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A delay differential equation model of SEIV in presence of media coverage

Piu Samui[a], Jayanta Mondal[b], Amar Nath Chatterjee[c,*]

[a]Department of Mathematics, Swami Vivekananda University, West Bengal-700121, India
[b]Department of Mathematics, Diamond Harbour Women's University, Sarisha, West Bengal-743368, India
[c]Department of Mathematics, K. L. S. College, Nawada, Magadh University, Bodh Gaya, Bihar-805110, India

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Abstract

During an epidemic, existing mass media plays a fundamental role in promoting effective, trustworthy and convenient information regarding disease symptoms and prevention measures against the infection. In this research paper, we aim to explore the impact of media awareness projected to a SEIV compartmental model incorporating newly modulated saturated incidence function and discrete-time delay during an epidemic. We considered the time lag in between the process while unaware susceptible individuals would be aware through the campaign media. Sensitivity analysis reveals the influence of the model parameters in the progression of the epidemic. Numerical simulations enable us to visualize the importance of media awareness to convey predictions regarding the mitigation and apparent eradication of the epidemic.

Keywords: SEIV model, Time delay, Hopf-bifurcation;, Media awareness, Sensitivity analysis
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1 Introduction

Infectious diseases are supposed to be the leading root of mortality and the deterioration in comprehensive health and health policies, particularly in developing countries. The world has been confronting several life-threatening epidemics viz. Tuberculosis (TB), Malaria, Measles, Influenza, Dengue, HIV/AIDS, HCV, Ebola, Cancer, Chickenpox, Rabies, Marburg virus disease, Spanish flu, MERS-CoV etc., and newly emerged SARS-CoV-2 or COVID-19 infection. Morbidity due to infectious diseases is triggering a global crisis causing about 11 million deaths per year including neonatal mortality and infant mortality in developing countries [3]. After the emergence of the COVID-19 pandemic, the global health process achieved during the last two decades collapsed very rapidly and the World Health Organization (WHO) declared the year 2020 as the most devastating year in the history of global health [25]. In the critical situation of building resistance against COVID-19 infection, previously mentioned infectious diseases reemerged on a large scale. At the initial stage of an epidemic eruption, control of disease transmission should be the main aim of researchers as well as of the public health sectors of the countries. To promote awareness concerning the symptoms and patterns

*Corresponding author
Email addresses: piusamui18@gmail.com (Piu Samui), jayantajumath@gmail.com (Jayanta Mondal), anchaterji@gmail.com (Amar Nath Chatterjee)

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of infectious disease and to convey Government orders and public guidelines, mass media like TV news, newspapers, radio, and internet/social media (such as Facebook, WhatsApp, Instagram, Twitter, and YouTube) generally assist in sharing information and to connect the frontline workers, health workers, and the masses. However, it has been observed that in a handful of circumstances, continuous media campaigns regarding an epidemic outbreak generated rumours and anxiety among people and indeed, this situation may delay in controlling the epidemic. Despite this disadvantage, media is the predominant weapon in controlling infectious disease progression by decreasing social communication and gathering [21, 22].

The leading modes of transmission of an infectious disease might be classified into two broad ways - either horizontally, or vertically (in some cases via both pathways). In the case of horizontal transmission, the transmission occurs through contact among persons belonging to the same generation. Disease transmission directly from the mother to an embryo, fetus, or newborn baby at the time of pregnancy or childbirth is referred to as vertical transmission. The etiological agents of infectious diseases are viruses, bacteria, fungi and parasites. In this research article, our main focus is centred on those infectious diseases which are spread by viral agents and follow the horizontal pathway of transmission.

From a mathematical perspective, the models which are related to epidemiology, provide an idea related to the mechanisms of progression, mitigation and control of contagious diseases. In 1927, Kermack-Mckendrick [13] first introduced a basic SIR compartmental model to describe infectious disease systems using the mass action term in the form $\frac{CSI}{N}$, where C, S and I denote the disease transmission rate, susceptible individuals, and infected individuals respectively and N stands for the total population. Epidemiological research has been conducted enormously, specifically after the COVID-19 pandemic, intended to understand, anticipate and mitigate the spread of the infection, and to evaluate the efficacy and efficiency of prevention strategies ensuring the responsiveness of the health system [36, 24, 18, 37, 34].

Researchers have already developed various epidemiological models taking into account the disease transmission along with or without time delay. Incalculable papers have been published applying non-linear incidence rate [8, 38, 17, 30, 11, 12, 31, 23]. The delay differential equation helps us to justify a variety of physical, biological, and social science phenomena, including the spread of disease and population growth [26, 5, 27]. During the disease's transmission, the disease's progression is not instantaneous. The virus takes time to travel from one person to another. Thus, the growth of new infections takes time. The delay inclusion in the differential equation model helps us to predict the behaviour of the epidemic system.

The incidence rate plays the main role in formulating a mathematical model. If the number of infected individuals increases then the force of infection will decrease. According to the biological point of view, there are many reasons to initiate the time delays in the mathematical model of disease transmission. The various articles have been published with a discrete-time delay and non-linear incidence rate [29, 28, 19, 33]. Different biological mechanisms considering time delay in epidemiological models are as follows:

(i) Delay due to short-term immunity: Several types of infections take steps or time lag to recover with immunity reinfection. Some type of diseases provides lifelong immunity [16, 4].

