Research Article

The Impact of Awareness Programs with Recruitment and Delay on the Spread of an Epidemic

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A compartment epidemic model with delay is given to discuss the impact of awareness programs on the spread and control of infectious diseases in a given region. It is assumed that there is a constant recruitment rate in the cumulative density of awareness programs, and further it is assumed that awareness programs can influence the susceptible to a limited extent. The system exhibits two equilibria: the disease-free equilibrium is stable if the basic reproduction number is less than unity for any delay and the unique endemic equilibrium exhibits Hopf-bifurcation under certain conditions. Numerical simulations prove the results of analysis and the significance of awareness programs in preventing and controlling the diseases, by investigating the relationship between the proportion of the infective and the dissemination rate and the implementation rate, respectively.

1. Introduction

Plenty of evidence shows that awareness programs, which can influence the susceptible to a limited extent due to some objective factors, play an important role in the spread and control of infectious diseases. For example, during the outbreak of SARS, H1N1 influenza pandemic, and HIV epidemic, public media had massive reports on the number of the infections and deaths per day, which had a great impact on the diseases control [1–4]. That is due to the fact that the spread of diseases is often accompanied by a rise in awareness of those in the social vicinity of infected individuals and a subsequent change in behavior, such as keeping social distancing, wearing protective masks, and vaccination [5, 6]. Such reactions can manifest themselves in lower susceptibility as people try to prevent themselves from catching the disease, but also in lower infectivity because of self-imposed quarantine or better hygiene, shorter durations of infectiousness, or longer immunity [7]. And once the infective are cured, they will be aware of the disease [8].

Recently some scholars used mathematical models to discuss the impact of awareness programs on the diseases spreading and controlling in a given region [6, 8–11]. Joshi et al. [9] formulated a model to investigate the effect on the HIV epidemic in Uganda and compared their model with three types of the susceptible to a standard SIR model and then pointed out that the awareness programs in Uganda are successful in combating diseases. Li and Cui [1] analyzed a SIS epidemic model incorporating media coverage under constant and pulse vaccination; then they obtained the exact periodic infection-free solution which is globally asymptotically stable under some conditions. Numerical simulations prove the results of analysis and the significance of awareness programs in preventing and controlling the diseases, by investigating the relationship between the proportion of the infective and the dissemination rate and the implementation rate, respectively.
focused their attention at the contact rate, and most of them assumed that awareness programs will aid in modifying the contact rate between the susceptible and the infective [17–29]. Liu and Cui [17] used the contact rate (β1 − β2I/(m + I)) and found that both of the two equilibria are asymptotically stable. Mirsa et al. [20] used the constant k with the contact rate M/(k + M) to limit the effect of awareness programs on the susceptible and sought out the conditions that Hopf-bifurcation occurs. Tchuenche and Bauch [23] used an exponentially decreasing function e−M(t) to affect the force of infection; then numerical results showed the potential short-term beneficial effect of awareness programs. Pang and Cui [26] used β(I) = μ1 − μ2f(I) to show the contact rate after awareness programs alerts and found that though it is not a determined fact to eradicate the infection of the diseases, the effective awareness programs can postpone the arrival of the infection peak. Elenbaas et al. [28] introduced a segmented function to describe the media impact 𝑒−𝑀𝑡, when they formulate an epidemic model. A Filippov epidemic model was proposed to describe the real characteristics of media impact on the spread of infectious diseases by incorporating a piecewise continuous transmission rate βe−αtSI in [29]. Mathematical and bifurcation analyses with regard to the local and global stability of equilibria and local sliding bifurcations are performed.

But what we regard as unreasonable is that most of the articles assume that the cured infective become unaware of the disease. In fact people have a certain consciousness about the disease once they get sick. Therefore we propose a delayed mathematical model for predicting the future course of any epidemic by considering some of the infect join the aware susceptible class whereas the remaining fraction of recovered people will become aware and join the aware susceptible class whereas the remaining fraction of the unaware susceptible with the infective and then natural death rate. All the constants in the system are assumed to be positive.

