed for $\tau = 30, \tau_2 = 48$.

6. Discussion and Conclusion

Media are widely acknowledged as a key tool for influencing people behavior towards the disease to devise proper policies for controlling the epidemic. Awareness programs through media make people aware about the disease and instruct them on how to take various precautions (e.g. taking preventive medicine, vaccination, social distancing etc.), to reduce their chances of being infected. Awareness among the human population thus may profoundly influence the pattern of disease spread and more importantly it helps in reducing the rate of infection.

In this paper, the effect of awareness programs and time delays on the disease dynamics of infectious diseases are considered. The main assumption regards the awareness or unawareness rate, which is taken as a function of media campaign. The existence and stability criteria of the disease-free and of the endemic equilibria have been derived in terms of the basic reproduction number, R_0. When R_0 is less than unity, disease remains endemic in the system, whereas for R_0 above unity, the disease cannot persist in the system and is therefore eradicated. Furthermore this indicates a transcritical bifurcation occurrence at $R_0 = 1$.

Our study suggests that if we increase the rate of implementation of awareness program through the media, the number of infected individuals decline and

the system remains stable. This occurs up to a threshold value of the awareness program implementation. Above that threshold the system becomes unstable and triggers the onset of persistent oscillations. The danger of the latter is well-known in ecology, as while the populations are at the bottom of the troughs, erratic perturbations of the environmental conditions, through climatic changes for instance, may push some of these populations to vanish, with consequent relevant effects on the whole ecosystem. Similar considerations hold for epidemic issues as those considered in the present case. The constant immigration rate may be one of the possible causes of such outcomes. In particular, for a moderate range of value of immigration rate the system shows unstable dynamics, while for lower and higher values the system settles to a stable behavior.

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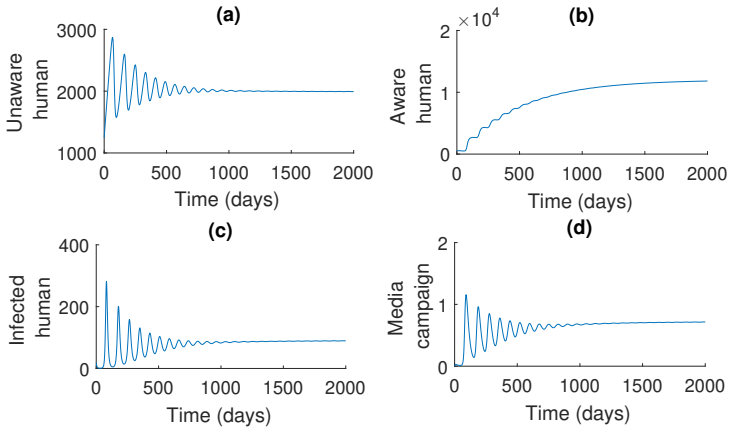

Figure 1: The time series solution of the system with no delay is plotted using the parameter given in Table 1. The system is stable.

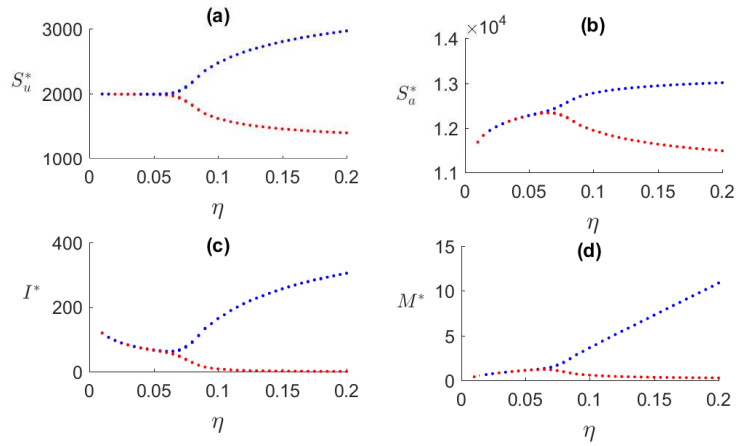

Figure 2: The system populations oscillations are plotted as function of η, with $R_0 > 1$ and $\beta = 0.00025$. Here, steady state values of all populations are plotted together with the minimum/maximum of the periodic solution when it exists.