(ii) Delay affected by latency: We know that various infectious diseases spread from one host to another by a vector carrying a pathological agent. Malaria and dengue fever are such a type of diseases that spread by vector and the diseases take some time to spread in humans. According to various organizations' reports in all the countries of the World, the transmission rate of such types of diseases is very high [2, 1].

We have proposed a SEIV model with a new modulated saturated incidence rate i.e. $\frac{CSI}{1+\alpha I}$ and assuming that the disease is transmitted horizontally via direct or indirect contact. Also in our proposed SEIV epidemic model, we have incorporated the mass media effect along with the time delay effect. In this context, we incorporate the media coverage by considering the incidence $\beta(I, \alpha_i) = 1 - \frac{I}{I+\alpha_i}$. With respect to media responses against disease transmission, a large number of mathematical modelling have been already studied [35, 9, 20]. These articles, mainly have studied the effect of mass media on the well-known Susceptible-Exposed-Infectious-Vaccinated-Recovered (SEIV) model with various extensions. The main focus of our study on a delayed SEIV model is to explore the role of mass media responses to control disease transmission in the presence of time delay.

In this paper, Section 2 deals with the formulation of a delayed SEIV model. In this Section, we have studied the existence and stability conditions of the equilibrium points executed by the system. We have studied the sensitivity analysis of the system parameters in Section 3. Section 4 is accomplished with numerical illustrations of the analytical findings. In the last Section (Section 5), we discussed our overall findings with biological interpretations.

2 SEIV framework

In this present study, we consider a delayed SEIV epidemic model with new modulated saturated incidence with media coverage. For our study, we assume that the total population is constant and it is denoted by N. We divide the total population into four subclasses briefly, susceptible class S, exposed class E, infected class I, and treatment class or vaccinated recovery class V. We assume here that infectious diseases spread only by direct, or indirect contact following a horizontal pathway. Our proposed four-dimensional ODE compartmental model with discrete time delay is formulated as below:

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - \beta(I,\alpha_i)[C_1\psi(S(t-\tau),I(t-\tau)) + C_2\psi(S(t-\tau),E(t-\tau))] + \xi V - \delta S,\\
\frac{dE}{dt} &= \beta(I,\alpha_i)[C_1\psi(S(t-\tau),I(t-\tau)) + C_2\psi(S(t-\tau),E(t-\tau))] - \sigma E - \delta E,\\
\frac{dI}{dt} &= \sigma E - \gamma I - \delta I,\\
\frac{dV}{dt} &= \gamma I - \xi V - \delta V,
\end{aligned} \tag{2.1}$$

where,

$$\psi(S(t-\tau),I(t-\tau)) = \frac{S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}, \quad \psi(S(t-\tau),E(t-\tau)) = \frac{S(t-\tau)E(t-\tau)}{1+\alpha I(t-\tau)}, \quad \text{and} \quad \beta(I,\alpha_i) = \Big(1 - \frac{I}{\alpha_i + I}\Big),$$

and the model is satisfied non-negative initial conditions:

$$S(\phi) = S_0, E(\phi) = E_0, I(\phi) = I_0, V(\phi) = V_0, \text{ where } \phi \in (-\tau, 0]. \tag{2.2}$$

The susceptible individuals are recruited at a constant rate Π. The per capita contact rate between the susceptible class and infected class is C_1. C_2 is the contact rate between the infected class and the exposed class, α is a positive constant, δ is the mortality rate of all adult classes. The latent time delay is $\tau > 0$ and it makes a susceptible individual infected after interaction with exposed and infected individuals. The exposed individuals are recruited to the infected class at a rate of σ. γ is the rate at which the infected individuals are being recruited to treatment class. ξ is the at which the individuals leave the compartment V and enter the susceptible class again. $\Big(1 - \frac{I}{I+\alpha_i}\Big)$ is the mass media function employed in our model [7], where α_i represent the rate of media effect in these function. We consider a new modulated saturated incidence rate of the form $\frac{CSI}{1+\alpha I}$ which indicates that the infection force decreases when the number of infectives is large.

Table 1: Variables and biological relevant parameters values used for numerical simulations of the system (2.1).

Parameters	Biological meaning	Assigned value (unit) day^{-1}
Π	Constant recruitment rate of susceptible class	0.3
C_1	Per capita contact rate between infected class and exposed class	$0.001 \sim 0.01$
C_2	Per capita contact rate between susceptible class and infected class	$0.002 \sim 0.2$
α	Positive constant	0.001
δ	Natural death rate of all adults class	$0.006 \sim 0.06$
σ	Rate by which the exposed classes are recruited to infected class	$0.02 \sim 0.2$
γ	Rate by which the exposed classes are recruited to infected class	$0.04 \sim 0.4$
ξ	Treatment rate	0.02
α_i	Rate of media response	0.1

For later use, we rewrite the compact form of system (2.1) as follows,

$$\frac{dX}{dt} = M(X), \quad X = (x_1, x_2, x_3, x_4)^T, \quad M = (M_1, M_2, M_3, M_4)^T, \tag{2.3}$$

where, $M_i's$ is indicate the RHS of system (2.1).

2.1 Positive invariance regions

From a biological point of view, non-negativity for a population means its existence. The results are given through the following theorems.