Consider that the cumulative density of awareness programs driven by media in that region at time t is M(t), which is related to the infective. We make the constant μ represent the executed rate of awareness programs. As the time passes, some campaigns lose their impact on people and lead to the diminution of the awareness programs, so we introduce μ0 to denote the rate of their depletion. Moreover m0 represents the density level of media coverage on the disease from other regions. Using the fact that there is a limited extent of the awareness programs influence on the susceptible due to some objective factors, we introduce k to limit the effect and consider the interaction between them as Holling type-II functional response. It is plausible that the policy makers need some time to gather the cases of the infective generally; then we introduce τ and consider that the cumulative density of awareness programs at time t being executed will be in accordance with the infected cases reported at time t−τ (τ > 0). Keeping the above facts in mind, the dynamics of model is governed by the following system of nonlinear delay differential equations:

\[
X'(t) = b - \beta X(t) Y(t) + \lambda_0 X_m(t) - \lambda X(t) X(t) M(t) + p \nu Y(t) - d X(t),
\]

\[
X_m'(t) = \lambda X(t) X(t) M(t) - \lambda_0 X_m(t) + q \nu Y(t) - d X_m(t),
\]

\[
Y'(t) = \beta X(t) Y(t) - \nu Y(t) - d Y(t),
\]

\[
M'(t) = m_0 + \mu Y(t - \tau) - \mu_0 M(t).
\]

Here \(X(0) = X_0 > 0, X_m(0) = X_{m0} \geq 0, \) and \(Y(\theta) = Y_0 \geq 0\) for \(\theta \in [-\tau, 0]\) and \(M(0) = M_0 \geq 0\).

In the above model, the constants \(\beta\) and \(d(d = b)\), respectively, represent the contact rate of the unaware susceptible with the infective and the natural death rate. All the constants in the system are assumed to be positive.

2. Mathematical Model and Equilibrium Analysis

In the region under consideration the rate of immigration of the susceptible is b. It is assumed that the disease spreads due to the direct contact between the susceptible and the infective only and due to awareness programs the susceptible avoid being in contact with the infective and form a different class with a proportion \(\lambda\), named the aware susceptible. So the total population is divided into three classes: the susceptible, the aware susceptible, and the infective, the proportions of which at time \(t\) in the total population are \(X(t), X_m(t),\) and \(Y(t)\). It is assumed that the aware susceptible may lose awareness with passage of time and become susceptible with the proportion of \(\lambda_0\) again. The infective can be cured with a proportion \(\nu\) and a fraction \(q\) of recovered people will become aware and join the aware susceptible class whereas the remaining fraction \(p(p + q = 1)\) will join the susceptible.
Using the fact that $X(t) + Y(t) + X_m(t) = 1$, $p + q = 1$

system (1) is reduced to the following system:

\[
X_m'(t) = \lambda \left( 1 - X_m(t) - Y(t) \right) \frac{M(t)}{1 + kM(t)} - (\lambda_0 + d) X_m(t) + qY(t),
\]

\[
Y'(t) = \beta \left( 1 - X_m(t) - Y(t) \right) Y(t) - (v + d) Y(t),
\]

\[
M'(t) = m_0 + \mu Y(t - \tau) - \mu_0 M(t).
\]

Now it is sufficient to study system (2) in detail rather than system (1).

For the analysis of system (2), we need the region of attraction which is given by the set

\[
\Omega = \{ (X_m, Y, M) \in \mathbb{R}^3_+ : 0 \leq X_m, Y \leq 1, 0 \leq M \leq (m_0 + \mu)/\mu_0 \},
\]

and it attracts all solutions initiating in the interior of the positive orthant.

The system (2) has two equilibria.

(i) Disease-free equilibrium $E_0(\lambda m_0/((\lambda_0 + d)(\mu_0 + k m_0)), 0, m_0/\mu_0)$.