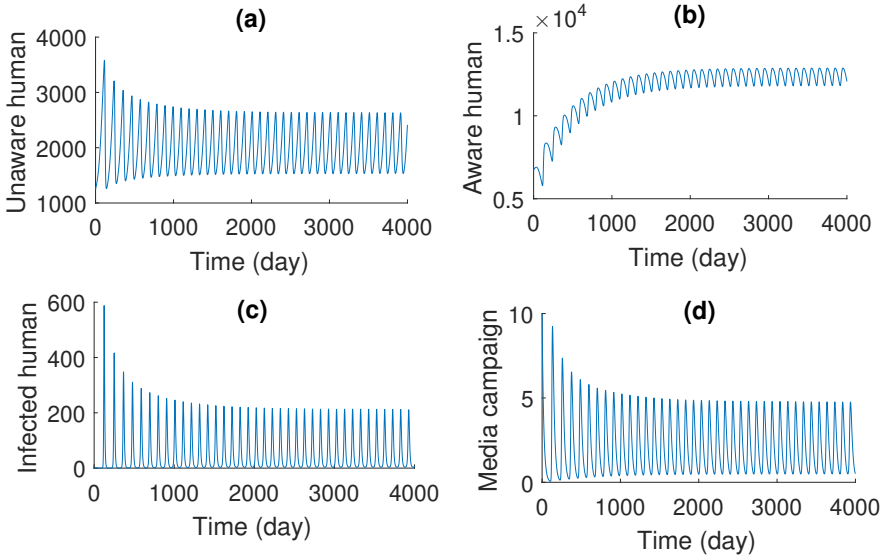

Figure 3: The system populations are plotted taking $\eta = 0.1 > \eta^*$, other parameters as in Table 1.

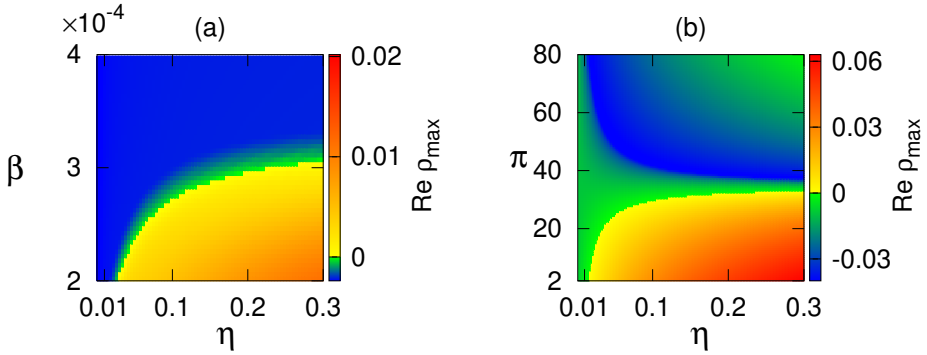

Figure 4: The stability region is shown (a) in $\eta - \beta$ plane, (b)in $\eta - \pi$ plane. The colour code denotes max$[Re(\rho)]$ whenever the endemic steady state is feasible. Other parameter values are as in Table 1. Here, recall that ρ denotes the characteristic root of equation (13).

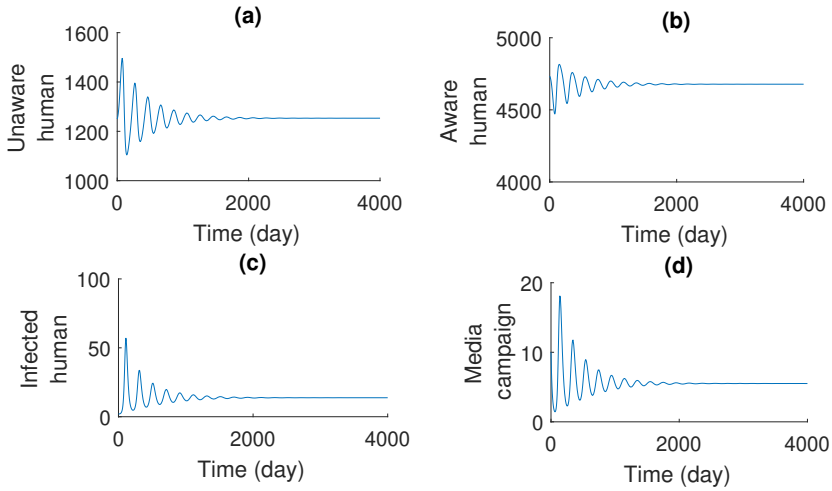

Figure 5: The time series solution of the system for $\tau_1 = 15$ is plotted and the endemic state is LAS.

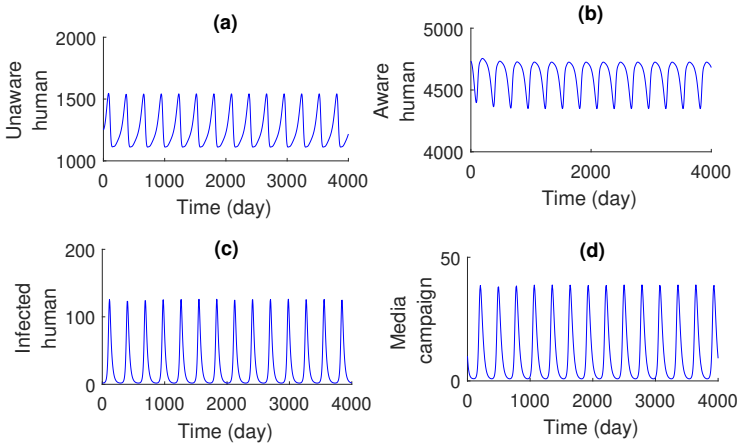

Figure 6: The time series solution of the system for $\tau_1 = 75$ is plotted using the parameter as given in Table 1 and periodic solution is observed.