Theorem 2.1. The solutions of system (2.1) are positive with non-negative initial conditions (2.2) in following region of attraction set:

$$\Omega = \Big\{(S,E,I,V) \in \mathbb{R}^4_+ \mid 0 < S+E+I+V \leq \frac{\Pi}{\delta}\Big\}.$$

Proof . Following Lemma in [6, 10], in the region $\mathbb{R}^4_+$ the solution of system (2.1) exists. All solutions are positive for all $t > 0$. Now we check from the system (2.3) that when selecting $X(\varphi) \in \mathbb{R}^4_+$, such that $S = 0$, $E = 0$, $I = 0$, and $V = 0$, it follows $M_i(X)|_{x_i=0, X\in\mathbb{R}^4_+} \geq 0$, with $x_1 = S_0$, $x_2 = E_0$, $x_3 = I_0$, $x_4 = V_0$.

Here, using the Lemma [10], and the Theorem [6], any solution of system (2.1) with $X(\varphi) \in C$, and $X(t) = (t,\ X(\varphi))$, where $X(\varphi) \in \mathbb{R}^4_+,\ \forall\ t$. Therefore the solution of the system (2.1) exists in $\mathbb{R}^4_+$ and all variables remain positive with $t > 0$. Hence, the non-negative cone $\mathbb{R}^4_+$ is an invariant region.

Applying above assessment of system (2.1), we get the region of attraction set:

$$\Omega = \Big\{(S,E,I,V) \in \mathbb{R}^4_+ \mid 0 < S+E+I+V \leq \frac{\Pi}{\delta}\Big\}. \tag{2.4}$$

Thus, the analytical results imply that for all time $t > 0$ the solutions of the system (2.1) are bounded which biologically implies robustness of the system. □

2.2 Equilibrium analysis

The system (2.1) has two steady state solutions:

(i). The disease - free equilibrium point $\mathcal{U}_0(S^0,\ 0,\ 0,\ 0)$ with $S^0 = \Pi\delta^{-1}$.

(ii). The endemic equilibrium point $\mathcal{U}^*(S^*,\ E^*,\ I^*,\ V^*)$, where,

$$S^* = \frac{\Pi\sigma(\xi+\delta) + I^*[\xi\gamma\sigma - (\gamma+\delta)(\xi+\delta)(\sigma+\delta)]}{\sigma\delta(\xi+\delta)}, \quad E^* = \frac{\gamma+\delta}{\sigma}I^*, \quad V^* = \frac{\gamma}{\xi+\delta}I^*,$$

and I^* is real and positive root(s) of the following equation

$$L_1(I^*)^2 + L_2 I^* + L_3 = 0, \tag{2.5}$$

where

$$\begin{aligned}
L_1 &= \sigma^2\delta\alpha_i(\gamma+\delta)(\sigma+\delta)(\xi+\delta),\\
L_2 &= (C_1\sigma + C_2(\gamma+\delta))(\alpha_i(\gamma+\delta)(\sigma+\delta)(\xi+\delta) - \xi\gamma\sigma) + \sigma^2\delta(1+\alpha_i\alpha)(\gamma+\delta)(\sigma+\delta)(\xi+\delta),\\
L_3 &= \sigma^2\delta\alpha_i(\xi+\delta)(\sigma+\delta)(\gamma+\delta)(1-R_0).
\end{aligned}$$

Here the term R_0 is denoting the basic reproduction number of the system which is computed by next-generation matrix method and is defined as

$$R_0 = \frac{\Pi(C_1\sigma + C_2(\gamma+\delta))}{\delta(\sigma+\delta)(\gamma+\delta)}. \tag{2.6}$$

From equation (2.5) we obtain

$$I^* = \Big[-L_2 \pm (L_2^2 - 4L_1L_3)^{1/2}\Big] \Big/ \Big(2L_1\Big). \tag{2.7}$$

Therefore, I^* has real positive root when $L_3 > 0$, which implies that $R_0 > 0$ and S^* is positive definite while $\xi\gamma\sigma > (\gamma+\delta)(\xi+\delta)(\sigma+\delta)$.

2.3 Stability of delayed model

We linearize the system (2.1) by replacing $S(t) = S^* + S_1$, $E(t) = E^* + E_1$, $I(t) = I^* + I_1$, $V(t) = V^* + V_1$ and consider the first order term. Thus the system in linearized form is as follows:

$$\begin{aligned}
\frac{dS_1}{dt} &= -\frac{\beta(I^*,\alpha_i)S_1(t-\tau)}{1+\alpha I^*}(C_1I^* + C_2E^*) - \frac{\beta(I^*,\alpha_i)C_1S^*E_1(t-\tau)}{1+\alpha I^*} + \xi V_1 - \delta S_1 \\
&- \frac{\beta(I_1,\alpha_i)S^*}{1+\alpha I^*}(C_1I^* + C_2E^*) - \frac{\beta(I^*,\alpha_i)S^*I_1(t-\tau)}{(1+\alpha I^*)^2}(C_1 - C_2\alpha_1E^*), \\
\frac{dE_1}{dt} &= \frac{\beta(I^*,\alpha_i)S_1(t-\tau)}{1+\alpha I^*}(C_1I^* + C_2E^*) + \frac{\beta(I^*,\alpha_i)C_1S^*E_1(t-\tau)}{1+\alpha I^*} \\
&+ \frac{\beta(I_1,\alpha_i)S^*}{1+\alpha I^*}(C_1I^* + C_2E^*) + \frac{\beta(I^*,\alpha_i)S^*I_1(t-\tau)}{(1+\alpha I^*)^2}(C_1 - C_2\alpha_1E^*), \\
\frac{dI_1}{dt} &= \sigma E_1 - (\gamma+\delta)I_1, \\
\frac{dV_1}{dt} &= \gamma I_1 - (\xi+\delta)V_1.
\end{aligned} \tag{2.8}$$