(ii) Endemic equilibrium $E_*(X^*_m, Y^*, M^*)$.

Define the basic reproduction number $R_0 = \beta/(v + d)$. The existence of equilibrium $E_0$ is trivial; then we prove the existence of $E_*$ in detail. When $R_0 > 1$, in the equilibrium $E_*$ the values of $X^*_m, Y^*$, and $M^*$ are

\[
X^*_m = 1 - \frac{v + d}{\beta} - Y^*,
\]

\[
Y^* = \frac{\mu_0}{\mu_0} + \frac{\mu}{\mu_0} Y^*,
\]

with $Y^*$ satisfying the equation

\[
A_1 Y^{*2} + A_2 Y^* + A_3 = 0,
\]

where

\[
A_1 = \mu k \beta (\lambda_0 + d + q v),
\]

\[
A_2 = \mu k (\lambda_0 + d + q v) + \lambda \mu (v + d)
\]

\[
+ \beta (\lambda_0 + d + q v)(\mu_0 + k m_0),
\]

\[
A_3 = \lambda m_0 (v + d) + (\lambda_0 + d)(v + d - \beta)(\mu_0 + k m_0).
\]

Solving (4) we get

\[
Y^* = \frac{-A_2 \pm \sqrt{A_2^2 - 4 A_1 A_3}}{2A_1}.
\]

We obtain $A_1 > 0$, and when $\beta \lambda < k(\lambda_0 + d)(\beta - v - d)$,

\[
A_3 = \lambda m_0 (v + d) + (\lambda_0 + d)(v + d - \beta)(\mu_0 + k m_0)
\]

\[
= \beta \lambda m_0 \left( \frac{1}{R_0} - 1 \right) + \beta \lambda m_0
\]

\[
+ \beta (\lambda_0 + d)(\mu_0 + k m_0) \left( \frac{1}{R_0} - 1 \right)
\]

\[
= \beta \lambda m_0 \left( \frac{1}{R_0} - 1 \right) + \beta \mu_0 (\lambda_0 + d) \left( \frac{1}{R_0} - 1 \right)
\]

\[
+ \beta m_0 \left[ \lambda - k(\lambda_0 + d) \left( 1 - \frac{1}{R_0} \right) \right] < 0,
\]

then we get $Y^* = (-A_2 + \sqrt{A_2^2 - 4 A_1 A_3})/2A_1$ for $Y^* > 0$.

Remark 1. From the expression of $Y^*$, it is easy to note that $dY^*/d\lambda < 0$ and $dY^*/d\mu < 0$, which shows that the equilibrium proportion of the infective decreases as the rate of dissemination of awareness among the susceptible and the implementation rate of awareness programs increases.

### 3. Stability Analysis

In this section we present the local stability of $E_0, E_*$ and explore the conditions of Hopf-bifurcation by taking delay $\tau$ as a bifurcation parameter.

#### 3.1. Stability of Equilibria without Delay ($\tau = 0$)

**Theorem 2.** The disease-free equilibrium $E_0$ is locally asymptotically stable if $R_0 < 1$.

**Proof.** The Jacobian matrix corresponding to the system (2) when $\tau = 0$ is given below:

\[
J = \begin{bmatrix}
-\lambda M - \lambda_0 - d & \lambda M + v q & \lambda (1 - Y - X_m) - 1 \\
-\beta Y (1 - X_m) - (v + d) - 2 \beta Y & \mu & 0 \\
0 & -\mu_0 & -\mu_0
\end{bmatrix}.
\]