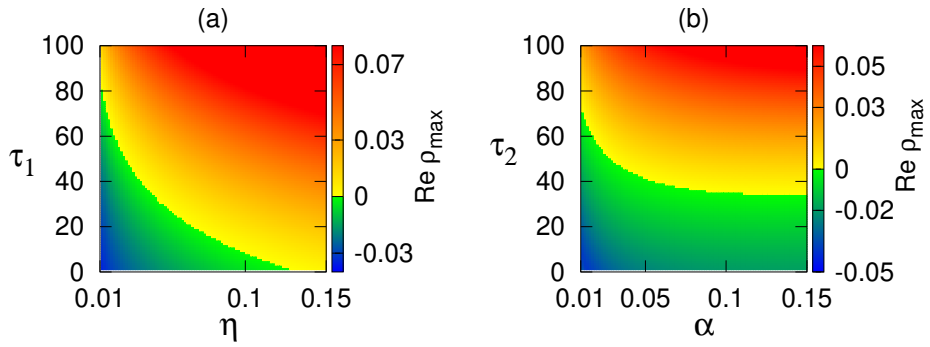

Figure 7: The stability region is shown: (a) in $\tau_1 - \eta$ plane taking, (b) in $\alpha - \tau_2$ plane, whenever the endemic steady state is feasible, other parameter values are as in Table 1. τ_1 The colour code denotes max$[Re(\rho)]$ whenever the endemic steady state is feasible.

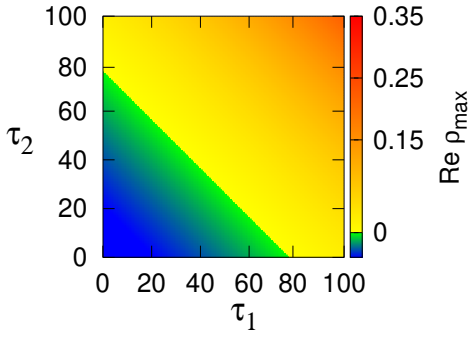

Figure 8: The stability region is shown in $\tau_1 - \tau_2$ plane, whenever the endemic steady state is feasible, other parameter values are as in Table 1. The colour code denotes max$[Re(\rho)]$ whenever the endemic steady state is feasible.

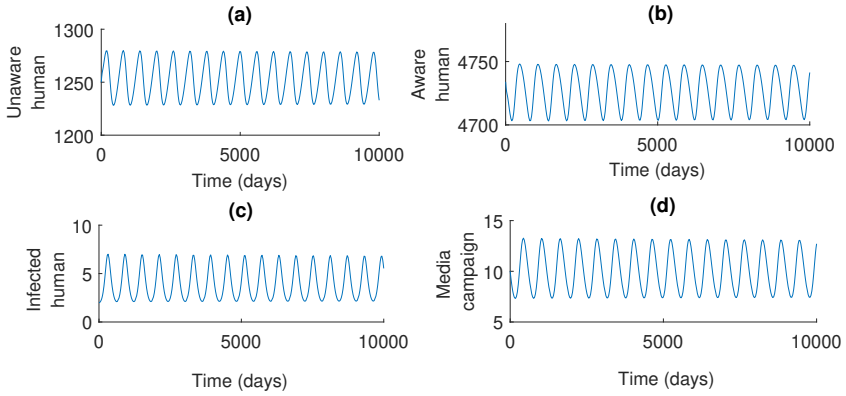

Figure 9: The time series solution of the system for $\tau_1 = 30, \tau_2 = 48$ is plotted and periodic solution is observed.

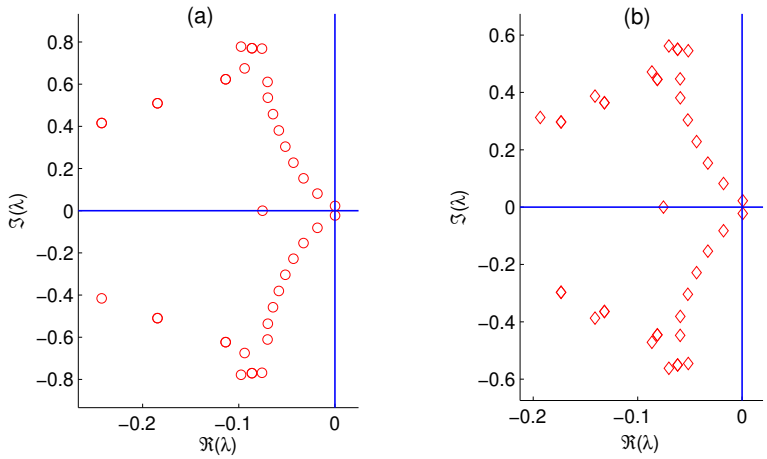

Figure 10: The characteristic roots are plotted: (a) for $\tau_1 = 30, \tau_2 = 48$ and (b) for $\tau_1 = 10, \tau_2 = 70$.