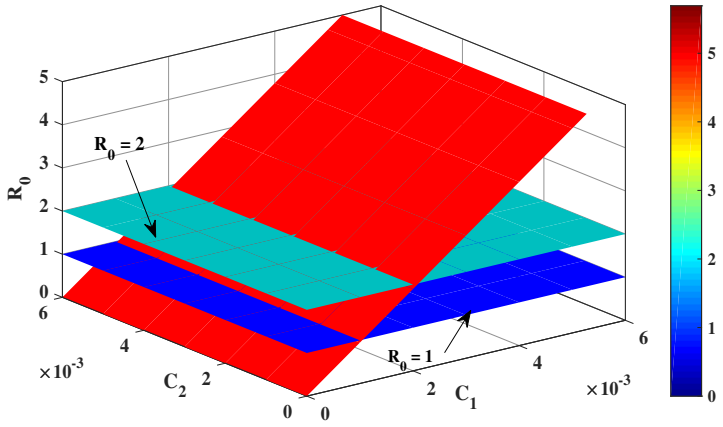

Figure 1: The figure indicates the changes of the value of the basic reproduction number R_0 when the contact rates C_1 and C_2 vary simultaneously in our system (2.1). The parametric values are same as in Table 1.

Inside this Section, we study the local stability of the system (2.1) about the endemic equilibrium point. The linearized form of the system (2.8) can be expressed in a matrix form as follows:

$$\frac{dX}{dt} = GX(t) + HX(t-\tau), \tag{2.9}$$

where $G = [a_{ij}]_{4\times4}$ and $H = [b_{ij}]_{4\times4}$ are the following matrices:

$$G = \begin{pmatrix} -\delta & 0 & 0 & \xi \\ 0 & -a_{22} & 0 & 0 \\ 0 & \sigma & -a_{33} & 0 \\ 0 & 0 & \gamma & -a_{44} \end{pmatrix} \quad \text{and} \quad H = \begin{pmatrix} -b_{11} & -b_{12} & -b_{13} & 0 \\ b_{11} & b_{12} & b_{13} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

considering

$$\begin{aligned}
a_{22} &= \sigma + \delta, \ a_{33} = \gamma + \delta, \quad a_{44} = \xi + \delta, \\
b_{11} &= \frac{\beta'(I^*,\alpha_i)S^*}{1+\alpha I^*}(C_1I^* + C_2E^*) + \frac{\beta(I^*,\alpha_i)C_1}{1+\alpha I^*}(C_1I^* + C_2E^*), \\
b_{12} &= \frac{\beta(I^*,\alpha_i)C_1S^*}{1+\alpha I^*}, \quad b_{13} = \frac{\beta(I^*,\alpha_i)C_1S^*}{1+\alpha I^*}(C_1 - C_2\alpha E^*).
\end{aligned}$$

For positive delay, the characteristic equation of system (2.1) is

$$\Phi(\eta) = |\eta I_4 - G - e^{-\eta\tau} H| = 0. \tag{2.10}$$

Thus the characteristic equation (2.10) of the model (2.1) can be written as

$$\Psi(\eta, \tau) = \eta^4 + \vartheta_1\eta^3 + \vartheta_2\eta^2 + \vartheta_3\eta + \vartheta_4 + e^{-\eta\tau}|\kappa_1\eta^3 + \kappa_2\eta^2 + \kappa_3\eta + \kappa_4| = 0, \tag{2.11}$$

where

$$\begin{aligned}
\vartheta_1 &= \delta + a_{22} + a_{33} + a_{44}, \quad \vartheta_2 = \delta a_{22} + a_{33}a_{44} + (\delta + a_{22})(a_{33} + a_{44}),\\
\vartheta_3 &= \delta a_{22}(a_{33} + a_{44}) + a_{33}a_{44}(\delta + a_{22}), \ \vartheta_4 = \delta a_{22}a_{33}a_{44},\\
\kappa_1 &= b_{11} - b_{12}, \ \kappa_2 = a_{22}b_{11} - \delta b_{12} + (a_{33} + a_{44})(b_{11} - b_{12}),\\
\kappa_3 &= (a_{33} + a_{44})(a_{22}b_{11} - \delta b_{12}) + a_{33}a_{44}(b_{11} - b_{12}), \ \kappa_4 = a_{33}a_{44}(a_{22}b_{11} - \delta b_{12}).
\end{aligned}$$

In the case when $\tau = 0$, the above characteristic equation of (2.11) is converted to

$$\eta^4 + h_1\eta^3 + h_2\eta^2 + h_3\eta + h_4 = 0, \tag{2.12}$$

where the terms $h_i's, i = 1, 2, 3, 4$ are given as

$$h_1 = \vartheta_1 + \kappa_1, \ h_2 = \vartheta_2 + \kappa_2, \ h_3 = \vartheta_1 + \kappa_3, \ h_4 = \vartheta_4 + \kappa_4.$$

Therefore, using Routh-Hurwitz criterion, all roots of the equation (2.12) have negative real parts if

$$h_i > 0; \ i = 1, 2, 3, 4, \ h_1h_2 - h_3 > 0 \text{ and } h_3(h_1h_2 - h_3) - h_1^2h_4 > 0. \tag{2.13}$$

So we can conclude that for $\tau = 0$, the system (2.1) is locally asymptotically stable around $\mathcal{U}^*$.