Using the fact that $X(t) + Y(t) + X_m(t) = 1$, $p + q = 1$

system (1) is reduced to the following system:
The characteristic equation at $E_0$ is of the form
\[ (\eta + \mu_0) \left[ \eta + \frac{\lambda m_0}{km_0 + \mu_0} + \lambda_0 + d \right] \]
\[ \times \left[ \eta + \frac{\beta\lambda m_0}{\lambda m_0 + (\lambda_0 + d)(\mu_0 + km_0)} + \beta \left( \frac{1}{R_0} - 1 \right) \right] = 0, \]
where $\eta$ is the eigenvalue. We get
\[ \eta_1 = -\mu_0 < 0, \]
\[ \eta_2 = -\left( \frac{\lambda m_0}{km_0 + \mu_0} + \lambda_0 + d \right) < 0, \]
\[ \eta_3 = -\beta \left[ \frac{\lambda m_0}{\lambda m_0 + (\lambda_0 + d)(\mu_0 + km_0)} + \left( \frac{1}{R_0} - 1 \right) \right], \]
so $\eta_3 < 0$ when $R_0 < 1$. Thus $E_0$ is locally asymptotically stable if $R_0 < 1$. \(\square\)

The form of characteristic equation at $E_*$ is
\[ \eta^3 + Q_1 \eta^2 + Q_2 \eta + (Q_3 + Q_4) = 0, \]
where
\[ Q_1 = B_1 + B_3 + \mu_0, \]
\[ Q_2 = \mu_0 (B_1 + B_3) + B_1 B_3 - \beta Y^* B_2, \]
\[ Q_3 = \mu_0 B_1 B_3 - \beta \mu_0 Y^* B_2, \]
\[ Q_4 = -\beta \mu_0 Y^* B_4, \]
\[ B_1 = \lambda M^* \frac{1}{1 + kM^*} + \lambda_0 + d, \]
\[ B_2 = \lambda M^* \frac{1}{1 + kM^*} - \gamma q, \]
\[ B_3 = \beta \left( X_m^* - 1 \right) + 2\beta Y^* + (\nu + d), \]
\[ B_4 = \lambda \left( X_m^* + Y^* - 1 \right) \frac{1}{(1 + kM^*)}. \]

Theorem 3. When the endemic equilibrium $E_*$ exists, it is locally asymptotically stable provided $m_0(\lambda_0 + d + q\nu) > \mu_0$.

Proof. For the characteristic equation
\[ \eta^3 + Q_1 \eta^2 + Q_2 \eta + (Q_3 + Q_4) = 0, \]
it is easy to show
\[ Q_1 = \lambda M^* \frac{1}{1 + kM^*} + \lambda_0 + d \]
\[ + \beta \left( X_m^* - 1 \right) + 2\beta Y^* + (\nu + d) + \mu_0 \]
\[ = \lambda M^* \frac{1}{1 + kM^*} + \lambda_0 + d + \beta \left( X_m^* - 1 \right) \]
\[ + 2\beta Y^* + \beta \left( 1 - X_m^* - Y^* \right) + \mu_0 \]
\[ = \lambda M^* \frac{1}{1 + kM^*} + \lambda_0 + d + \beta Y^* + \mu_0 > 0, \]
\[ Q_2 = \mu_0 (B_1 + B_3) + \left( \lambda M^* \frac{1}{1 + kM^*} + \lambda_0 + d \right) \]
\[ \times \left[ \beta \left( X_m^* - 1 \right) + 2\beta Y^* + (\nu + d) \right] \]
\[ - \beta Y^* \left( \lambda M^* \frac{1}{1 + kM^*} - \gamma q \right) \]
\[ = \mu_0 (B_1 + B_3) + (\lambda_0 + \gamma q + d) \beta Y^* > 0, \]
\[ Q_3 + Q_4 = \mu_0 B_1 B_3 - \beta Y^* \left( \mu B_1 + \mu_0 B_2 \right) \]
\[ = \mu_0 \beta Y^* \left( \lambda M^* \frac{1}{1 + kM^*} + \lambda_0 + d \right) \]
\[ - \beta Y^* \left[ \mu \left( X_m^* + Y^* - 1 \right) \frac{1}{(1 + kM^*)^2} \right. \]
\[ + \mu_0 \left( \lambda M^* \frac{1}{1 + kM^*} - \gamma q \right) \]
\[ = \beta Y^* \mu \left( 1 - X_m^* - Y^* \right) \frac{1}{(1 + kM^*)^2} \]
\[ + \mu_0 \beta Y^* \left( \lambda_0 + d \right) + \gamma q \mu_0 \beta Y^* > 0. \]
And
\[ Q_1 Q_2 - (Q_3 + Q_4) \]
\[ = (B_1 + B_3) (\mu_0 B_1 + \mu_0 B_3 + B_1 B_3 - \beta Y^* B_2) \]
\[ + \mu_0^2 (B_1 + B_3) - Q_4 \]
\[ \nu \mu _0 (B_1 + B_3) + B_3 [(B_1 + B_3)(B_1 - B_2) + \mu B_4], \]

(15)

where

\[ (B_1 + B_3)(B_1 - B_2) + \mu B_4 \]

\[ = \left( \frac{\lambda M^*}{1 + k M^*} + \lambda_0 + d + \beta Y^* \right) \left( \lambda_0 + d + qv \right) \]

and substituting \( \nu = \omega > 0 \) into (19) and separating real and imaginary parts, we get the following transcendental equations:

\[ Q_2 \omega - \omega^3 = Q_4 \sin (\omega r), \]

(20)

\[ Q_4 \omega^2 - Q_3 = Q_4 \cos (\omega r). \]

Squaring and adding the above equations and substituting \( \psi = \omega^2 \), we get

\[ h(\psi) = \psi^3 + P_1 \psi^2 + P_2 \psi + P_3 = 0, \]

(21)

where \( P_1 = Q_1^2 - 2Q_2, P_2 = Q_2^2 - 2Q_1Q_3, \) and \( P_3 = Q_3^2 - Q_4^2 \).
If the coefficients in \( h(\psi) \) satisfy the conditions of Routh-Hurwitz criterion, then \( E_+ \) is locally asymptotically stable for all delays \( \tau \) \( > 0 \), provided it is stable in absence of delay. In the following we consider that the values of \( P_1 \) (i = 1, 2, 3, 4) do not satisfy the Routh-Hurwitz criterion.

**Lemma 5.** If \( B_3 > 0 \), \( \lambda_0 + d + q \psi < \mu \lambda (\psi + d) (1/\beta(1 + k M^*)^2) \), (21) will have at least one positive root \( \psi_0 \).

**Proof.** For \( h(\psi) = \psi^3 + P_1 \psi^2 + P_2 \psi + P_3 \), it is obvious that

\[
P_1 = (B_1 + B_3 + \mu_0)^2 - 2 [(B_1 + B_3) \mu_0 + B_1 B_3 - \beta B_2 Y^*],
\]

\[
P_2 = [(B_1 + B_3) \mu_0 + B_1 B_3 - \beta B_2 Y^*]^2 - 2 (B_1 + B_3, \mu_0) (B_1 B_3 \mu_0 - \mu_0 \beta B_2 Y^*)
\]

\[
P_3 = (Q_3 + Q_4) (Q_3 - Q_4)
\]

\[
= B_3 (\mu_0 B_1 - \mu_0 B_2 + \mu B_4) (Q_3 + Q_4)
\]

\[
= B_3 (Q_3 + Q_4) \times \left[ \mu_0 (\lambda_0 + d + q \psi)
- \mu \lambda (1 - X_m^* - Y^*) \frac{1}{(1 + k M^*)^2} \right]
\]

\[
< B_3 (Q_3 + Q_4) \left[ (\lambda_0 + d + q \psi)
- \mu \lambda (\psi + d) \frac{1}{\beta(1 + k M^*)^2} \right] < 0.
\]

Then we have \( h(0) < 0 \) and \( h(\infty) \to +\infty \); thus (21) has at least one positive root \( \psi_0 \). □

Denote \( \omega_0 = \sqrt{\psi_0} \); then (21) has a pair of purely imaginary roots \( (\pm i \omega_0) \).