In case of positive delay, at $\mathcal{U}^*$ if all roots of (2.11) are negative or roots have negative real parts then the system becomes locally asymptotically stable. Now, we find the necessary condition of $\mathcal{U}^*$ for changes in its stability. Let us consider that the characteristic equation (2.11) has purely imaginary solutions. Let $\eta = i\omega$, $\omega \in \mathbb{R}$ be a root of (2.11). We obtain

$$(\kappa_4 - \kappa_2\omega^2)\cos\omega\tau + (\kappa_3 - \kappa_1\omega^2)\omega\sin\omega\tau = -\omega^4 + \vartheta_2\omega^2 - \vartheta_4, \tag{2.14}$$

$$(\kappa_1\omega^2 - \kappa_3)\omega\cos\omega\tau + (\kappa_4 - \kappa_2\omega^2)\sin\omega\tau = \vartheta_3\omega - \vartheta_1\omega^3. \tag{2.15}$$

Squaring and adding the equation (2.14) & (2.15), we obtain

$$\omega^8 + (\vartheta_1^2 - 2\vartheta_2 - \kappa_1^2)\omega^6 + (\vartheta_2^2 + 2\vartheta_4 - 2\vartheta_1\vartheta_3 - \kappa_2^2 + 2\kappa_1\kappa_3)\omega^4 + (\vartheta_3^2 - 2\vartheta_2\vartheta_4 - \kappa_3^2 + 2\kappa_2\kappa_4)\omega^2 + \vartheta_4^2 - \kappa_4^2 = 0. \tag{2.16}$$

Let $\zeta = \omega^2$, then we rewrite the equation (2.16) we get

$$\zeta^4 + \varrho_1\zeta^3 + \varrho_2\zeta^2 + \varrho_3\zeta + \varrho_4 = 0, \tag{2.17}$$

where

$$\begin{aligned}
\varrho_1 &= \vartheta_1^2 - 2\vartheta_2 - \kappa_1^2, \quad \varrho_2 = \vartheta_2^2 + 2\vartheta_4 - 2\vartheta_1\vartheta_3 - \kappa_2^2 + 2\kappa_1\kappa_3\\
\varrho_3 &= \vartheta_3^2 - 2\vartheta_2\vartheta_4 - \kappa_3^2 + 2\kappa_2\kappa_4, \quad \varrho_4 = \vartheta_4^2 - \kappa_4^2.
\end{aligned}$$

When all roots of the equation (2.17) are negative then the Routh–Hurwitz criterion is satisfied. Equation (2.11) does not indicate that roots are purely imaginary. So, we have the following proposition.

Proposition 2.2. If we assume that the system without delay is stable then at $\mathcal{U}^*$ the system is LAS for all $\tau > 0$ with the condition:

$$\varrho_1 > 0, \varrho_4 > 0, \quad \varrho_1\varrho_2 - \varrho_3 > 0, \quad (\varrho_1\varrho_2 - \varrho_3) - \varrho_1^2\varrho_4 > 0.$$

If $\varrho_4 < 0$ holds then equation (2.17) will lead to at least one positive root. Also there will be purely imaginary root, $\pm i\omega_0$ corresponding to the delay τ for (2.17) when ω^2 is the minimum positive root of (2.17). According to Butler's lemma, [10], the endemic equilibrium $\mathcal{U}^*$ will be stable for $\tau < \tau^*$.

Now we evaluate the critical value of τ for which the delayed system (2.11) attains its stability.

From (2.14),

$$\tau^* = \frac{2\pi r}{\omega_0} + \frac{1}{\omega_0}\cos^{-1}\Big[\frac{\mathcal{X}}{\mathcal{Y}}\Big], \ r = 0, 1, 2, 3, ... \tag{2.18}$$

where

$$\begin{aligned}\mathcal{X} &= (\kappa_2 - \vartheta_1\kappa_1)\omega_0^6 - (\vartheta_2\kappa_2 - \vartheta_3\kappa_1 - \vartheta_1\kappa_3 + \kappa_4)\omega_0^4 + (\vartheta_2\kappa_4 + \vartheta_4\kappa_2 - \vartheta_3\kappa_3)\omega_0^2 - \vartheta_4\kappa_4,\\ \mathcal{Y} &= \kappa_1^2\omega_0^6 + (\kappa_2^2 - 2\kappa_1\kappa_3)\omega_0^4 + (\kappa_3^2 - 2\kappa_2\kappa_4)\omega_0^2\kappa_4^2.\end{aligned}$$