Now we turn to the bifurcation analysis. We use the delay \( \tau \) as the bifurcation parameter. We view the solutions of (21) as a function of the bifurcation parameter \( \tau \), and let \( \eta(\tau) = \gamma(\tau) + i \omega(\tau) \) be the eigenvalue of (21) such that, for the initial value of the bifurcation parameter \( \tau_0 \), we have \( \gamma(\tau_0) = 0 \) and \( \omega(\tau_0) = \omega_0(\omega_0 > 0) \). To establish the Hopf bifurcation at \( \tau = \tau_0 \), we need to show that \( \frac{d \Re \eta(\tau)}{d \tau}|_{\tau=\tau_0} > 0 \).

**Lemma 6.** One has the following transversality condition:

\[
\left. \frac{d \Re \eta(\tau)}{d \tau} \right|_{\tau=\tau_0} > 0.
\]  

**Proof.** Differentiating with respect to \( \tau \) from (21), we get

\[
\left( \frac{d \eta}{d \tau} \right)^{-1} = \frac{3 \eta^2 + 2 Q_1 \eta + Q_2 - Q_4 \Re e^{-\eta \tau}}{Q_4 \eta e^{-\eta \tau}}.
\]

So

\[
\text{Sign} \left\{ \frac{d \eta}{d \tau} \right\}_\tau = \tau_0
\]

\[
= \text{Sign} \left\{ \frac{d \eta}{d \tau} \right\}_\eta = \omega_0
\]

\[
= \text{Sign} \left\{ \frac{d \eta}{d \eta} \right\}_\eta = \omega_0
\]

\[
= \text{Sign} \left\{ 3 \eta^2 + 2 Q_1 \eta + Q_2 - Q_4 \Re e^{-\eta \tau} \right\}_\eta = \omega_0
\]

\[
= \text{Sign} \left\{ 3 \eta^2 + 2 Q_1 \eta + Q_2 - Q_4 \Re e^{-\eta \tau} \right\}_\eta = \omega_0
\]

\[
= \text{Sign} \left\{ \left( 3 \omega_0^4 + 2 Q_4 \omega_0^3 + (Q_2^2 - 2 Q_4 Q_3) \right) \right\} \frac{Q_4 \omega_0}{Q_4} > 0.
\]

This proves the lemma. Now we have the following theorem. □

**Theorem 7.** The endemic equilibrium \( E_+ \) of the system is locally asymptotically stable when \( \tau < \tau_0 \) and becomes unstable for \( \tau > \tau_0 \) provided

\[
\frac{\mu \eta_0}{2 \omega_0^2} < \lambda_0 + d + q \psi < \min \left\{ \frac{\lambda M^*}{1 + k M^*}, \frac{\mu \lambda (\psi + d)}{\beta(1 + k M^*)^2} \right\}.
\]

When \( \tau = \tau_0 \), a Hopf-bifurcation occurs, leading a family of periodic solutions bifurcating from \( E_+ \) as \( \tau \) passes through the critical value \( \tau_0 \), where

\[
\tau_0 = \frac{1}{\omega_0} \arccos \frac{Q_4 \omega_0^2 - Q_3}{Q_4}.
\]
4. Numerical Simulations and Results

To check the feasibility of our analysis in Section 3 about \( E_0 \), we present some numerical computations in this section using MatLab by choosing the following set of parameter values: \( d = 0.002, \beta = 0.4, \mu_0 = 0.08, \mu = 0.2, \nu = 0.46, q = 0.6, \lambda = 0.2, \lambda_0 = 0.4, m_0 = 0.04, k = 0.5, \) and \( \tau = 10 \) (see Figure 1), and then we get Figure 2 with different initial values, from which we can know that \( E_0 \) is stable and all the trajectories approach it. The numerical simulations support the analysis given.