2.4 Analysis of hopf-bifurcation

Theorem 2.3. The steady state at $\mathcal{U}^*$ is LAS when $\tau < \tau^*$ with $\varrho_4 < 0$ and the system switches to unstable state when $\tau > \tau^*$. Also, the system attains a hopf-bifurcation at P^* when $\tau = \tau^*$ if $4\omega_0^6 + J_1\omega_0^4 + J_2\omega_0^2 + J_4 > 0$, where

$$\begin{aligned}J_1 &= 3\vartheta_1^2 - 6\vartheta_2 - 3\kappa_1^2\\ J_2 &= 2\vartheta_2^2 + 4\vartheta_4 - 4\vartheta_1\vartheta_3 - 2\kappa_2^2 + 4\kappa_1\kappa_3\\ J_3 &= \vartheta_3^2 - 2\vartheta_2\vartheta_4 - \kappa_3^2 + 2\kappa_2\kappa_4\end{aligned}$$

Proof . We only need to prove the last condition. Differentiating the equation (2.11) w.r.t τ, we get

$$\frac{d\tau}{d\eta} = \frac{4\eta^3 + 3\vartheta_1\eta^2 + 2\vartheta_2\eta + \vartheta}{\kappa_1\eta^4 + \kappa_2\eta^3 + \kappa_3\eta^2 + \kappa_4\eta}e^{\eta\tau} + \frac{3\kappa_1\eta^2 + 2\kappa_2\eta + \kappa_3}{\kappa_1\eta^4 + \kappa_2\eta^3 + \kappa_3\eta^2 + \kappa_4\eta} - \frac{\tau}{\eta}.$$

Now, using the relation (2.14) we can get:

$$\begin{aligned}sign\Big[\frac{d(Re\eta)}{d\tau}\Big]_{\tau=\tau^*} &= sign\Big[Re\Big(\frac{d\eta}{d\tau}\Big)^{-1}\Big]_{\eta=i\omega_0}\\ &= sign\Big[\frac{4\omega_0^6 + J_1\omega_0^4 + J_2\omega_0^2 + J_4}{\kappa_1\omega_0^6 + \kappa_2\omega_0^4 + \kappa_3\omega_0^2 + \kappa_4}\Big].\end{aligned} \tag{2.19}$$

and the latter is positive if $4\omega_0^6 + J_1\omega_0^4 + J_2\omega_0^2 + J_4 > 0$ which indicates the transversality condition. Then the system attains hopf-bifurcation when τ passes its critical value $\tau = \tau^*$, and the system bifurcates from the equilibrium point $\mathcal{U}^*$ where τ^* is very small and positive value given in the equation (2.18). □

Accordingly, summarizing above results we construct the following theorem [14, 15]:

Theorem 2.4. We assume that $\mathcal{U}^*(S^*, E^*, I^*, V^*)$ exists and the condition (2.13) is satisfied for system (2.1) with non-negative initial conditions (2.2). Then

($T1$). When $\tau < \tau^*$, the system is locally asymptotically stable at the equilibrium point $\mathcal{U}^*$. .

($T2$). When $\tau > \tau^*$, the system is unstable at the endemic equilibrium point $\mathcal{U}^*$.

($T2$). When $\tau = \tau^*$, the system attains hopf-bifurcation around the endemic equilibrium point $\mathcal{U}^*$.

3 Sensitivity analysis of the system

In our proposed model, we have considered only such types of disease which are only spread by contact. For this concern, we have some responsibilities to find out a desirable way to decrease disease transmission. We are trying to observe those parameters, which are responsible for an increase in disease transmission. Our concentration to carry out the sensitivity of those parameters formerly which have been given in Table 1 associated to R_0 to measure the robustness of the system (2.1) and their influences in the disease dynamics. In epidemiology, basically, disease transmission and control of diseases entirely depend on R_0. First, now investigate the sensitivity of R_0 with respect to the parameters of the without delay system following the method of [32, 26] and using the formula

$$\Xi_X^{R_0} = \frac{\partial R_0}{\partial X} \times \frac{X}{R_0}.$$

Table 2: Sensitivity indices of R_0 with respect to the model parameters.

Parameters	C_1	δ	Π	σ	C_2	γ
Values	0.6849	-0.3318	1.0000	-0.0030	0.3151	-0.5956

From Table 2 it is clear that the value of R_0 will either increase or decrease depending on the parameters Π, C_1 and C_2. Therefore, if the values of the recruitment rate of the susceptible population, and contact rate of the susceptible population with the infected and exposed class will increase (decrease) then the values of R_0 will also increase (decrease). On the other hand δ, σ and γ have inversely proportional relationship with R_0. If we increase the value of σ and γ then the value of R_0 decreases (see Figure 2).

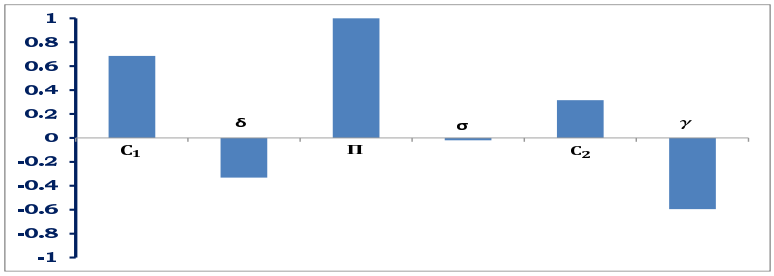

Figure 2: The figure shows tornado plot regarding the sensitivity indices of model parameters linked to R_0.

4 Numerical simulations

In this Section, numerical simulations of the non-delayed and delayed model are carried out with the parameter units as in Table 1. For those numerical simulations, we expect that the initial conditions had been taken to be consistent in $(-\tau,\ 0)$. Here the model variables are perturbed marginally from the equilibrium $\mathcal{U}^*$. Here our most important goal is to study the stability of the system (2.1) at $\mathcal{U}^*$. With the help of our analytical findings, we plot a surface plot (see Figure 1) of R_0 among R_0 and the contact rates C_1 and C_2. In this figure, we see that as C_1 and C_1 increase, R_0 reaches its threshold value 1, and the system switches from a disease-free state to an endemic state. Thus disease-free state persists with a low contact rate.

Figure 2 shows the partial rank correlation coefficient sensitivity analysis. All relevant parameters are varied against R_0 throughout the range given in Table 1. Parameters with $PRCCs > 0$ will increase R_0 when they are increased, while the parameters with $PRCCs < 0$ will decrease R_0 when the corresponding parameters increase. From this figure, it shows that Π, C_1, and γ play the most significant role in controlling the epidemic. Here $\Xi_\Pi^{R_0} = 1$ is the largest sensitive index and $\Xi_\gamma^{R_0} = -0.5956$ is the least sensitivity index. If Π decreases 1% then R_0 also reduces 1%. Also, R_0 is an increasing function of C_1 and C_2. Also, it is observed that R_0 is a decreasing function of δ and α respectively.

Figure 3 shows the trajectories of the model variables for different values of the delay factor τ. The initial values are managed by perturbing model variables from $\mathcal{U}^*$. Here it is clearly observed that oscillations in the solution persist for longer due to increasing value of delay. We have calculated the threshold value of delay $\tau^* = 13.6$ days. When $\tau = 10 < \tau^*$ the initial oscillation does not persist and the system becomes stable. When $\tau = 15 > \tau^*$, the initial

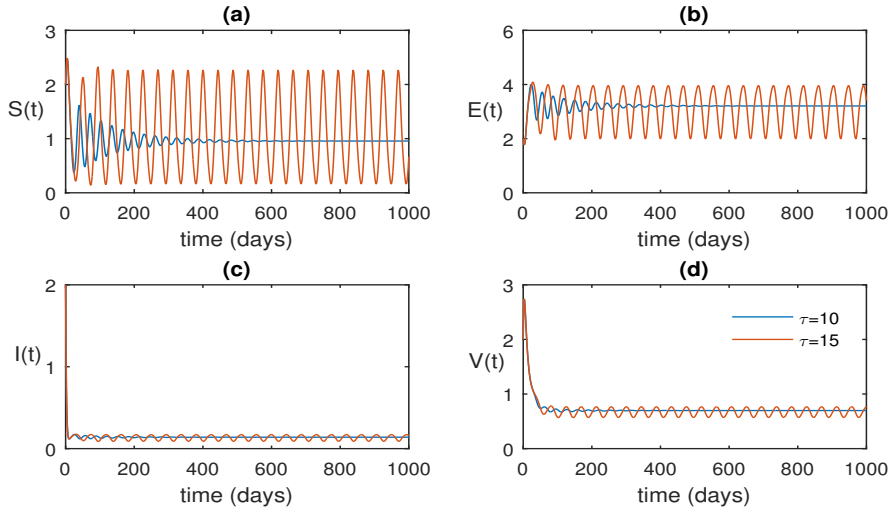

Figure 3: Trajectories of model variables for different values of τ with $\alpha_i = 0.1$. The model parameters are as in Table 1.

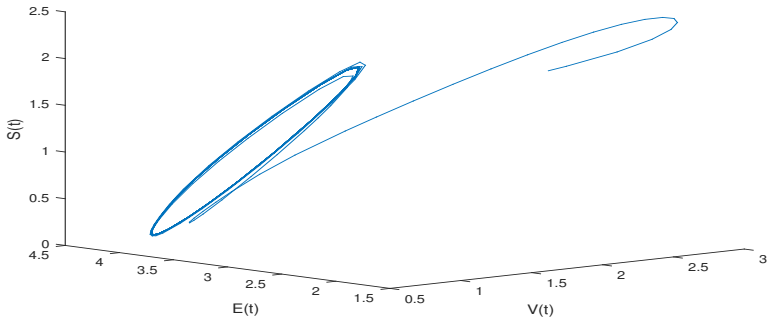

Figure 4: Phase plane at $\tau = 15$ with $\alpha_i = 0.1$.

oscillations continue for a longer period and attain cyclic oscillations (see Figure 4). When $\tau > \tau^*$ the system becomes unstable and the oscillatory solutions increase in amplitude until they reach values where the approximate model becomes unrealistic.