Then we choose the following set of parameter values which satisfy the condition in Theorem 7: \( d = 0.00004, \beta = 0.3, \mu_0 = 0.14, \mu = 0.35, \nu = 0.2, q = 0.15, \lambda = 0.019, \lambda_0 = 0.001, m_0 = 0.000005, \) and \( k = 0.0003 \). The numerical value of \( \tau_0 \) computed is found to be 30.12. When \( \tau > 0 \), we give different \( \tau \) as follows: \( \tau = 20, \tau = 25, \tau = 35, \) and \( \tau = 40 \) (Figures 3 and 4). As shown in Figures 3 and 4 the variables approach their equilibria when \( \tau \) is less than \( \tau_0 \), whereas, as \( \tau \) exceeds its critical value \( \tau_0 \), all variables start showing oscillatory behavior. This indicates that, in the latter case, sometimes the infective will be high and sometimes will be low and it may be difficult to make the prediction regarding the size of epidemic. It is clear that \( \tau \) plays a key role in the prevention and control of diseases.

In the following we let \( \tau = 10 \) and make \( \lambda, \mu \) change from 0.05 to 0.8 and research the influence of the dissemination rate \( \lambda \) and the implementation rate \( \mu \) on the infective \( Y(t) \) separately. The variation of \( Y(t) \) with respect to time \( t \) for different values of \( \lambda \) and \( \mu \) is shown in Figure 5, and we discover all of the proportions of \( Y^* \) reductions as \( \lambda \) and \( \mu \) increase, which proves the conclusions of the remark, but \( \lambda \) has greater influence on \( Y(t) \) than \( \mu \). In addition the reason why \( Y(t) \) has a similar trend in Figure 5 is that \( \lambda \) and \( \mu \) play a similar trend with \( M(t) \). There really is an effort here to make clear that \( \lambda \) and \( \mu \) play a key role in the prevention and control of diseases.

A comparison between the oscillations in \( Y \) and \( M \) is presented in Figure 6. From this figure, we obtain that, as the infective increase, the awareness programs also start growing with some time lag due to time delay in the execution of awareness programs. As soon as the awareness programs reach a potentially high value, the infective start decreasing due to the execution of awareness programs in the infective. Awareness programs after certain time also start decreasing, which eventually results in the increase of the infective. Both
Figure 3: The stability of the endemic equilibria $E_*$ with $\tau = 20$ and $\tau = 40$, respectively.

Figure 4: The stability of the endemic equilibria $E_*$ with $\tau = 25$ and $\tau = 35$, respectively.

Figure 5: When $\tau = 10$, the stability of $Y^*$ with variational $\lambda$ and $\mu$, respectively.
of the figures show the interplay between Y and M, which proves the existence of delay.

5. Discussion

In this paper, a nonlinear mathematical model with delay and awareness programs driven by media has been proposed and analyzed. It is assumed that pathogens are transmitted via direct contact between the susceptible and the infective. The model exhibits two equilibria, and the disease-free equilibrium has been shown to be stable for basic reproduction number \( R_0 < 1 \) when \( \tau \geq 0 \). For \( R_0 > 1 \), it leads to the existence and stability of an endemic equilibrium under some conditions in absence of time delay. But for \( \tau > 0 \) this equilibrium is locally asymptotically stable when the delay is suitably small; that is, \( \tau < \tau_0 \), while a loss of stability by a Hopf-bifurcation can occur as delay increases. Numerical simulations prove the stability of equilibria and show that the value of delay sustainable for the disease is 30.12. The numerical results suggest that if we want to reduce the proportion of the infective, we can increase the dissemination rate \( \lambda \) and implementation rate \( \mu \). At last we obtain the existence of \( \tau \) from Figure 6. In short media is a key tool for influencing people behavior towards the disease to devise proper policies for controlling the epidemic.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publishing of this paper.

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