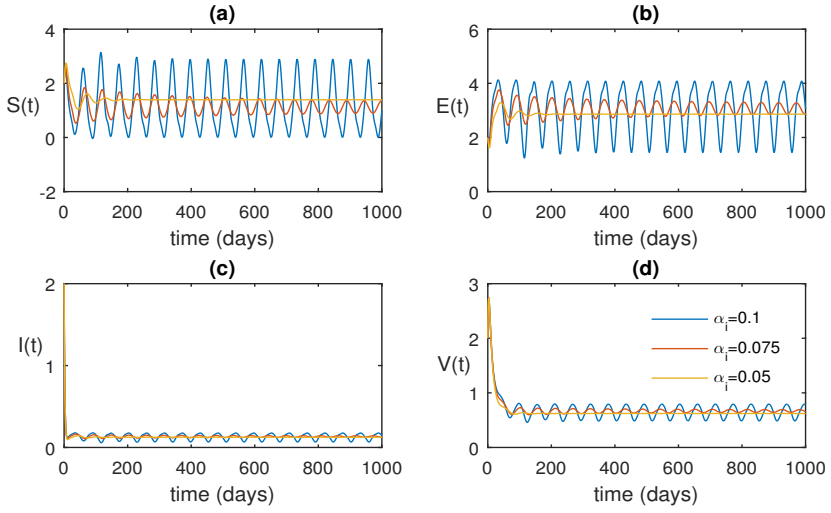

Figure 5: Trajectories of model variables for different values of α_i with perturbation.

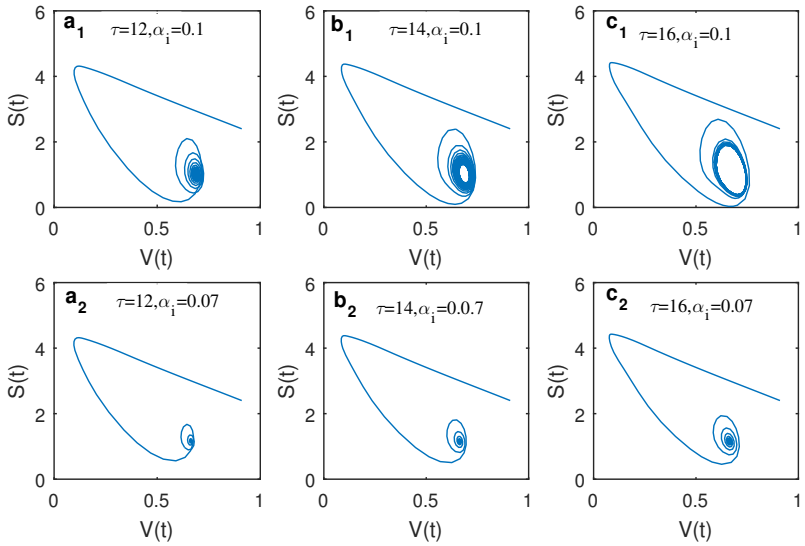

Figure 6: Trajectories of model variables for different values of τ with perturbation.

Figure 5 displays the trajectories of the model variables for different values of the media responses with delay factor $\tau = 15 > \tau^*$. This figure shows that introducing and then increasing the media responses make the system stable. When $\tau = 15 > \tau^*$ the initial oscillation does not persist and the system becomes stable when media responses increase. It is also observed that if media responses are low and $\tau = 15 > \tau^*$, the initial oscillations continue until they pass through the threshold value $\tau^* = 13.6$ days and the system attains cyclic oscillations (see Figure 6).

Figure 6 illustrates a phase diagram of the delay-induced system for altered values of τ with two different values of media responses α_i. In Figure 6(c_1) the trajectories spiral inwards whereas in Figure 6(b_1) the curve's appearance spirals outwards. The top row of Figure 6 shows that for a critical value $\tau^* = 13.6$ days such that the system (2.18) is LAS when $\tau < \tau^*$. The numerical findings validate our analytical findings. The bottom row of Figure 6 shows that the system shifts to local asymptotic stability as media responses increase.

5 Discussions and conclusions

In this paper, we have established a mathematical model to study the media impact on the delay-induced SEIV model inside the ailment transmission time period. We have studied the delay-induced model analytically and numerically. We know that disease transmission totally depends on the basic reproduction number R_0. Depending on the basic reproduction number R_0, the disease will persist or fade out. If R_0 lies between zero and one then the disease dies out and it will persist when R_0 is greater than one. We have derived R_0 for our proposed model and analyzed the sensitivity indices of the parameters.

Our analytical findings reveal that the system will be unstable due to time delay at the endemic equilibrium $\mathcal{U}^*$. The increasing value of delay results in a disease-free state and furthermore, the system always persists unstably, or it may cycle with an infinite sequence of regions consecutively LAS and unstable. It is similarly located that the media responses perform a pivotal function in decreasing the impact of the delay. If media responses increase, the system switches from an unstable to a stable state. Our analysis establishes that the time delay competes with the media responses $\beta(I, \alpha_i) = \left(1 - \frac{I}{I+\alpha_i}\right)$. This encounter causes delay τ and $\beta(I, \alpha_i) = \left(1 - \frac{I}{I+\alpha_i}\right)$ to control the system subject to their corresponding numerical values. It is also witnessed from numerical findings that as $\beta(I, \alpha_i)$ increases, the system attains its stable region in spite of the effect of the threshold value delay at which the system attains Hopf-bifurcation. These simulations are shown in Figure 4 and Figure 6.

Hence our analytical and numerical outcomes can be summarised as increasing value of time delay results in the system oscillations which leads to the instability of the system. However, the unstable system can be stabilized with media responses. Appropriate media responses can neutralize the delay impact and the system attains its stable state.

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